SEIZURES FOLLOWING RESPIRATORY VIRAL INFECTION

CORNELIA CĂLCÎI[1], SVETLANA HADJIU[2], MARIANA SPRÎNCEAN[3], NINELI REVENCO[4], LUMINIȚA DOBROTA[5]

[1,2,3,4] *"Nicolae Testemițanu" State University of Medicine and Pharmacy, Chișinău, Republic of Moldova,* [1,2,3,4] *Institute for Maternal and Child Healthcare, Chișinău, Republic of Moldova,* [5] *"Lucian Blaga" University of Sibiu*

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Abstract: *A Febrile seizure (FS) is a disorder that occurs in children between 6 months and 5 years of age, in association with fever but without evidence of intracranial infection. FS may be of any type, although they are usually generalized tonic-clonic or tonic, and are classified as complex if the seizure duration is longer than 15 minutes, if more than one seizure occurs in 24 hours, or if focal features are present. Electroencephalography (EEG) has not been found to be useful in the evaluation of a child with febrile seizures, but some authorities believe that the EEG is a poor predictor of either febrile or afebrile seizures recurrence. The most urgent diagnostic decision is whether to do a lumbar puncture. The computed tomography (CT) scan or magnetic resonance imaging (MRI) are not warranted in the evaluation of FS. Because EEG is of questionable value after FS, routine EEG is not necessary.*

INTRODUCTION

Seizures in acute viral infections are defined as seizures that occur in children aged 6 months to 5 years in association with fever, called febrile seizures (CF), and are not the result of a cerebral pathology (eg. neuroinfections, head trauma and epilepsy). They are also not associated with any of the extra-cerebrovascular causes (electrolyte imbalance, hypoglycemia, drug use or drug withdrawal) and afebrile seizures. Febrile seizures are a challenge in pediatric practice because of their increased incidence and tendency to recurrence..(1) In recent years, several complications that may occur after such event are described, and new strategies are addressed for their assessment. Guides of the American Academy of Pediatrics (2011) and the Japanese Society of Pediatric Neurology (2016) (2,3) have been published. Most children with febrile seizures have an excellent prognosis and few develop long-term health problems. Diagnosis of CF is clinical and most importantly, to exclude intracranial infections, especially after a complex CF.(2) Management consists of controlling the symptoms and treating the cause of fever. Parents and caregivers are often embarrassed and frightened after an episode of CF and must be properly informed and guided about the management of their child's fever by health professionals.(3)

The purpose of this study is to evaluate international guidelines for a correct approach among specialists involved in pediatric care.

Epidemiology. FS is quite common in pediatric practice, with a frequency of 2-5% between 6 months and 5 years in the US and European countries, with a peak incidence between 12-18 months.(1) They are found in all ethnic groups with a higher frequency in the Asian population (5-10% in India and 6-9% in Japan).(3) The boys /girls ratio is around 1.6 to 1. This syndrome is more common in children from a precarious socioeconomic environment, possibly due to a lack of population access to healthcare. Seasonal and diurnal variations have been observed by researchers in the US, Finland, Japan, and the prevalence of febrile seizures occurring in the winter months in the afternoon.(4)

Etiology and pathogenesis. It is known that FS results from the vulnerability of the central nervous system (CNS) to fever, in combination with genetic predisposition and environmental factors. CF is a response of age-dependent CNS to fever. This explains the increased frequency of convulsions before the age of 3 when the seizure threshold is low.(5) Thus, the mechanism of FS production is a state of physiological hyperexcitability under low convulsive threshold conditions. Genetic determinism is a positive family history of 25-40% of affected children.

Molecular genetic studies suggest that genetic factors play an important role in the development of FS. Approximately one third of all children with CF have a family positive history. The risk of febrile seizures is 20% when there is an affected brother and 33% when there are parents who have suffered from the same problem. The genes that may be involved were located in chromosomes 1q31, 2q23-34, 3p24.2-23.3q26.2-26.33, 5q14-15, 5q34, 6q22-24, 8q13-21, 18p11.2, 19p13.3, 19q and 21q22. Several models of genetic transmission are argued: autosomal dominant and polygenic or multifactorial.(6) It has been proven that for the development of febrile first-degree access, fever can be a more significant risk factor than the rapidity of its increase. Children with febrile seizures have a low seizure threshold. A viral infection is a cause of over 80% of CF. Rubeola, flu and other viruses are other causes in FS.(5)

The risk of FS is temporarily increased for a few days after administration of vaccines, especially the combination (diphtheria-tetanus-pertussis), and the pneumococcal conjugate vaccine and Fluvax inactivated vaccine (Fluvax).

