Article

Evidence-based Fever Management in Patients with Traumatic Brain Injury: A Case Study[1]

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Abstract

Fever management in patients with traumatic brain injury (TBI) has been a challenge in neurosurgical nursing practice since fever has impact to the higher mortality and disability including length of stay. In addition, evidence-based guideline for fever management in TBI patients is little known to support nursing practice of how to implement. This article aims at presenting an evidence-based nursing intervention for fever management in TBI patients. By applying an evidence-based care approach, two case studies selected from SICU/trauma ward in a tertiary hospital in Songkhla province were reviewed to identify the pattern of fever, factors related to fever among patients with TBI, and outcomes after implementing the guideline.

The results revealed that patterns of fever were different. In severe TBI case, high fever (> 38 °C) from the day of admission throughout the first 72 hours was observed and its cause related to neurogenic fever. High fever with infection causes can lead to a high risk of IICP, and is then difficult to manage. Another case with mild TBI had developed low grade fever (< 38 °C) within first 72 hours as a result of multiple injuries. Non-specific factors of fever caused by inflammatory response of wound healing and operation were easier to manage. Hence, the greater the severity of TBI, the more comprehensive fever management required. In addition, factors related to fever management were related to the nurses' knowledge, health care team participation, and available resources. It is a challenge for nurses to manage fever in TBI patient based on evidence-based guideline implementation in order to achieve better outcomes.

Keywords: fever management; IICP; traumatic brain injury

Introduction

Increased intracranial pressure (IICP) or intracranial hypertension (ICH) is a common problem seen in patients with moderate to severe traumatic brain injury (TBI) and it is a dangerous condition that occurs in up to 75% of patient who have severe TBI. In addition, it is associated with up to 95% fatality.[1] These conditions are due to the increased ICP, which is the pressure inside cranium, and results from defective compensation of the brain components (i.e. blood, brain tissue, and cerebrospinal fluid) when the physiology of the brain is disturbed from brain injury.[2]

There are several specific factors that may induce or aggravate IICP in TBI patients, for instance, obstruction of venous return blood (e.g. head position, agitation, etc.), respiratory problems (e.g. airway obstruction, hypoxia, hypercarbia, etc.), fever, severe hypertension, hyponatremia, anemia, and seizures. However, fever is one of the most significant factors leading to IICP or having more severe condition.[3]

Fever in moderate/severe TBI patients is a common symptom and is regarded as one of the difficult problems in management. Mostly, the rising of body temperature after TBI may be experienced by patients whose conditions in the acute phase or within the first two days after injury.[4] For example, a study in China showed the proportions of fever of approximately 20-50% in TBI patients, and more than 50% in TBI patients in a neurological intensive care unit experience a body temperature above 38.5 ^{o}C.[5] However, the criteria to diagnose of fever in TBI patients are a body temperature $\geq$ 38.3 ^{o}C when measured at a core temperature site[6] or $\geq$38 ^{o}C at the axillary site.[7]

The causes of fever are divided into two types based on the origin: infectious fever and non-infectious fever (no source of infection).[4] Infectious fever may be caused by exogenous stimuli such as inflammation or infection stimulating the neuroimmune response. So the macrophages and/or microglia will respond by releasing endogenous cytokines into the circulation and the circum-ventricular organs, resulting in an activation of the cyclooxygenase 2 pathways and inducing the production of prostaglandin E2 (PGE2). This can cross blood-brain barrier to penetrate the brain and stimulate the thermoregulatory center which can result in a rise in body temperature. In addition, non-infectious fever or neurogenic fever or posttraumatic hyperthermia (PTH) is the changing of the hypothalamic "set point" and has body temperature high as a result of brain injury. The injury to hypothalamus after brain injury can lead to abnormal increase in body temperature.[8,9]

