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Review Article

Long-term prognosis and predictors of outcomes after childhood convulsive status epilepticus

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ABSTRACT

Objective: Childhood convulsive status epilepticus (CSE) is widely known to be associated with short-term and long-term mortality and morbidity, but the role of CSE itself on adverse outcomes is debatable. The additional effect of CSE characteristics on outcomes after CSE and whether prolonged seizures cause any long-term hippocampal injury which leads to developmental or memory impairment is uncertain. This review provides an overview of long-term prognosis after childhood CSE, highlighting data from recent literature.

Findings: In previously normal children, the long-term prognosis after childhood CSE is favorable, with low incidence of epilepsy, motor, and cognitive difficulties. Mesial temporal sclerosis is uncommon in children after prolonged febrile seizures. In children with symptomatic causes and those with pre-existing neurological abnormalities, there is substantial morbidity after childhood CSE. Etiology is the primary determinant of outcome after childhood CSE and the additional effect of CSE characteristics such as seizure duration seems to be less than previously believed.

Keywords: Childhood, Status epilepticus, Prolonged febrile seizures, Long term, Prognosis, Outcomes

INTRODUCTION

Convulsive status epilepticus (CSE) is the most common medical neurological emergency in children, with an overall estimated incidence of 20/100,000 children per year.[1-3]

The definition of CSE has evolved over time, with the commonly used definition of CSE as a seizure lasting for a duration of 30 minutes or longer. Recently, the International League Against Epilepsy Task Force on classification defined status epilepticus (SE) as "a condition resulting either from the failure of the mechanisms responsible for seizure termination or from the initiation of mechanisms which lead to abnormally prolonged seizures (after time point t1). It is a condition that can have long-term consequences (after time point t2), including neuronal death, neuronal injury, and alteration of neuronal networks, depending on the type and duration of seizures."[4] The Task Force reached a consensus opinion that time point t1 (time for initiation of treatment) should be at 5 min and time of t2 (indicating the potential threat of brain damage in humans after this time point) at 30 min for generalized convulsive seizures, and t1 of 10 min and t2 of >60 min for focal SE with impaired consciousness.[4]

In a landmark study published in 1970, Aicardi and Chevrie reported the outcome of CSE in children as "grave, mental, or neurological residua or both being present in at least 57% of patients."[5] Subsequent prospective studies, however, have reported better outcomes.[6] This may

be a reflection of improvements in emergency management and intensive care for children presenting with CSE and also a reduction in the incidence of bacterial infection-related acute symptomatic seizures as a result of universal immunization in recent decades. The long-term sequelae after childhood CSE may include neurological, cognitive, and behavioral impairments and impact on quality of life.[7,8] However, the role of CSE itself on the sequelae and whether the outcomes are influenced by the underlying etiology, patient's age, the type of CSE, and duration of CSE is debatable. Furthermore, while there is some evidence to suggest that prolonged febrile seizures (PFSs) may result in short-term hippocampal injury leading to developmental or memory impairments, whether these changes then lead to the development of mesial temporal sclerosis (MTS) and/or temporal lobe epilepsy (TLE) is uncertain.[7,9-11]

The aim of this review is to provide an overview of the long-term prognosis and the predictors of adverse outcomes following childhood CSE.

LITERATURE SEARCH

We performed a search on PubMed for original articles published up to November 30, 2020, with the search term "status epilepticus" combined with the terms "prognosis," "outcome," "mortality," "fatality," "death," "morbidity," "recurrence," "cognition," "MTS," "hippocampal sclerosis," and "quality of life." The search was also done with the terms "prolonged febrile seizures" and "prolonged febrile convulsion." The references of identified articles were examined for additional relevant studies. We only included the studies reported in English and included patients between the ages of 1 month and 18 years at the time of SE.

PROGNOSIS IN CHILDREN AFTER CSE

It is widely known that CSE is associated with increased mortality and morbidity. The long-term adverse outcomes reported in children that have previously had childhood CSE include epilepsy, MTS, CSE recurrence, motor and/or intellectual disability, behavioral impairments, and impact on quality of life.

MORTALITY

Short-term mortality

Short-term or immediate mortality in CSE is defined as mortality within the first 30 days after SE. In studies reported before the year 2000, the short-term mortality rates were ranging from 3% to 9%, whereas after 2000, the rates are significantly lower, with the more recent studies reporting rates below 1%.[1,12-15] The lower rates in the newer studies are most likely due to the advances in intensive care and out-of-hospital emergency management of CSE and the reduction in the incidence of CSE due to acute symptomatic causes such as bacterial meningitis. Furthermore, it is important to bear in mind that most of the older studies of CSE do not specifically investigate children. CSE associated mortality increases with age in adults and hence can result in a higher case fatality rate in these studies.[16,17]

Long-term mortality

The cumulative mortality rates range between 5.4% and 13% within 10 years after CSE as reported in population-based studies.[18-21]