Premature babies are more prone to FS and postnatal treatment with corticosteroids may increase this risk. Prenatal exposure to alcohol and nicotine may also be a cause of the onset of FS. Among other risk factors, some studies include: deficiency in Zn, vitamin B12, folic acid, selenium, Ca, Mg, CF, retardation in intrauterine development, retention in neuropsychiatric development.(3)

There are 3 main factors that can cause febrile convulsions: fever, age and genetic predisposition.(6)

Risk factors that can be associated with the onset of a

[5]Corresponding author: Luminița Dobrotă, Str. Lucian Blaga, Nr. 2A, Sibiu, România, E-mail: luminitadobrota@yahoo.com, Phone: +40722 501145
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FS include:
- grade I or II with a history of FS or afebrile seizures
- poor care conditions
- neonatal retardation> 28 days
- retard in progression
- very high fever
- viral infections (influenza A, human-6 herpes virus, metapneumovirus)
- iron deficiency anemia
- a low serum concentration of Na
- difficulty breathing
- neonatal asphyxia
- umbilical blood circulation
- vaccination - diphtheria-tetanus-pertussis (DTP), measles, rubella, varicella.

The most common infections associated with FS in children are varicella, influenza, middle ear infections, upper and lower respiratory tract infections (such as tonsillitis, pneumonia, bronchitis, and sinusitis), dental infections, gastroenteritis (especially those caused by rotavirus).(7)

Clinical manifestations. In most cases, FS is encountered during the first days of fever. Crises occurring at a distance > 3 days of fever are suspect. Most often, fever is > 39. FS are classified into simple and complex, based on duration, physical characteristics and the pattern of recurrence. The most common are simple FS, which occurs in the proportion of 80-85% of cases.(2) Loss of consciousness at the time of crisis is a constant factor. Salivation, breathing disorders, pallor, or cyanosis, are also found in the simple FS. They can also be accompanied by tonic-clonic movements and eye-fixing. Their duration may be up to 15 minutes (most frequently up to 5 minutes), followed by short drowsiness and not repeated within 24 hours. Atonic and tonic seizures are described. Complex FS has a duration of more than 15 minutes (called status epilepticus), the crises being focal and can be repeated over the same day. They may be associated with a postictal hemiparesis (Todd's paralysis). Usually, these children are younger and may show a delay in neuropsychological development (table no. 1). Children with febrile status epilepticus frequently have structural changes in the hippocampus and a higher risk of developing epilepsy.(3)

Table no. 1. Clinical classification of FS

Characteristics	Simple FS (95%)	Complex FS (5%)
Description of crises	Generalized tonic-clonic convulsions without focal traits	There are focal features of crises involving, for example, only a part of the body
Duration of crises	Less than ten minutes (less than 5 minutes)	More than 10 minutes
Repeating crises	There is no recurrence within 24 hours (one crash/24 hours)	Two or more seizures occur within 24 hours
State of consciousness	Normal	No full recovery after 1 hour
Neurological consequences	Absent	There are neurological, post-ictal consequences
Neurological deficiency	Absent	There is a short period of paralysis, defined as Todd's paralysis after a feverish appearance
Epileptic status risk (SE)	Around 5‰ (= general population)	SE is developed
Family history	Negative for epilepsy, ± positive for CF	Positive for idiopathic or genetic epilepsy
The state of neurological health	Normal	Well defined neurological abnormalities
Age	Between 6-36 months	Frequently less than 1 year
EEG	Not indicated. Usually normal after 7 to 10 days of FS	Clear focal or generalized irritation abnormalities
Need for Anti-epileptic Drugs (DrAE)	Convulsions are solved spontaneously	DrAE can be administered to interrupt the crisis
Risk of epilepsy development	1%	4 – 6%

Clinical evaluation. A detailed anamnesis is required to determine the cause of fever, the relationship between fever and seizures, the characteristics of fever, of seizure, and the duration of sedative drowsiness. The history must also contain information about the recent vaccination of the child or if he/she is receiving antibacterial treatment. Similarly, elements such as toxin ingestion, craniocerebral trauma, neuropsychological development of the child, and the history of seizures of the affections to other members of the family should be considered.(1)

