The Egyptian Journal of Critical Care Medicine (2015) 3, 1–8

The Egyptian College of Critical Care Physicians

The Egyptian Journal of Critical Care Medicine

http://ees.elsevier.com/ejccm
www.sciencedirect.com

ORIGINAL ARTICLE

Impact of body temperature and serum procalcitonin on the outcomes of critically ill neurological patients

Abeer Feasal, Abdou El Azab, Karim Mashhour *, Amr El Hadidy

Critical Care Medicine Department, Cairo University, Egypt

Received 1 October 2014; revised 5 March 2015; accepted 12 May 2015
Available online 3 June 2015

KEYWORDS

Serum procalcitonin;
PCT;
Hyperthermia;
Head trauma;
Stroke

Abstract *Introduction:* Fever is common in patients with acute stroke, and mostly it is due to infectious complications. The neurologic effects of fever are significant, increased temperature in the post-injury period has been associated with increased cytokine activity and increased infarct size.

Aim: To test the hypothesis that fever and increased serum procalcitonin are associated with poor outcomes after neurological injury.

Methodology: Fifty patients (30 males (60%) and 20 females (40%) mean 43.8 ± 11.7 years) were divided into two groups: Group I: 25 traumatic patients (i.e., head injury) and Group II: 25 non-traumatic patients (i.e., stroke). Temperature was measured from admission until the patients were discharged or died, and PCT was measured on day 1 of admission and after 48 h of admission.

Results: Fever has been associated with poor outcome, as fever is linked to worse GCS scores (12.6 ± 1.2 vs. 7.7 ± 2.6 in patients with fever, *P* 0.001), longer MV durations (3.6 ± 1.0 vs. 22.4 ± 9.1 days, in patients with fever, *P* 0.001), longer ICU length of stay (8.1 ± 4.7 vs. 23.0 ± 8.0 days in patients with fever, *P* 0.001) and increased mortality (*P* = 0.001). There were significantly higher PCT levels in the mortality group versus the survived group at day 1 (4.15 ± 0.82 vs. 2.47 ± 0.059 ng/ml, respectively, *P* 0.0001) and after 48 h of admission (5.20 ± 1.14 vs. 3.19 ± 0.092 ng/ml, respectively, *P* 0.0001).

Conclusion: Fever had a strong link to worse GCS, longer MV durations, increased length of ICU stay, higher mortality rates and worse overall outcomes in neurocritical patients. High PCT levels can predict mortality in those patients.

* Corresponding author.
E-mail address: Kmash@link.net (K. Mashhour).

Peer review under responsibility of The Egyptian College of Critical Care Physicians.

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1. Introduction

Fever is a common condition in patients with stroke and other brain injuries. Hyperthermia appears to correlate with poor outcome in these patients, although a direct causative link has not been established yet. After controlling for illness

http://dx.doi.org/10.1016/j.ejccm.2015.05.001

severity, diagnosis, age, and complications, fever has been found to be strongly associated with increased lengths of intensive care unit (ICU) and hospital stays, a higher mortality rate and worse overall outcomes [1,2].

Sometimes fever in stroke patients is due to infectious complications. In some patients with acute stroke and fever, the infection source cannot be identified. In some of these cases where fever exists without an obvious infection source, fever does not respond to empirical antibiotic treatment and is thought to be due to a central nervous system lesion. The presence of fever, in general, in patients with acute stroke has been associated with poor outcomes [3].

The neurologic effects of fever are significant as increased temperature in the post-injury period has been associated with increased local cytokine activity, increased infarct size, and poorer outcomes in the acute phase of injury. This is, in part, related to the fact that patients at risk of intracranial hypertension may be significantly affected by an increase in temperature because the intracranial blood volume increases with fever. This reduces compliance and puts the brain at risk for further injury. Hyperthermia, when high enough (>43 °C), has been reported to cause neuronal injury in normal brain, and lengthy periods of moderate (40 °C) hyperthermia have been reported to alter brain structure and function [4].

Additionally, traumatic brain injury (TBI) patients are at risk for secondary injury from fever because for every 1 °C increase in core temperature, there is a 5–7% increase in the metabolic rate. This taxes the stressed energy reserves of these severely brain injured catabolic patients. The higher metabolic demand of fever exacerbates this problem and can lead to additional muscle and fat store losses [5].

In TBI patients procalcitonin (PCT) increased only moderately in most patients and peaked at days 1, 2 after trauma, and the concentrations rapidly decline thereafter. Complications, such as sepsis, infection, blood transfusion, prolonged intensive care unit treatment, and poor outcomes were more frequent in patients with initially high PCT levels (>1 ng/ml) [6].

Traumatic brain injury patients are especially prone to develop complications such as infections and sepsis. Because clinical symptoms and conventional markers are not always reliable signs for sepsis and infection diagnoses; therefore, biomarkers such as PCT are often used as a diagnostic tool in these patients. However, similar to patients undergoing elective surgery, an increase of PCT during the early postoperative or post-traumatic period may occur independent of the sepsis or infection diagnoses [6].

Inflammatory response is also a principal early component in the pathophysiology of stroke. Serum PCT, a marker of septicaemia and infection severity, has also been proposed as an indicator of systemic inflammatory response in noninfectious situations in these patients [7].

2. Aim of the work

The purpose of this study was to test the hypothesis that fever and increased serum PCT are associated with poor outcomes after neurological injury.

3. Patients and methods

3.1. Patients

Study was conducted on 50 acute neurological insult patients (30 males and 20 females) that were admitted to the neurocritical unit at Cairo University between January 2012 and September 2012.

3.1.1. Patients were divided into two equally large groups

- Group I: 25 patients (traumatic patients, e.g., head injury).
  - 3 pts with diffuse axonal injury.
  - 5 pts with concussion.
  - 4 pts with cerebral contusion.
  - 2 pts with cerebral haemorrhage.
  - 2 pts with fracture base of skull.
  - 4 pts with subdural haemorrhage.
  - 3 pts subarachnoid haemorrhage.
  - 2 pts intracerebral haemorrhage.

- Group II: 25 patients (non-traumatic patients, e.g., stroke).
  - 14 pts with thrombotic stroke.
  - 4 pts with embolic stroke.
  - 7 pts with haemorrhagic stroke.

3.1.2. Exclusion criteria

- Sepsis patients.
- Septic shock patients.
- Chronically ill patients.
- Patients with known hyper-bilirubinemia (>0.4 mg/ml) or hypertriglyceridemia (>10 g/l). We excluded pts with hyper-bilirubinemia and hypertriglyceridemia due to possible interference with PCT measurement.

3.1.3. All patients were subjected to the following

1. History evaluation.
2. Clinical examination.
3. Glasgow coma scale assessment.
4. Body temperature measurement.
5. PCT level measurement.
6. Intensive care unit duration (in days) assessment.
7. Mechanical ventilation (MV) duration (in days) assessment.

3.2. Methods

Body temperature was measured in the axillary region with a medical thermometer. We defined ICU fever as body temperature above 38 °C. Body temperature was measured daily from day 1 of admission until patient discharge or death.

3.3. Procalcitonin measurements

We measured PCT on day 1 of admission (PCT1) and 48 h after admission (PCT2).