Moreover, the physiological cerebral changes induced by variations in brain temperature can be explained as follows. If brain temperature increases, it induces an increase in metabolism of brain and blood flow, leading to increase intracerebral vascular volume and IICP.[10] Furthermore, the increase of brain temperature can lead to increase of oxygen consumption, CO_2 gas production, and decrease in pH (acidosis). This CO_2 will cross the blood-brain-barrier leading to acidosis and affect extracellular environment of neurons that coordinate vascular control. The acidosis leads to vasodilatation and also increased ICP. The last effect of changing in brain temperature is a breakdown of the blood-brain-barrier which it is out cause an increased permeability, and leads to cerebral edema and finally an increased ICP. When ICP exceeds a critical point, displacements and brain herniation will occur.[11]

Importantly, fever was shown to be associated with worse neurologic outcome of TBI patients, including increased intracranial pressure, lower Glasgow Coma Score (GCS), increased length of stay in ICU, poorer functional status, long-term functional impairment, and finally increased fatality rate.[8,12,13] A small increase in brain temperature can result in a considerable change to neuron metabolism. For every 1 ^{o}C rise

in body temperature, there is a 13% increase in metabolic rate.[4] and neuronal parameters change (i.e. cerebral blood flow, CO_2, pH, O_2, blood-brain-barrier). These effects may induce an increase in intracranial pressure (IICP) and have an impact on neuronal cells, which is the secondary injury after TBI.[10]

Due to the high risk and impact of fever in TBI related to IICP, it is necessary to understand the pattern of fever and the factors related to high fever in TBI patients. Appropriate management of fever may help improve the neurological function and decrease mortality of TBI patients in both the short- and long-term, and also help to reduce medical care costs by reducing the length of hospital stay.

Evidence Based Guideline for Fever Management in TBI

In order to summarize an evidence-based fever management method, ten main articles based on the level of evidence were found. The evidence from 5 experimental studies,[6,14-17] 1 cases control study,[18] 2 cross-sectional studies,[9,19] 1 observational-cohort study,[13] and 1 systematic review[20] were then analyzed and synthesized for development of the fever management guideline in TBI patients. To ensure the quality of evidence, the authors had one more faculty member in the nursing practicum course to assess as it was a part of the guideline development.

The summary of intervention in fever management guideline was categorized into three groups based on the patient's temperature assessment (Table 1).

Table 1 Fever Management Intervention Based on Body Temperature

Body temperature	Types of intervention	Machine setting and methods	Body temperature assessment & monitoring
1. Body temperature ≥38.3 °C (tympanic site) or >38.0 °C (axillary site)[21] and any number of risk factors.	1. Water circulating blanket (3 pieces of circulating blanket). 2. Water cooling gel-coat system (4 pieces of gel-coat pad).	- It had automatic temperature control and set temperature of water at ≤ 24 °C which is the starting temperature for reduction of fever and comfort[22] - The temperature setting can be adjusted up/down for 1-2 °C/hour and temperature setting can be set in the range from 4-42 °C depending on BT of the patient[6,23] - Patients exhibiting shivering during cooling are observed and to prevent patient from shivering, the patient is wrapped with cloth around the arm from shoulder cover to finger including from the knee cover to feet - Patient continues to receive antipyretic as prescribed by the physician	- This cooling is applied to reduce fever for 24 hours to achieve the target temperature of ≤37.2 °C (tympanic site) or ≤36.9 °C (axillary site)[6] - After cooling for 1 hour, if the BT show target temperature of ≤37.2 °C (tympanic site) or ≤36.9 °C (axillary site), cooling device is stopped and temperature with vital signs recorded every 4 hours - For the case with BT still >37.2 °C (tympanic site) or >36.9 °C (axillary site), cooling will be continued and BT and vital signs recorded every 4 hours