A prospective pediatric population-based study from North London, United Kingdom, reported an overall case fatality rate of 11% within 8 years after CSE. Among 226 children in the study, 7 (3%) died within 30 days of CSE and 16 (8%) died during follow-up.[1,21] A more recent retrospective hospital-based study followed up 460 children for a median duration of 63 months reported mortality of 3.8%.[22]

Risk factors for death

In the North London study, the majority of deaths were as a result of complication of their underlying medical condition, while in about a quarter, the deaths were associated with either CSE or intractable epilepsy. The main risk factor for mortality was the presence of pre-existing clinically significant neurological impairments. Of note, no deaths were reported following idiopathic CSE and PFS, which is consistent with previous reports suggesting that SE in itself may be less harmful.[21]

There are several studies which report higher risk of death in children <1 year of age[7,18,23] while some other studies have shown no association between age and mortality.[15,21] The higher prevalence of death in younger children below 1 year of age may indicate the high incidence of acute symptomatic CSE in this age group, in keeping with previous observations of higher mortality due to acute symptomatic etiology. Although in some studies, a direct association between longer duration of CSE (>24 h) and higher mortality has been reported, it is highly likely that longer duration of CSE is a marker of the severity of underlying cause which increases the risk of death.[18,24]

Therefore, death in children with CSE is most likely due to complications of their underlying brain disorder rather than due to prolongation of seizure.

SUBSEQUENT EPILEPSY

The risk of subsequent epilepsy 2 years after the first episode of unprovoked CSE is 15–40%, which is similar to the risk of seizure recurrence after first self-limited short unprovoked

seizure.[7,25-27] In the North London study, in children with CSE, the cumulative incidence of epilepsy was 24.7%, with nearly 90% occurring within 18 months of CSE.[27] Focal epilepsy is the most common type. Unilateral CSE with slight fever could be initial presentation of Dravet syndrome or a focal structural lesion.[5,28,29] The incidence of subsequent epilepsy, however, varies depending on the cause of CSE. In the North London cohort, the incidence of subsequent epilepsy was 14.3% after PFS, 13.3% in acute symptomatic CSE, 45.5% in remote symptomatic, and 50% in unclassified CSE. Absence of fever was the only significant predictor of incident epilepsy with an odds ratio of 7.5 (95% confidence interval 2.25–25.1).[27]

Overall, the long-term risk of developing epilepsy after PFS or febrile CSE ranges between 4% and 15%.[19,27,30-32]

The hypothesis that PFS or febrile CSE has a causal role in the development of MTS and associated TLE is derived from retrospective studies from tertiary epilepsy centers (thus potentially subject to selection bias).[33-36] However, prospective and population-based studies have failed to demonstrate a causal association.[19,31,37-40] Although prolonged seizures may result in acute hippocampal injury, there is not sufficient evidence to suggest that this results in long-term consequences such as MTS and/or TLE.[7,9,11,27,41-44] Overall, there is low incidence of TLE (<6%) and MTS (<7%) after PFS which implies that although PFS might increase risk of hippocampal injury in those with pre-existing abnormalities, the direct contribution of PFS in development of MTS, TLE, or both seems less than has long been believed.[9,11,27,31,43-45]

RECURRENCE OF CSE

The estimated overall risk of recurrence of CSE in children ranges between 10% and 70.5%.[7,22,27,46] The risk of CSE recurrence within 8.9 years was 43.3% due to all causes of CSE in the North London study.[27] In another retrospective hospital-based study from the United States, the recurrence rate of CSE was as high as 70.5% with a median follow-up period of 63 months, which may be due to the study design and recruitment bias.[22]

The main risk factor for CSE recurrence is etiology and presence of neurological abnormality at baseline.[22,27] In the North London cohort, children with pre-existing neurological abnormality were 3.8 times (95% CI 1.8–8.0) more likely to have a CSE recurrence during follow-up. In addition, compared to children who presented with CSE as first episode, children who had already had previous CSE at baseline were 4.5 times (95% CI 1.8–11.1) more likely to have CSE recurrence during follow-up. In the PFS group, 10 children (29%, 95% CI 16–45) had CSE recurrence, and of these, four had epilepsy diagnosis during follow-up.[27]

MOTOR AND/OR COGNITIVE DISABILITY

Children with a history of CSE have a higher prevalence of neurologic disability including focal neurologic deficits (e.g., hemiplegia, diplegia, extrapyramidal syndromes, and cerebellar syndrome). It is often difficult to determine whether reported motor and/or cognitive impairments are new or predates the CSE episode as most studies do not report them separately.[7,46] Furthermore, in most studies, cognitive outcome is determined by no formal neurocognitive testing or using global measures such as full scale intelligence quotient, which are not designed to identify specific problems such as memory impairment or dyslexia.

