Case report

Extreme hyperpyrexia with cervical spinal cord injury: Survival using an external pad based hypothermia protocol

Jonas P. DeMuro a, d, *, Michael N. Mongelli b, Adel F. Hanna b, Burke A. Cunha c, d

a Winthrop University Hospital, Department of Surgery, Division of Trauma & Critical Care, Mineola, NY 11501, United States
b Department of Surgery, Winthrop University Hospital, Mineola, NY 11501, United States
c Infectious Disease Division, Winthrop University Hospital, Mineola, NY 11501, United States
d State University of New York, School of Medicine, Stony Brook, NY, United States

A R T I C L E   I N F O

Article history:
Accepted 15 March 2013

A B S T R A C T

Background: Extreme hyperpyrexia (T > 41.5 °C [106.7 °F]) represents an unusual challenge in critical care medicine, which has historically resulted in 100% mortality when it is secondary to the thermal dysregulation seen in “quad fever.” Purpose: A novel approach to this disorder, utilizing a protocol driven external cooling device for therapeutic hypothermia to reestablish normothermia, and survival is described. Patient sample: We present the case of a 20 year old male who developed quad fever with extreme hyperpyrexia (T > 41.1 °C [106.7 °F]). Results: He was successfully treated, and survived utilizing an external cooling device based hypothermia protocol. The current literature on this topic is reviewed. Conclusion: While “quad fever” has an extremely high mortality, with the success in this pilot case, early, aggressive use of an external cooling device based hypothermia protocol is advocated.

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1. Introduction

While fever is common in hospitalized patients, extreme hyperpyrexia, namely a temperature >42.1 °C [106.7 °F], is rare. Among the relatively few causes of this phenomenon in hospitalized patients are malignant neuroleptic syndrome, cholinesterase deficiency, drug fever and central fevers [1,2]. In a spinal cord injury (SCI) patient, central fever may be due to “quad fever.” By definition, “quad fever” is an extreme elevation in body core temperature beyond 40.8 °C (105.4 °F) in a patient with SCI [3]. The SCI patient particularly at high risk for the development of hyperpyrexia is the one with a high cervical spine injury that is quadriplegic, although it has been described in the paraplegic with a mid-level or higher thoracic spine injury [4,5]. These cases of hyperthermia usually have no identifiable cause other than the autonomic dysfunction seen in the SCI patient. Severe autonomic dysfunction can result in thermoregulation, as well as neurogenic shock, cardiac dysrhythmias (bradycardia and even asystole), orthostatic hypotension, autonomic dysreflexia, and hyperhidrosis. [3] The incidence of “quad fever” is rare, and the highest reported temperature is 44 °C (111.2 °F) [6]. Among the quadriplegic patients who developed hyperpyrexia, it has historically proved fatal. Although antipyretics are commonly used to treat fever of different etiologies and are generally efficacious, they are ineffective in treating the hyperpyrexia seen in this severe autonomic thermoregulation [3,4].

2. Case presentation

A 20 year-old male was admitted to the emergency department after suffering a gunshot wound to the neck. This trauma resulted in a midcervical level lesion with complete quadriplegia (ASIA grade A, GCS = 15). The patient experienced new onset hyperpyrexia ten days after admission noted to be 41.1 °C (106 °F) measured via a temperature sensing Foley catheter. He had no prior significant medical or surgical history. Despite treatment with broad-spectrum antibiotics, pencillines, and consultation with an infectious disease specialist, no infectious aetiology was found. Review of his medications ruled out the possibility of a drug-induced fever, and thyrotoxicosis was excluded by thyroid function tests as well. The diagnosis of “quad fever” was made as a diagnosis of exclusion retrospectively. Of note, he did not experience any other manifestations of autonomic instability, during the hyperpyrexia, or subsequently.

In an effort to lower his body temperature, pharmacologic efforts included single doses of the following: intravenous ibuprofen (Caldolor) 800 mg, oral acetaminophen 650 mg, as well
as an intravenous steroid, dexamethasone 4 mg. In addition, physical measures included utilization of an external cooling blanket, and multiple ice packs to the axillary and inguinal regions. Despite all of the above measures, the patient’s body temperature continued to rise, peaking at 42.1 °C (107.8 °F). After this temperature spike, the patient’s heart rate was 138 beats per minute and his blood pressure was 118/51 mm Hg.