Body temperature	Types of intervention	Machine setting and methods	Body temperature assessment & monitoring
2. Body temperature 37.5-38.2 °C (tympanic site) or 37.5-38.0 °C (axillary site) and ≥2 risk factors to develop the fever.	- Cold water sponging and cold packs	- The material used for sponging include cold water from a tap in bucket (at approximately a room temperature) and 5 sponge towels[14,24] - 5-6 cold packs - Cold water is used for sponging and cold packs placed at neck, both axilla and groins for 30 minutes as step by step	- The BT is measured at 30 minutes immediately after applying the intervention, and then recorded every 1 hour. - After achieving normal BT at 36.5-37.4 °C, cooling is stopped and BT recorded every 4 hours
3. Body temperature 37.5-38.2 °C (tympanic site) or 37.5-38.0 °C (axillary site) and <2 risk factors to develop the fever.	- Putting cold packs	- The material is only cold packs, 5-6 pieces - Cold packs are put on neck, both axilla and groins and cold packs changed every 30 minutes	The BT is measured every 4 hours until achieving normal BT at 36.5-37.4 °C, and then cooling stopped.

The factors related to fever assessment form was used to assess the factors associated with fever in TBI patients. There were 12 specific factors based on literature review, namely type of injury (i.e. diffuse axonal injury, frontal lobe injury, subarachnoid hemorrhage (SAH), intraventricular hemorrhage (IVH), intracerebral hemorrhage (ICH), GCS at emergency room (ER) ≤ 8, CT brain found cerebral edema, blood sugar (BS) on admission > 150 mg/dl, systolic blood pressure < 90 mmHg, white blood cell (WBC) on admission > 14,500 cell/mm,^{3} body temperature (BT) on admission >36 °C, infection (e.g. pneumonia, urinary tract infection: UTI, etc.), intubation, intraventricular catheterization, placement of ICP monitor, and any drug for fever.

Regarding the guideline implementation, it was applied in two TBI patients admitted in the surgical intensive care unit (SICU) and trauma ward of one tertiary hospital in Songkhla province. The cases were informed about the care they may receive and other information related to assessment, intervention, and any alert signs which may detect and managed for appropriate methods following the guideline in table 1. Data on temperature and factors related to fever were accessed from patients every day during practice. The other signs and symptoms were collected from daily routine nursing documentation. The body temperature and IICP were evaluated every day for at least 7 days to reflect the pattern of fever.

Case Studies

Two case studies (Mr. A and Ms. B.) are presented as follows:

The demographic and medical information

Mr. A was 21 years old admitted on 26 April, 2015. He had had a motorcycle accident and was diagnosed with severe TBI. CT scan showed the contusion at right (Rt.) frontotemporal, SAH at Rt. parietal, thin epidural at left (Lt.) parietotemporal. Other injuries were, Rt. clavicle fracture, Lt. distal femur fracture 33-B3, Rt. lung contusion, liver injury grade IV, hematuria, Rt. rib 6-7 fracture, and thin subdural at Rt. Inter-hemisphreric. He had received operation by Open Reduction Internal Fixation (ORIF) with distal femoral Locking compression plate (LCP) (on skeleton traction 40 lbs at Lt. leg) and Lt. Burr hole with irrigation (Table 2).

Ms. B was 76 years old admitted on 1 May, 2015. She had a car accident during walking across the road. Her diagnoses are mild TBI and multiple organ injury (liver injury, bladder contusion, Fx at Lt. inferior and posterior rami, Fx at Lt. fibula and medial malleolus, lung contusion, and Rt. subdiaphragmatic injury). She had 3 operations which were (1) exploratory laparotomy with Rt. thoracotomy with suture to stop liver bleeding with packing, (2) exploratory laparotomy for removing packing and repair of Rt. ก้iaphragm, (3) exploratory laparotomy for removing packing and stopping bleeding (Table 2).

