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Acute Neuralgia in Infants can be managed with Cerebral Hypothermia and Temperature Control

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Abstract

In the adult intensive care unit, brain hypothermia (BHT) has been reported to be effective in neuroprotection after resuscitation and cardiac arrest. In contrast, in the neonatal intensive care unit, the pathophysiology of brain injury from hypoxic-ischemic encephalopathy (HIE) has been attributed to ischemia/reperfusion-induced circulatory impairment using neonatal brain cryotherapy. The International Liaison Committee on Resuscitation, 2010, recommends brain cryotherapy for HIE associated with severe neonatal pseudoparenchymal death. The utility of BHT for neuroprotection in infants and children, especially childhood acute encephalopathy, is expected. In theory, BHT could be useful in basic medical research and animal experiments. However, clinical plans for the treatment of pediatric acute encephalopathy have limitations. No international studies have been conducted and there is no clinical evidence of neuroprotection using BHT. This review describes the pathogenesis of neuronal injury in hypoxic and hypoperfused brains. History of BHT, its effects and mechanism of action. Methods of Cooling and Monitoring BHT; Side Effects of BHT. Review recent literature on target temperature management used to maintain and control body temperature in adults in the ICU. Finally, we discuss the development of BHT and targeted temperature management as a treatment for pediatric acute encephalopathy.

Keywords: Brain hypothermia therapy; Target temperature management; Neuronal protection; Hibernation

Introduction

Acute encephalopathy of childhood and adolescence refers to a rapidly progressive pathobiological condition of the brain. It is characterized by the central nervous system (CNS). Functional impairment caused by diffuse or extensive non-inflammatory cerebral edema [1]. According to the Japanese Society of Pediatric Neurology recommendations for acute encephalopathy in children and infants, disturbance of consciousness should last at least 24 hours and Glasgow Coma Score should be 11 or less [2].

Some encephalopathic disorders are multifactorial, but others, such as previous viral infections or hepatic or uremic encephalopathy, have well-established etiologies. Influenza virus is the most common pathogen in Japan, followed by human herpesvirus 6, rotavirus, and respiratory syncytial virus. In recent years, there has been an increase in the number of cases of acute encephalopathy associated with other viruses such as human metapneumovirus, rhinovirus, and cytomegalovirus. Among acute encephalopathic syndromes, acute encephalopathy with biphasic seizures followed by diffuse depression (AESD) was the most common type (29%) and Middle East respiratory syndrome (MERS) was the second most common type (16%).), acute necrotizing encephalopathy (ANE) was the least common type (4%). The other syndrome, occurring in 2% of cases, was hemorrhagic shock encephalopathy syndrome (HSES) [3]. Although the annual incidence is low, the number of cases of acute encephalopathy in Japan is significantly higher than in Western countries. Children infected with COVID-19 also developed encephalitis, ANE, acute disseminated encephalomyelitis (ADEM), cytotoxic lesions of the corpus callosum spleen, reversible hindencephalopathy syndrome (PRES), and other neurological deficits. There are also some reports that The major pathomechanisms of acute encephalopathy can be divided into four categories [4].

Metabolic failure, cytokine storm, excitotoxicity, and unknown mechanisms. Non-inflammatory cerebral edema is a pathological hallmark of acute encephalopathy. Non-inflammatory cerebral edema increases intracranial pressure and decreases cerebral perfusion pressure, ultimately leading to herniation syndrome and brainstem injury caused by CNS-mediated respiratory and circulatory disturbances. Regardless of the cause of the encephalopathy, all cases of acute encephalopathy have at least one symptom of altered mental status. Altered states of mind are subtle and can develop gradually as follows: B. apraxia, such as inability to draw simple pictures, or manifest and rapidly progressing and can lead to coma or death within minutes. Inattentiveness, poor judgment, and poor motor coordination are symptoms of a disturbed mental state. Seizures are common in many people, are often febrile, and may last for a long time (status febrile epilepticus) [5]. Febrile convulsions, febrile status epilepticus, and fever-associated febrile status epilepticus trigger the increasingly reported AESD, one of the most common forms of acute encephalopathy. However, despite the relatively high morbidity and mortality associated with this condition, there is little evidence for the recognition and treatment of acute encephalopathy. Additionally, neurological complications are common in patients with coronavirus disease 2019 (COVID-19). Childhood COVID-19-associated multisystem inflammatory syndrome (MIS-C) can lead to cerebrovascular events and abnormal eye movements. There have also been several reports of children infected with COVID-19 developing encephalitis, ANE, ADEM, and cytotoxic lesions of the corpus callosum [6].

The clinical course of metabolic disorders and inborn errors of metabolism can be characterized as insidious, progressive or static, followed by the development of acute encephalopathy, including lethargy,

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behavioral changes, or gait disturbances caused by infection or fasting. Certain treatments such as corticosteroids, immunoglobulins, free-radical scavengers, penetrants, immunosuppressants, plasmapheresis, and hypothermia have been extensively studied. This review provides an up-to-date summary of available data and perspectives on targeted hypothermia-based management of pediatric acute encephalopathy. Target hypothermia in acute patients from 1 September 2000 to 1 September 2022 using the electronic medical database MEDLINE/PubMed and the National Institutes of Health Clinical Trials Registry (accessed 1 September 2022) We searched for therapeutic publications and conducted a literature search. Identify encephalopathy [7]. The article was included when published in English. Keywords include cerebral hypothermia, targeted temperature management, therapeutic hypothermia, induced hypothermia, acute encephalopathy, pediatric, pediatric, preschool, neonatal, infant, acute febrile encephalopathy, and status epilepticus. will be Randomized clinical trials, reviews, and other study design abstracts and full-text articles were considered from studies describing relevant data on pediatric acute encephalopathy. An additional search was performed via Google Scholar to include relevant articles on prospective and retrospective designs as well as real-world data on pediatric acute encephalopathy [8].

