



## Case Report

## Management strategies of burns associated hyperthermia: A case report



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## ARTICLE INFO

## Article history:

Received 2 May 2017

Accepted 30 May 2017

Available online 14 June 2017

## Keywords:

Burns

Hyperthermia

Management strategies

## ABSTRACT

**Introduction:** Hyperthermia in the burn injured patient has been noted to exist either in the presence or absence of infection. However, there is a need to manage this feature aggressively as if left unmanaged; it might lead to other deleterious effects.

**Objective:** Thus, this case report assesses the management strategies instituted in the management of burns associated hyperthermia.

**Case report:** A thirty-four year old male who sustained thermal burns was admitted with a baseline temperature of 36.1 °C. However elevated temperature to the level of 40.40 °C was noted but other tests proved the absence of infection. Several strategies were instituted to resolve the hyperthermic state.

**Conclusion:** It was noted that these strategies possess limited efficacy and there is need to assess other modalities that works best and cost effective. Further research is also warranted in understanding the nature of hyperthermia in the absence of infection in the burned patient.

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## 1. Introduction

A high body temperature has been described as the most common sign of systemic response to injury and virtually all burn patients have elevated core body temperatures and even a full haemogram may reveal leukocytosis [1,2]. Thus, high temperature after burn injury has been described as an unreliable indicator of infection. Nonetheless, infection is a relatively common complication in patients with burn injuries [1]. It is estimated that up to 75% of deaths following burn injury are related to infection. Immunosuppression is a universal feature of major thermal injury and burn patients are more susceptible to microbial colonisation and infection. The hypermetabolic response elicited by a burn injury is marked by increased energy expenditure and muscle protein catabolism [2].

Hyperthermia which has been defined as an elevated body temperature due to failed thermoregulation, further increases energy expenditure and loss in burn patients. Therefore, there is a metabolic benefit in attenuating high body temperatures after burn in such patients [1]. This however must be done such that the burned patient does not become hypothermic. Hyperthermia has been classified into mild (>37.5 °C) and severe (>38.8 °C) and it reflects the body's inability to dissipate heat. However, the mechanism of hyperthermic states in burn injured patient is not properly understood [3].

Cytokine levels usually increase immediately after burn and approaches normal levels only by three to six months in the post burn period [2]. Burn injuries with greater total burned surface areas are however characterised by more marked inflammatory responses specified by increasing concentrations of pro-inflammatory cytokines that enhance the catabolic process [3]. The three endogenous pyrogens that mediate hyperthermia after burn are interleukin-1 (IL-1), tumor necrosis factor and interferon [4]. Prostaglandins and leukotrienes have also been implicated in this issue [5]. In addition to inducing hyperthermia, these pyrogens also modulate a large number of host defense responses [4].

Furthermore, the ability to regulate central body temperature is absent in patients with severe burn injury. The hypermetabolic processes associated with burn injury increases the metabolic response rate to recompense for the water and heat loss. Loss of water reaches 4 L/m<sup>2</sup> burn area per day [6–9]. The response to this insult, partly enhanced by increased ATP consumption is to increase core and skin temperatures by 2 °C above normal compared with unburned patients [10]. This response is similar to that of cold acclimatisation and patients who do not mount this response are most likely to be experiencing sepsis or possess diminished physiologic capabilities to maintain needed body temperature [11–13].

Mild hyperthermia in the first 24 h following injury is almost always the result of pyrogen release. After 72 h, burn patients develop systemic inflammatory response syndrome (SIRS), characterised by tachycardia, relative hypotension, and hyperthermia [4]. In fact, body temperature may be as high as 40 °C and the

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leukocyte count may be as high as 20,000 cells/mL during satisfactory recovery [4].

Thus, elevation of body temperature above normal, leukocytosis, and other signs of inflammation are common among burn patients and should be expected. However, such high body temperatures if left unattended to add up to the already established hypermetabolic state of the patient. For instance, one study that sought to investigate the influence of hyperthermia on the hypermetabolic response in burned children revealed the association of severe hyperthermia with further increase in energy expenditure and muscle protein catabolism in otherwise hypermetabolic burned children [1]. In similar lines, another study also suggested that a sustained body temperature of 40 °C could lead to cellular injury and subsequently, death [8].

Despite these, there is limited exploration of how best to manage burns associated hyperthermia in the Ghanaian setting and studies have focused mostly on managing burns associated hypothermia. Thus, this case report represents management strategies instituted to manage a severely burned patient who developed hyperthermia in the Burn ICU.