At the same time, vital signs and a complex physical examination to determine the causes of fever should be monitored. It is also important to investigate meningeal signs (Brudzinski, Kernig, Lesaj), especially in children less than 6 months of age. A lumbar puncture may be required if necessary. An examination of the muscular tone, osteotonic reflexes, is needed to exclude signs of an outbreak. The ophthalmoscopic examination must also be performed to exclude the increase in intracranial pressure. Neurocutaneous stigmas need to be examined to exclude facomatosis as a cause of convulsions (hypopigmented spots, Lisch nodules, neurofibromas, etc.).(2,3) When a child with FS comes to the emergency department, it is important to collect the following data: a detailed and accurate history, complete clinical evaluation, including a neurological examination, to exclude some secondary causes of seizures and meet the mandatory steps in FS patient behavior, which are set out below.(8)

Determining the degree of harm to the child:
- state of consciousness
- infectious impregnation (fever, nausea, vomiting, anorexia, asthenia etc.)
- convulsive manifestations: generalized seizures: tonic, tonic-clonic, clonic, atonic, and focal or unilateral seizures)
- outbreak neurological manifestations
- cardiovascular manifestations
- neurovegetative manifestations (respiratory, arrhythmia, cyanosis, vasomotor)
- complications (trauma by falling or wounding by surrounding objects, aspiration of fluid in the airways, side effects of the drugs used, status epilepticus, acute cerebral edema, decortication, deception, epilepsy, cerebral palsy, death).(1)

Table no. 2. "Red flags" for children who show febrile convulsions

Complex febrile convulsions
Positive meningeal signs
Alteration of consciousness over a period of more than 1 hour from the onset
A tense anterior fontant
Tachycardia that is not correlated with fever figures
Signs of severe respiratory illness: tachypnea, reduced oxygenation, chest compartmentation breathing.

Recommendations in collecting anamnesis:
- the onset of fever illness (acute or insidious)
- signs of fever illness associated with seizures (nasal congestion, rhinorrhea, ophthalmia, headache, sore throat, polypnea, abdominal pain, vomiting, diarrhea (mucopurulent stools with blood strips)
- the onset of the seizure
- the duration of the seizure
- description of the seizure (type, location)
- associate (or not) with loss of consciousness

- presence/absence of spontaneous urination
- biting the tongue
- the length of the postcritical period
- transient or permanent motor deficiency (Todd's paralysis) in the post-critical period
- post-clinical neurological manifestations (the child is more "soft", non-receptive to play)
- repeating the seizure crisis the same day
- highlighting risk factors for the recurrence of the seizure crisis: young onset of onset; a history of FS in relatives; short duration of fever before the first seizure; relatively low fever at the time of the initial seizure crisis; a possible family history of afebrile convulsions
- chronic diseases in the past
- past recurrent infections
- previous convulsive crises
- antecedents of pre~, intra~ and postnatal suffering (hypoxia, obstetrical trauma etc.). (2, 3)

Diagnostic criteria:

- fever, over 38.5 °C
- motor manifestations: tonic, clonic, tonic-clonic, myoclonic
- changes in breathing, apnea or irregular, noisy breathing
- central cyanosis
- loss of consciousness
- postcritical hay
- variable length: tens of seconds - tens of minutes.

Objective Exam:

- intensity of fever
- consciousness (coma)
- signs of meningeal irritation
- tension of the fontanel
- outbreak neurological signs (difficult unilateral motor)
- injuries of the head (suggestive of trauma)
- tachycardia, hypotension (sepsis)
- tachypnea (pneumonia).

***Differential diagnosis*.** It is necessary to make a differential diagnosis with

- febrile shivers (characterized by rhythmic movements, oscillators without loss of consciousness)
- febrile delirium (acute and transient confusion associated with high fever, EEG: retardation in the delta frequency band for several days)
- febrile syncope (may be triggered by fever, fear, emotion, cardiac pathology)
- lots of complained spasm (1-3 years occurs, is manifested by apnea, cyanosis, complained triggered by hypertonic, may be accompanied by tonic-clonic seizures)
- anoxic reflexive convulsions (children become suddenly cyantotic due to painful events or shocks)
- breathing attacks (children voluntarily retain their breathing and gradually lose consciousness)
- intoxications (with organophosphorus)
- deficiency of electrolytes (dehydration), rachitogenic tetania (spring, accompanied by other signs of rickets)
- Sandifer syndrome (opistonus caused by gastroesophageal reflux)
- vertigo paroxismal benign
- CNS infections - meningitis, encephalitis, cerebral abscess (sometimes difficult to diagnose because 40% of cases have no meningeal signs)
- genetic epilepsy with febrile plus (GEFS +)
- status epilepticus refractar
- epilepsies associated with febrile infections (FIRES)
- the evolution of epileptic syndrome in epilepsy: fever triggers episodes of seizures.(1,5)

