What is central fever- the definition

Central fever (CF) is defined as elevated temperature with no identifiable cause. It is basically a non –infectious source of fever in patients with traumatic brain injury.

Abstract:

Fever in patients with severe head injury is a commonly -encountered diagnostic and management problem. Neurogenic fever (NF) or central fever is a non-infectious source of fever in the patient with head injury and, if untreated, can cause damage to the brain in many ways. Until recently, NF was thought to be a relatively rare consequence of traumatic brain injury (TBI), but other studies have reported that four to 37 percent of TBI survivors experience this sequelae. Patients with TBI are immunocompromised to a certain extent and this predisposes them to sepsis, which should be a primary concern particularly in comatose patients. Central fever is essentially a diagnosis of exclusion. It is only when sepsis is excluded, can we consider central fever. Though in the acute phase of severe TBI, brain temperature is indeed higher than the core temperature, but that significance is uncertain with regard to outcome prediction, since there has been a paucity of work on the use of direct methods of brain temperature monitoring. In summary, the pathophysiology and management of NF is not well understood and needs more research and understanding for better management and a favourable outcome.

Risk Factors

Many patients experience early hyperthermia (at least one episode of body temperature > 38.5°C within the first two days) after traumatic brain injury.

There is an increased risk of development of NF among patients with severe TBI who had experienced either diffuse axonal injury (DAI) or frontal lobe injury of any form. Other risk factors predicting early hyperthermia include Glasgow Coma Scale score in the emergency department < 8, paediatric trauma score < 8, cerebral oedema or diffuse axonal injury on initial head computed tomography, admission blood glucose > 150 mg/dL (8.2 mmol/L), admission white cell count> 14,300 cells/mm3, and systolic hypotension.

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Pathophysiology

Cerebral temperature has been recognised as a strong factor in ischaemic brain damage.Fever is extremely common after acute cerebral damage, and cerebral temperature is significantly higher than body core temperature.' Body core temperature may markedly underestimate cerebral temperature, especially during the phases when temperature has the greatest impact on the central nervous system . TBI results in many different types of injury, and at this point, it is unclear if one particular type is associated with an increased incidence of NF. NF results from a disruption in the hypothalamic set point temperature, which results in an abnormal increase in body temperature, and is thought to be caused by injury to the hypothalamus. It may be the selective loss of warm sensitive neurons,the osmotic changes detected by the organum vasculosum laminae terminalis(OVLT) or the humoral changes modifying the firing rate of heat sensitive neurons in the medial preoptic nucleus.

Neurological Effect

The neurological 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 a rise in temperature because the intracranial blood volume increases with temperature. This reduces compliance and puts the brain at risk for further injury.Hyperthermia, from fever or other sources, when high enough (> 43°C), has been reported to cause neuronal injury in normal brains, and lengthy periods of moderate (40°C) hyperthermia have been reported to alter brain structure and functioning.Additionally, the TBI patients are at risk of secondary injury from fever because for every 1°C rise in body temperature, there is a 13% increase in the metabolic rate. This taxes the stressed energy reserves of the severely brain injured, catabolic patients. The higher metabolic demand of fever further exacerbates this problem, and can lead to additional loss of muscle and fat store.

Paroxysmal sympathetic hyperactivity is another source of hyperthermia in patients with TBI.

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Clinical features/Diagnosis

It is basically a diagnosis of exclusion and needs very thorough and detailed diagnostic work up of the TBI patient. Criteria for diagnosing central fever have been suggested-

1. It typically has a rapid onset with high temperatures(more than 39 degrees) and responds poorly to antibiotics or antipyretics.
2. No prior infections or fever at least 1 week prior to the event.
3. Negative work up for fever of infectious origin or drug induced fever.

The combination of negative cultures; absence of infiltrate on chest radiographs; diagnosis of subarachnoid hemorrhage, intraventricular hemorrhage, or tumor; and onset of fever within 72 hours of admission predicted central fever with a probability of .90.

The patient with NF are relatively bradycardiac, having a notable absence of perspiration, having a plateau -like temperature curve (no diurnal variation) that persists for days to weeks, the temperature being characteristically very high, and resistant to antipyretic medications. NF may be associated with the presence of prolonged unawareness or coma state and diabetes insipidus.This often leads to expensive, invasive, and often painful tests in order to make the diagnosis.' Differentiating a patient of NF from a patient who is having a true infectious or inflammatory source of the fever is a critical diagnostic decision for the clinicians caring for the TBI patients. The two treatment regimens differ significantly; thus rapid and proper diagnosis and treatment are essential for control of fever and optimisation of patient outcome following TBI.

Management

Rapid control of the fever is essential as it is associated with worsened outcome in both experimental and clinical studies. The treatment of NF includes use of both external cooling methods until the diagnosis is made and appropriate drug therapy. Many drugs which have successfully been used either anecdotally, or in case reports, to treat NF, include: bromocriptine,baclofen, amantadine, dantrolene, and propranolol. As each of these drugs has significant potential side effects (for example, hypotension and gastrointestinal bleeding), routine use without a relatively firm diagnosis of NF is not prudent.

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