## 2. Case presentation

PM is a 34 year old male who sustained thermal burns whilst discharging fuel from a petrol tanker. He was rushed to the tertiary health facility as soon as the injury occurred by co-workers on 28/04/2016. At the triage area, he was alert and had a Triage Early Warning Score (TEWS) of 4 but the presence of facial burns made the nurse triage him to a higher color. His vital parameters on admission recorded: axillary temperature- 36.1 °C, pulse- 110 bpm, respiration- 22 cpm and blood pressure recorded 120/90 mmHg. Further assessment at the Red Wing of the Accident Emergency Unit revealed singed nasal hairs, adequate air entry bilaterally, vesicular breath sounds with transmitted sounds, charred and oedematous face. Within some minutes hoarseness of voice was noted and that prompted the emergency team to intubate him. He was placed on mechanical ventilation at Intermittent Positive Pressure Ventilation (IPPV) mode with fraction of inspired oxygen at 100%. Fluid resuscitation was commenced with lactated ringers based on calculations obtained using the Parkland formula. Wound assessment also revealed mixed thickness burns with total burned surface area (TBSA) of 35%. Continuous sedation and pain relief was achieved with IV midazolam 2 mg/h and IV morphine 2 mg/h via perfusors. A nasogastric tube was passed aid in feeding as well as a urethral catheter to monitor the adequacy of the fluid resuscitation. Blood samples were obtained for various investigations and full haemogram results indicated a total White Blood Cell Count of  $8.7 \times 10^3/\mu\text{L}$ . Later that day, he was transferred to the Burn ICU for continuity of care. The temperature patterns throughout that day were between the ranges of 36.1–37.8 °C. On 29/04/2016, a change in temperature pattern was noted as it ranged from 38.5 °C to 39 °C. Thus, a full haemogram was requested but WBC count was noted to be  $8.9 \times 10^3/\mu\text{L}$  which indicated that it is in the normal range and as such IV paracetamol 1 g was administered in addition to IV cefuroxime 750 mg three times daily and IV metronidazole 500 mg three times daily. After administration of IV paracetamol, the temperature was noted to have dropped to 38.4 °C. On 30/4/2016, a similar pattern was noted. Thus, the urethral catheter was changed. Wound care was also rendered and wounds on observation did not look infected. On 01/05/2016, the highest core body temperature was recorded as 40.4 °C. Thus, tepid sponging of unburned areas was carried out alongside routine administration of IV paracetamol. Room temperature was also altered to a cool mode and excess top sheets removed. Within thirty minutes, the temperature was noted to have diminished to 39.8 °C but after halting the process of tepid

sponging, the temperature rose to 40 °C. Blood samples were also obtained for blood culture and a repeat full haemogram. However no organism was isolated and the WBC count was within the normal limits. The temperature followed similar patterns till he was re-epithelisation was noted and client was finally discharged home from the Burn Unit on 30/06/2016.

## 3. Discussion

Hyperthermia after burn injury may occur but its management appears rather challenging. Despite this, there has been paucity of studies in that area as the focus remains on ways of preventing or managing hypothermia. Controversies exist regarding whether milder degrees of fever secondary to infection or the systemic inflammatory response syndrome should be treated in critical care patients, as it may be part of a beneficial host response and treating it may not alter outcome [15,17].

The cytokine response to burn injury has been noted to characterise the Systemic Inflammatory Response Syndrome in burn injury. Wilmore and colleagues [18] showed that the hypermetabolic response can be attenuated by increasing ambient temperatures to 33 °C, a thermal neutral temperature. At this temperature, the energy required for vaporization is derived from the environment rather than from the patient. By maintaining ambient temperatures between 28 °C and 33 °C, resting energy expenditures decrease from a magnitude of 2–1.4 in patients with severe burn injury [9,10].

A critical look at the case report indicates that strategies instituted to manage hyperthermia in our unit appears to be of limited efficacy as in most instances, they are unable to attenuate the elevated temperatures satisfactorily. Physical methods of cooling employed in the unit such as reducing the number of top sheets and sponging have been indicated to be first line actions in the management of increased temperatures [16] but they appear to be of minimal help in the case of burn injuries. The use of cooling blankets has been reported useful but this is unavailable in our setting. In some cases of hyperthermia, more invasive methods of cooling may be implemented and this commences with infusion of cooled intravenous fluids [16]. However, this area also requires further research to establish its usefulness in the burn injured patient. In developed countries, the use of invasive approaches such as intravascular cooling catheters, haemodialysis or haemofiltration and cardiopulmonary bypass have been reported [16]. However, these approaches have been identified to be associated with various risks and dependent on resources available. In recent times, the use of an intravascular heat exchange catheter (Cool-Gard™ Alsius) has been indicated to be beneficial in managing hyperthermia in the burn injured patient [16].

However in the Ghanaian setting, these advance products are not available and as such other innovative measures may be needed to manage hyperthermia in the burn injured patient as the current methods utilised are of limited efficacy. Thus, it may be necessary to consider other adjunct measures to manage a burned patient with hyperthermia based on what happens during hypothermic states. For instance, hypothermic periods have been identified to impair glucose metabolism. Conversely, hyperthermia plus the hypermetabolic state periods may increase glucose utilisation and rapid depletion of glycogen stores [14]. Thus, serum glucose levels need to be monitored as the patient is particularly at risk of developing hypoglycaemia especially if nutritional intake is poor. In addition, hyperthermia plus the already established hypermetabolism state raises oxygen consumption [14] and as such it is imperative that oxygen saturation levels are monitored. Supplemental oxygen may be commenced based on deviations identified. The effectiveness of these strategies may however need further investigation.

With regards to pharmacological management, paracetamol has been reported as been beneficial with minimal side effects to the burn injured patient [19]. Also, NSAIDS have been indicated to be useful in managing hyperthermia but due to risk of exacerbating renal impairment and gastric ulceration, it might not be useful in some situations [16]. These may imply the need to critically monitor the burn injured patient whilst in the hyperthermic state. Also, further researches are warranted in understanding the mechanism of hyperthermia in burns (especially those that occur in the absence of infection) and assess other measures that can be employed in managing hyperthermia in resource constrained healthcare settings.

#### 4. Conclusion

Considering the fact that hyperthermia further heightens the hypermetabolic state, there is a need to aggressively manage it whenever noted. However, the strategies used in the Burns Intensive Care Unit have been noted to be of limited efficacy which may mean that burn patients are likely to become hyperthermic for a longer period of time. Though advances have been made in developed countries in this regard, such technology is unavailable in our setting. Thus, it may be appropriate to employ other adjunct measures in addition to the strategies noted to manage hyperthermia. Further studies are warranted in assessing what works best in developing countries.

#### Conflict of interest

None declared.

#### Funding

None.

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