Table 2 The TBI Patient's Demographic and Medical Information

Case	Age (year)	Admission date	Diagnosis	Results of medical investigation	Operation
Mr. A	21	26 April, 2015	- Motorcycle accident - Severe TBI	- CT scan found contusion at Rt. frontotemporal, SAH at Rt. parietal, thin epidural hemorrhage at Lt. parieto temporal, thin subdural hemorrhage at Rt. Inter-hemisphreric - Rt. clavicle fracture, Lt. distal femur fracture 33-B3 - CT scan was found Rt. lung contusion, liver injury grade IV, hematuria, Rt. rib 6-7 fracture	- ORIF with distal femoral LCP (on skeleton traction 40 lbs at Lt. leg) - Lt. Burr hole with irrigation
Ms. B	76	1 May, 2015	- Pedestrian injured - Mild TBI and multiple organ injury	- CT scan found subdural hematoma and subarachnoid hemorrhage - CT scan found liver injury, bladder contusion, lung contusion, and Rt. subdiaphragmatic injury - Fx at Lt. inferior and posterior rami, Fx at Lt. fibula and medial malleolus	- Exploratory laparotomy with Rt. thoracotomy and suture to stop liver bleeding with packing - Exploratory laparotomy for removing packing and repair of Rt. diaphragm - Exploratory laparotomy to remove packing and stop bleeding

Factors related to fever for Mr. A and Ms. B

The data from initial assessment showed that Mr. A had some potential factors related to fever at day 1 to day 6 of admission. These were type of injury that was severe, GCS $\leq$ 8 ($E_{2-3}V_TM_4$), BT at admission >36 °C, intubation, and no prophylactic drug for fever at that time due to severe liver injury grade IV in CT scan. However, at day 9 of admission, signs and symptoms of infection indicated urinary tract infection (UTI). UTI was one important source of fever found in this case until day 15 of assessment (Table 3).

By initial assessment of Ms. B (from the first day of admission), there were some factors that could lead to developing fever including type of TBI (SAH), high blood sugar on admission > 150 mg/dl, intubation, and no prophylactic drug for fever. This high BS level may be from the underlying disease of Diabetes Mellitus (DM). The major trauma in this case study was not the brain, fever may be from the other factors (Table 3).

Table 3 Factors Related to Fever in Two Cases of TBI Patients

Case	Factors related to fever in TBI patients				
	Factor 1	**Factor 2**	**Factor 3**	**Factor 4**	**Factor 5**
Mr. A	- Severe TBI (GCS ≤ 8 at admission) - Type of brain injury (SAH)	BT on admission > 36 °C	Intubation	No prophylactic drug for fever	Infection (UTI)
Ms. B	Type of TBI (SAH)	BS on admission > 150 mg/dl (Underlying disease was DM)	Intubation	No prophylactic drug for fever	None

Pattern of fever for Mr. A and Ms. B

Mr. A had high BT from the first day of admission until first day of operation of his leg (day 6 of admission) due to its severity of head injury with inflammatory response. Mostly, he had BT > 38 °C within 72 hours of admission which was related to neurogenic fever. In addition, after the operation of his leg, he had a high fever with BT mostly ≥ 39 °C. This may have been due to inflammatory response of healing wound/tissue injury combined with brain injury. The fever was more difficult to manage after 3 days of left leg operation. Mr. A showed sign of SIRS (Systemic Inflammatory Response Syndrome) which may have some infections and BT remained high (day 9 of admission). Later on, he developed UTI and was treated with antibiotic. Therefore, this persistent and high BT may have been from both causes: type of brain injury and infection. The important issue is that pattern of fever in this case was very dynamic, fluctuating and difficult to control. The pattern of fever in this case is shown in Figure 1.

Although Ms. B had a fever, it had only occurred 1 time in some shift within the first 72 hours (see figure 2). Most of BT showed low grade fever (< 38 °C). The major trauma was from other organs injury as well as non-specific factors were seen in this case. So, persistent or high fever was not found. However, the low grade fever found in this case may come from inflammatory response of wound healing and operation. The pattern of fever in this case was shown in Figure 2.