At that point, our hospital’s hypothermia protocol utilizing an external cooling pad device (Arctic Sun 2000) was implemented to treat the hyperpyrexia as all previous treatments had failed to control the temperature. His blood chemistry was significant for anaemia, an increase in his blood urea nitrogen likely secondary to hypovolaemia from insensible water loss, a mild elevation in the troponin, and leukocytosis during the hyperpyrexic event. Twelve hours after his maximum temperature spike, creatinine phosphokinase (CPK) level increased from 570 µ/L to 3016 µ/L. Within 5 h, normothermia was achieved (Fig. 1) and he was kept at 37 °C (98.6 °F) via the external cooling pad device for a full 24 h to reestablish normothermia. The patient did not develop hyperpyrexia again during his hospitalization, survived without additional neurologic injury, and was discharged to a rehab facility.

3. Discussion

Hypothalamic regulation autonomically controls body temperature [1,5]. These regulatory centres are able to maintain normothermia by utilizing cooling mechanisms to counteract endogenous heat production [7]. When patients suffer high cervical lesions, these regulatory centres may be disrupted, resulting in a pathophysiologial response to temperature control [3]. Also, reduced blood flow to the brain, a side effect of hyperpyrexia itself, can damage central homeostatic mechanisms, further compromising the efficacy of cooling processes [8–10]. These effects can result in excessive heat production and increased body temperature.

Cellular damage has been documented to occur upon exposure to temperatures of 40–41 °C (104–105.8 °F). The effects include protein denaturation, growth inhibition, and alterations in signal transduction [8]. Hyperthermia has known effects on normal organs including lesions in the central nervous system, kidney, heart, liver, and bone marrow. Systemic effects include coagulopathy, hypovolemia, acute tubular necrosis, abnormal liver enzymes which may progress to severe liver necrosis, brain oedema, extensive neuronal loss and gliosis, and intraparenchymal haemorrhage in the adrenal gland. Haematological effects include bone marrow suppression resulting in thrombocytopenia, as well as intravascular hemolysis [9,10].

Our patient developed the hyperpyrexia ten days into his hospital admission which is consistent with previously described cases in which “quad fever” developed within the first two weeks after trauma [3,4]. Therapeutic hypothermia protocols are utilized to induce lower body temperatures after cardiac arrest for neuroprotection, and to treat intractable intracranial hypertension.

However, a hypothermic protocol is not routinely utilized to treat hyperpyrexia. Literature search revealed only one previous study where an external cooling device (Arctic Sun 2000) was used to achieve normothermia in a hyperthermic patient [7]. Although the protocol was successful in inducing lower body temperatures via this noninvasive device, it was not used to treat “quad fever,” but rather neuroleptic malignant syndrome. There is one report of two cases which used an invasive endovascular cooling device to treat “quad fever.” In one of the cases, the device normalized the patient’s body temperature within 5 h, but the patient later died as a result of catheter-related deep vein thrombosis, a complication known to occur with invasive intravenous cooling devices. In the second patient, the cooling device was unsuccessful in controlling body temperature [11].

4. Conclusion

Due to the high mortality associated with extreme hyperpyrexia due to “quad fever” we advocate early aggressive treatment. With the failure of antipyretic medications in these patients, we advocate the use of a hypothermia protocol for aggressive, early treatment of these critically ill patients. To the best of our knowledge, we believe this to be the first reported case of use of a noninvasive cooling device for the treatment of extreme hyperpyrexia (42.1 °C [107.8 °F]) in a quadriplegic patient. Unlike the previous report utilizing an invasive, endovascular device, we found a noninvasive cooling device based protocol to be safe and efficacious at achieving normothermia in our case.

Conflicts of interest

The authors have no conflict of interest, neither financial or otherwise.

Disclosures

None.

Sources of funding

None.

Acknowledgment

None.

References


