

Quad Fever: Treatment through Lowering of Ambient Temperature

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Abstract

Hyperpyrexia is a rare and at times fatal condition seen in an Intensive Care Unit setup. We encountered a case of a 65-year-old patient with road traffic accident presenting with dorsal spine fracture at D₁₀ level. He underwent decompression and fusion for the same. He developed hyperpyrexia of sudden onset on the 10th day of admission with no source of infection and adequate broad-spectrum antibiotic coverage with adequate thrombo-embolic prevention in place. The patient showed no response to antipyretic agents and other cooling methods. The origin of hyperthermia was idiopathic, and we speculate that the cause was secondary to hyperthermic thermoregulatory dysfunction often quoted as “quad fever,” seen in spinal cord injury. We present a brief review of literature and the importance of early identification and treatment of this potentially fatal condition.

Keywords: Hyperpyrexia, paraparesis, quad fever, spinal cord injury, thermoregulation

INTRODUCTION

Incidence of fever in patients with spinal cord injury is as high as 60%–67% and the most common cause is infection. A rare hyperthermic condition, usually fatal, christened “quad fever,” is seen in cervical and rarely in high-/mid-dorsal spinal injuries. It is unresponsive to standard treatment modalities though endovascular and external cooling techniques have been sporadically successful. We share our experience in a low dorsal spine injury.^[1-4]

CASE REPORT

A 65-year-old male was admitted following a road traffic accident with Glasgow Coma Score of 15/15, normal vitals and temperature, acute paraplegia of 0/5, decreased sensations below D8 level, and American Spinal Injury Association score of A. Imaging revealed D10 fracture – dislocation with cord compression [Figure 1]. He underwent dorsal decompression – stabilization under nonneuroleptic general anesthesia. The patient was on prophylactic antibiotics, deep venous thrombosis prophylaxis, and other supportive measures till 9th day when there was a temperature spike (oral) of 38.9°C that further rose to 40.6°C on the following day [Figure 2].

Antipyretics, tepid sponging, cold gastric lavage, and axillary/inguinal ice packs were unsuccessful. Physical examination and pan-cultures (cerebrospinal fluid [CSF], blood, urine, and sputum done on the day of first spike of fever and repeated 48 h later) were unremarkable. Examination of surgical site showed no collection or discharge and the wound was healthy. His C-reactive protein (CRP), thyroid function, and cortisol levels were within the normal limits, with leukocytosis of 18,000 (neutrophils – 80%). Drug-induced hyperthermia was excluded.

The patient was shifted from a ward with temperatures around 30°C–35°C to Intensive Care Unit with temperature set at 18°C, followed by immediate and sustained temperature normalization (37°C) [Figure 2]. This was followed by gradual weaning back to the ambient temperature over 5 days and an uneventful course in hospital thereafter.

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Figure 1: Magnetic resonance imaging, sagittal view, showing D10 vertebral fracture dislocation causing cord compression. T1-weighted image (a) and T2-weighted image (b)

DISCUSSION

Fever is defined as a single temperature reading of 38.3°C or three consecutive readings of 38°C at least an hour apart.^[5,6] Fever above 41.5°C is severe hyperpyrexia. Hyperpyrexia is usually progressive and fatal causing neuronal damage, hypovolemia, reduced cardiac output, coagulopathy, acute tubular necrosis, liver damage, and pancytopenia, which worsen the central dysthermoregulation.^[1,3,7]

Causes for hyperthermia in the absence of infection include neuroleptic malignant syndrome and malignant hyperthermia. The first occurs usually due to administration of haloperidol and other anti-psychotic drugs and may be ruled out by absence of rhabdomyolysis. Malignant hyperthermia is a rare genetic disorder wherein hyperpyrexia occurs after usage of succinylcholine and halothane and consequent dysfunction of hypothalamic thermoregulation.^[3,5]

In our patient, no neuroleptic drugs were used and hyperthermia occurred several days after the exposure to anesthetic agents. Quad fever was first described in 1982 as hyperpyrexia beyond 40.8°C in cases of cervical/dorsal spine injuries. It is a diagnosis of exclusion after ruling out inflammatory/infectious causes.^[1-4] Various pathophysiological hypotheses for this fever include dysautonomia, hyperadrenergic hypermetabolic state, prostaglandin E2 elevation in CSF stimulating the hypothalamus, spinal thermoreceptor afferent dysfunction consequent to spinal cord injury, and associated head injuries.^[2,4,5,8,9]

Our patient exhibited no clinical evidence of wound infection, upper/lower respiratory infection, pressure sores or thrombophlebitis, and his pan-cultures were negative, with a normal CSF study. He developed fever 9th day after the injury despite being on broad-spectrum antibiotics and had a normal CRP with leukocytosis, in agreement with other case reports of this fever.^[1,2,4]

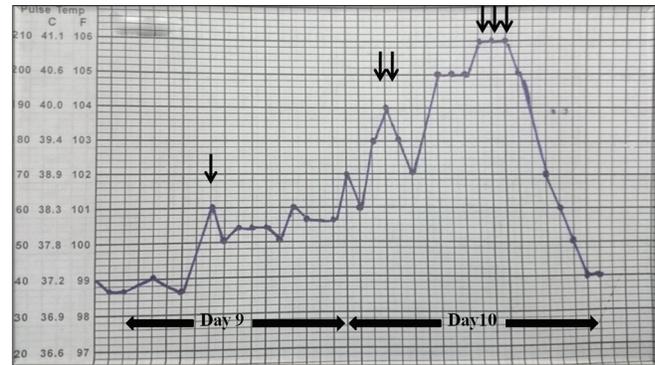


Figure 2: Temperature chart over duration of fever. Arrows representing various interventions temporally – antipyretics (single arrow), physical cooling (two arrows), air conditioning (three arrows)

The hypothalamic temperature receptors in the preoptic area are sensitive to blood temperature variations and maintain temperature by balancing heat generation and dissipation through autonomic, endocrine, and behavioral responses by changing signal output as the temperature varies from the set point of 37.1°C. Receptors in the skin, spinal cord, and abdomen also send impulses to the hypothalamus.

Temperature rise causes sympathetic inhibition with resultant cooling by vasodilation/perspiration, whereas temperature drop causes vaso-constriction, shivering, neurotransmitter-mediated hypermetabolism, and behavioral responses for warming.^[3,5,6]

In fever, hypothalamic thermal set point rises secondary to endogenous pyrogens with intact thermo-regulatory mechanism. However, in hyperthermia, the thermoregulatory set point remains unchanged and the body temperature increases due to failure of peripheral heat dissipation mechanism secondary to disease, drugs, or excessive internal/external heat. Hyperthermia is differentiated from fever by history and clinical course of events, the skin remaining hot and dry, and antipyretics routinely fail due to the normal thermal set point in hyperpyrexia.^[5,6] Therefore, treatment is directed toward peripheral physical mechanisms, namely, tepid sponging, icepacks in groin/axilla, internal cooling by cool intravenous fluids, gastric cold water lavage, endovascular cooling, external cooling pad device, use of agents which interfere with vasoconstriction (phenothiazines), and agents blocking muscle contractions (benzodiazepines).^[1,3,5,6,10]

Patient not responding to antipyretics and physical cooling but doing so dramatically to alteration of ambient temperature through air conditioning is reported for the first time.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published

and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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