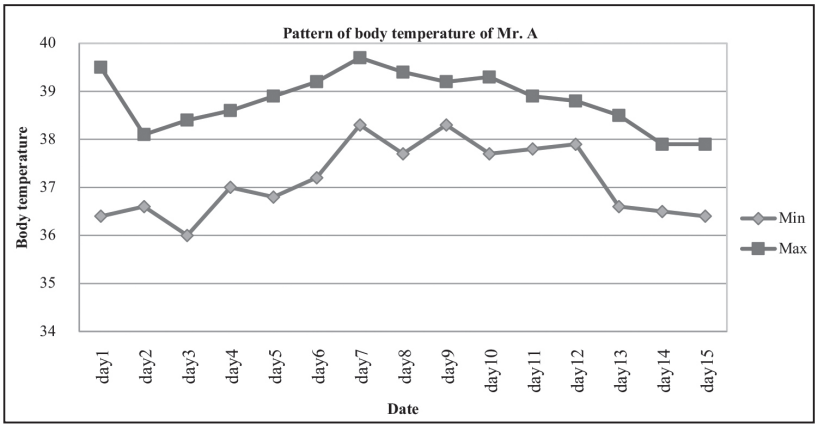

Figure 1 Pattern of Body Temperature of Mr. A from Admission Date to Last Day of Approaching.

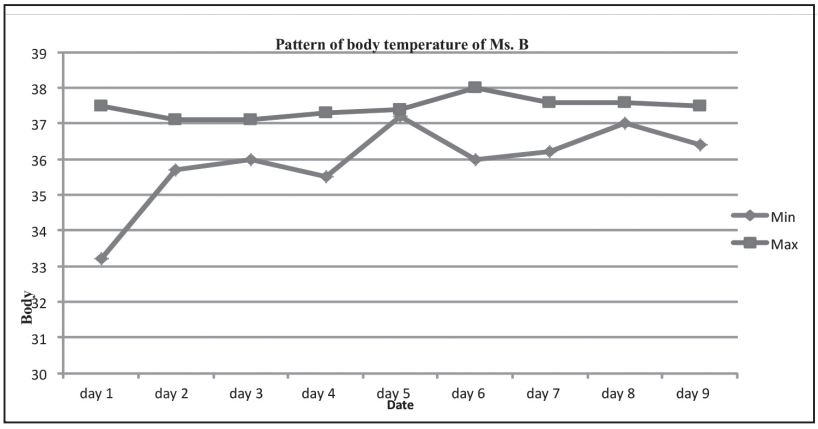

Figure 2 Pattern of Body Temperature of Ms. B from Admission Date to Last Day of Approaching.

Outcome from fever management for Mr. A and Ms. B

For Mr. A, the process and outcome for fever management to prevent IICP were evaluated for 1 week. This case was managed following the guideline in category 1 (BT ≥38.3 °C at axillary site). Due to various factors related to fever particularly infection caused, it was difficult to manage the fever. The BT remained high throughout 15 days of assessment with a temporary low grade of fever. In addition, Mr. A had a high risk of IICP as shown by the wide pulse pressure. While Ms. B received the intervention for fever management in category 2 and BT was easier to manage than Mr. A. The BT was decreased to the normal range and she did not show fever again.

Discussion

From case analysis, after implanting fever management guideline, IICP could be prevented. The case studies of Mr. A and Ms. B, showed the differences in pattern of fever and factors related to fever. Fever management intervention in a case of severe TBI (case Mr. A) was found to be poorly effective and difficult due to complications.