Overall, the risk of neurological sequelae within 5–10 years after CSE ranges between 14% and 37%, with most of the high-quality studies reporting rates of <15%.[7,14,19,27,47-54] In the North London study, new motor disability was observed in 2.1% and intellectual disability in 8.8% following CSE due to all causes.[27] At follow-up, the prevalence of motor and intellectual disability was 30.6% (95% CI 23.4–38.8) and 45.5% (95% CI 37.3–54), respectively.[27]

Etiology was the main determinant of neurological morbidity after CSE. In the North London study, no child with PFS and acute symptomatic CSE developed motor disability and one child who had PFS developed intellectual disability. Motor and intellectual disability was seen predominantly in remote symptomatic CSE and idiopathic and cryptogenic CSE group.[27] Data from the same cohort showed that children classified as non-PFS at baseline have a worse cognitive outcome associated with the presence of cognitive delay pre-CSE, whereas there were no long-term memory impairments in children with a history of PFS.[55] In another retrospective hospital-based study, about 29% of children with reportedly normal development at baseline had developmental regression or significant cognitive impairment at follow-up.[22] Similar to the results of the North London study, symptomatic etiology, developmental delay at baseline, and abnormal brain magnetic resonance imaging were associated with increased risk of abnormal neurocognitive outcome.

BEHAVIORAL IMPAIRMENTS

It is difficult to determine the impact of childhood CSE on behavioral impairment and psychiatric morbidity separate from other neurological outcomes as it is often not reported in most studies and where reported it is uncertain whether they are new or precede CSE episode, and also if these are due to the underlying etiology of the CSE or a consequence of the CSE itself. The population-based North London CSE in childhood study examined the long-term behavioral and psychiatric outcomes using standardized questionnaires and neuropsychiatric assessments in a cohort of 134 children with CSE.[56] After a mean follow-up of 8 years, 37% had behavioral

problems and 28% had a psychiatric disorder. Fifteen of these (11.2% of total CSE cohort) were either newly diagnosed, had an additional diagnosis or revision of their diagnosis, which indicate that children with behavioral difficulties may go undetected by caregivers and the professionals. Seizures before CSE and recurrent CSE increased the risk of adverse behavioral outcomes. Of note, 8 (21.6%) children who had a psychiatric diagnosis did not have epilepsy, and hence, their behavioral difficulties cannot be attributed to ongoing epilepsy and/or the use of antiepileptic medications. These data indicate that children with CSE in childhood may often have behavioral and psychiatric impairments several years after the episode and hence require screening.

QUALITY OF LIFE

Health-related quality of life (HRQoL) is very important outcome measure that determines the quality of an individual's well-being by looking at multidimensional aspects of a person's life such as physical and social function, behavior, cognition, and emotional well-being, which are difficult to quantify.[57] Despite considerable morbidity in this population, there are very few studies addressing quality of life in children with CSE.

A population-based long-term prospective study of adults with childhood-onset epilepsy demonstrated no significant difference between those who had experienced CSE in childhood and those who did not in relation to educational attainment and employment, marital, and socioeconomic status.[43] In contrast, a more recent cohort study of children with newly diagnosed epilepsy reported that children with CSE have significantly poorer HRQoL as compared to children who did not have CSE and that this factor is independent of other factors (demographics or clinical features) which are known to affect HRQoL in childhood epilepsy.[8] However, follow-up of this cohort showed that the short-term poorer HRQoL may resolve over long term with similar HRQoL for children with and without CSE at 10 years follow-up.[58]

PREDICTORS OF LONG-TERM PROGNOSIS AFTER CHILDHOOD CSE

It is now clearly established that etiology is the most important predictor of long-term mortality and morbidity after childhood CSE.[7,19,21,27,46,55,56] In children with symptomatic causes and pre-existing neurological abnormalities, there are increased mortality and higher incidence of neurological sequelae, whereas in previously neurologically normal children, the incidence is low. The additional effect of CSE characteristics such as younger age at CSE, focal seizure onset, and seizure duration on subsequent outcomes is uncertain due to the inherent difficulty in separating the effect of CSE itself from its cause.

CONCLUSION

The data from long-term follow-up studies, albeit limited, are reassuring and suggest that the long-term prognosis is favorable in previously normal children after childhood CSE. While there are considerable mortality and morbidity associated with childhood CSE, it is mainly seen in children with symptomatic causes and pre-existing neurological impairments. Recurrence of CSE is predominantly seen in children with previous neurological abnormalities. Etiology is the primary determinant of outcome, while the role of CSE characteristics such as seizure duration seems less than previously believed. With the change in the definition of CSE, future studies investigating outcomes in children with prolonged seizures (>5 min) may help determine whether earlier cessation of seizures (<30 min) may result in better outcomes.

Declaration of patient consent

Patient's consent not required as there are no patients in this study.

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Conflicts of interest

There are no conflicts of interest.

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