Therapeutic hypothermia

Hyperthermia is considered an independent factor for the poor prognosis of acute encephalopathy. Therefore, numerous pharmacological and non-pharmacological strategies, such as B. Target temperature management (TTM), are reported when general anesthetics fail. Neonatal cerebral hypothermia (BHT) only cools the head. Because brain temperature is reported to be 1–2 °C higher than core body temperature in acute encephalopathy, we performed BHT for 48 h using a whole-body cooling system to regulate body temperature to 33.5 °C–35 °C. Since the surface area of the head becomes smaller as the child grows, both head cooling and whole body cooling are used to lower the body temperature more efficiently. In recent years, attention has been focused on mild BHT treatment that keeps body temperature between 34.5°C and 36°C. In addition, the hospital developed his BHT strategy for treating acute encephalitis or encephalopathy and status epilepticus in children. Regarding the long-term effects of AES, hypothermia can help prevent the development of post-encephalopathy epilepsy, thereby improving the patient's quality of life [9].

BHT is effective in neonates with a large head surface area, but adolescents may require whole-body cooling as an effective means. Finally, in the field of emergency medicine, TTMs have been proposed that are controlled to temperatures not as low as BHT. TTM is defined as temperature management (34.5°C to 37°C) with intubation and continued use of anticonvulsants and muscle relaxants used within 24 hours of symptom onset. According to numerous case reports and retrospective studies, the neurological outcome of acute encephalitis or encephalopathy can be significantly improved by focused her TTM. TTM is widely used in neurocritical care to minimize secondary nerve damage and improve outcomes.

Brief history of therapeutic hypothermia

People have long known the use of alternative medicine to cool wounds, heat, and reduce inflammation. Hypothermia was used medically by the Egyptians 5000 years ago. It was first mentioned in the Edwin Smith Papyrus, an ancient medical treatise on medicine and surgery written around 3500 BC. Hippocrates, who advocated burying wounded troops in snow, introduced the concept that cooling slows down biological processes and ultimately leads to death. Cooling the body as a therapeutic intervention became widespread in his 17th century, when physicians such as John Froyer (1649–1734) began extensive experimentation with the use of hot and cold water in medicine. With the use of cryo-analgesia in amputations around the 19th century, surgeons discovered that the analgesic effect of cryo-analgesia was associated with reduced bleeding. Local head cooling for traumatic brain injury was used in the late 1800s, and whole-body cooling was first documented to treat head injuries [10].

Neuronal damage in hypoxic and hypoperfused brain tissue: pathogenesis

In addition to the normal environment, the brain requires optimal pH, temperature, blood flow, and osmolarity to perform its functions. However, small disturbances in this complex system can have a large impact on brain function. One of the putative etiologies of encephalopathy is impaired glucose or oxygen supply to the brain. Encephalopathy and eventually coma can occur because the delivery of substances to the brain is greatly reduced. Hypoxia, especially hypoxia-ischemia, can permanently damage delicate areas of the brain such as the thalamus, hippocampus, and cerebellum. The extent and irreversibility of injury depend on the extent and duration of hypoxia or reduced cerebral perfusion. HIE, which is usually caused by severe hypotension or cardiac arrest, is triggered by a neuronal ischemic injury cascade that includes excitatory amino acid release, intracellular calcium influx, lipid peroxidation, and cell death.

Gaps and future directions

In low- and middle-income countries, therapeutic hypothermia had no effect on the composite outcome of mortality or disability 18 months after neonatal encephalopathy, but alone significantly increased mortality. Although tertiary neonatal intensive care units are available, hypothermia should not be used to treat neonatal encephalopathy in low- and middle-income countries. According to a recent meta-analysis, hypothermia in neonatal encephalopathy reduces neurological deficits and cerebral palsy, but its effects on neonatal, infant, and child mortality remain unclear. The environment in which it runs affects the results. Low-quality studies overestimated the potential benefits of hypothermia. BHT requires systemic management at the PICU level. Steroid pulse therapy, globulin therapy, and intravenous therapy with various antiepileptic drugs are commonly used, sometimes in combination with BHT (25). To confirm the clinical outcome of this treatment, the clinical course of treatment should be assessed by dividing cases into two groups based on the treatment received taking BHT, taking BHT and concomitant medications. The number of studies on this topic is limited. However, informed consent is a key component of his BHT research.

Conclusions

The target temperature for treating acute encephalopathy depends on the underlying cause of the condition and the severity of symptoms. The use of target temperature management for acute encephalopathy should be individualized to each patient's specific needs and medical history and should only be performed under the guidance of a qualified healthcare provider. We hope to establish a more ideal systemic management of pediatric acute encephalopathy by using BHT and TTM in different or combined cases.

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