Mr. A had pattern of fever comprising very high BT, which fluctuated and persisted for many days. Within 72 hours of admission that patient had high grade fever probably due to the type of brain injury (SAH, frontal lobe injury, severe head injury) and related to neurogenic fever and inflammatory response after injury. Neurogenic fever occurs from direct disturbance of the hypothalamic "set point" of temperature, and leads to a rise in body temperature.[9] Injury involving SAH is associated with cerebral vasospasm. Patients with this injury may have generalized disturbance of thermoregulation which might be caused by hypothalamic dysfunction. In addition, the set-point elevation may be changed related to the frequent presence of thick clot in the suprasellar cistern or impaired heat dissipating mechanisms resulting from intense activation of sympathetic nervous system and generalized peripheral vasoconstriction.[25-26] In some cases, the neurogenic fever was shown to be persistent with fever continuing for more than 6 hours over 2 or more consecutive days.[27]

Another specific factor that created persistent of fever in this case was infection, specifically UTI. UTI related to fever has been found in around 20% in TBI patients.[28] However, the other factors need to be assessed since there are several factors related to fever (Table 3) to be controlled and prevented. Although it is a general cause, it can be aggravated fever in any time as shown in this case study.

Fever management of Mr. A, effected only a minimal decrease in BT in a few hours but with relapse to high body temperature. It reflected a lower effect of fever management intervention, which is incongruent with previous evidence that showed that the cooling device was effective when applied.[15] This may be due to the brain injury disrupting thermoregulation and patient still having infection. In addition, the limitation to using antipyretic drug in this case due to the high grade of liver injury, can result in early hyperthermia and persistent high BT which would be difficult to manage. The evidence has shown that the use of antipyretic drug with a cooling device is more effective for those who have intact thermoregulation. However, it may be more likely to be ineffective in TBI patient who has thermoregulatory setting impairment.[12] Moreover, the lower effect of fever management intervention in Mr. A may be due to guideline implementation, which will be described in the limitation of the study.

In contrast, pattern of fever for mild TBI (Ms. B) was different from that of Mr. A. These may have been no injury of the thermoregulation center. Since the major injury was to abdominal organs, the increase of body temperature may have been associated with tissue injury of those organs by trauma and surgery. The chemical mediators of inflammation (e.g. interleukin-1, prostaglandins) are released from injured cells and are carried from the blood circulation pass to the brain, then act on hypothalamic neurons, leading to increase in thermostatic set point which causes the fever.[29] Because of the different patterns of fever, it was easier to manage fever in Ms. B. The BT of Ms. B decreased to normal range as expected. Low grade fever from the non-serious cause of fever was reduced to normal when a combination of cold tepid sponging and cold pack at the important regions was used in this case. This result is consistent with the previous study by Aluka et al.[14] which evaluated the effectiveness of cold water sponging . However, that study was in pediatric patients and this is the reason for combination of cold water sponging with cold pack used on the adult patient in the current study.

However, some limitations when applying evidence-based fever management guideline. Firstly, the guideline was implemented in a short period of time. Secondly, some equipment were unavailable to be used such as the full set of blanket, then cold pack was used instead in helping cooling under the head and neck. Lastly, there was unable to control all activities in working environment such as co-worker, usual care, and room temperature in the setting.

Conclusion and Nursing Implication

Fever in TBI patients is one important factor that nurses are able to manage and reduce to prevent IICP. Fever may not be easily managed due to different associated factors. Understanding the pathophysiology and fever pattern is necessary. Findings showed that the different level of injury created different patterns of fever. Factors associated with fever need to be assessed for early management. Based on the findings, some implications for nursing practice are (1) nurses or physician should not ignore any factors related to fever, and (2) the assessment and monitoring of fever in TBI patients is necessary from the time of admission. In addition, understanding the pattern of fever may help nurses and physicians to detect and manage fever in time to receive treatment and prevent patients from developing a worse condition or IICP. Moreover, caring for TBI patients must include an interdisciplinary team to seek more control of the intervention and solve the problem as fast as possible and care should be integrated well with routine daily nursing care. The evidenced-based

fever management in this study could therefore be a useful tool to be used in nursing care for preventing IICP in TBI patients.

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