GEFS+ syndrome is a syndrome with autosomal dominant with at least six phenotypes caused by mutations in the respective genes (SCN1A, SCN2A, SCN1B and GABRG2). Compared to simple febrile access, GEFS+ syndrome can also be encountered over the age of 6, and is also associated with myoclonic, atonic, or absent accessions. FIRES syndrome develops in the context of a febrile infection which starts from 24 hours - 2 weeks usually to onset refractory status epilepticus, no age limit. It is difficult to distinguish the first access as an onset of epilepsy or syndromes GEFS + FIRES, which need to be evaluated over time, just by performing genetic profile.(2,3)

***Diagnosis evaluation*.** In case of simple febrile seizures without clinical alterations, no laboratory investigations are required. Biochemical analyzes for glucose, electrolyte, urea, calcium, phosphorus and magnesium are usually not required in the patient's assessment of complex FS and are only important when the child has shown signs of dehydration and edema. Glucose and electrolyte (Ca, Mg) - prolonged convulsions and postcritic drowsiness.(9) The lab exam must be individualized, guided by history and physical exam results. It is necessary to perform general blood analysis to determine the origin of the fever. It is also necessary to perform general urine analysis if the origin of the fever is not known. Lumbar puncture (LP) is recommended in children younger than 12 months (American Academy of Pediatrics), especially in children who have not been vaccinated with Hib or Streptococcus pneumoniae.(2,10) This should also be done in the presence of meningially signs and in febrile status epilepticus. Pleocytosis, low glucose levels and high levels of protein in the CSF are specific for meningitis, requiring isolation and culture identification. Thus, the CSF exam is indicated in the following situations: 1) in the infant (in children <6 months - mandatory) in the first episode of FS; 2) in children > 1 year (in children <18 months recommended), if there are signs of meningian irritation or when there is at least suspicion of meningitis. CSF may be normal at the onset of a bacterial meningitis, it is recommended ± repeat LP. Relative contraindications for LP: septic shock symptoms; clinical diagnosis of invasive meningitis with haemorrhagic rash; high intracranial pressure (IP) with edema - brain swelling; neurological symptoms of herniation in the posterior fossa of the brain.(10)

There are no specific changes on the EEG and this examination is limited to prediction of febrile access recurrence. Routine EEG is not needed to evaluate a child with FS. EEG should be performed in children with complex FS, status epilepticus at onset, or in CF association with afebrile, in the presence of a prolonged post-critical neurologic deficit, previous neurological abnormalities. The Cochrane Revision of 2017 did not show any randomized clinical trials that would indicate the need to perform EEG in complex febrile seizures and the distance to which it should be performed. The EEG examination may present: 1) Slow, focal abnormalities indicating a focal, acute illness (encephalitis, cerebral infarction, cerebral tumor, stroke); 2) Generalized slow anomalies indicating a diffuse process (encephalitis, cerebral edema, persistent cerebral anomaly, poststroke); 3) Localized spikes indicating a probable location of convulsive activity; 4) High-wave discharges, considered epileptogenic; 5) Paroxysmal anomalies, which are more specific to children with complex FS.(2,3)

Neurosonography, CT and MRI are not routine exams for febrile seizures, but may be helpful later. These should be done with the association of signs of a prolonged postcritical neurological deficit and signs of intracranian pressure, previous neurological abnormalities, complex FS, SE.(8,10)

Other investigations: simple skull radiography, ophthalmoscopy, biological examinations (hemograms, blood glucose, calcemia, magnesia), toxicological screening (lead,

aspirin, tricyclic antidepressants, etc.), consultations of specialists from other fields.(1,3)

Red flags in FS:

- the child has prolonged convulsive access
- the child presents complex FS
- the child has residual neurological signs (for example, Todd's paresis)
- a serious infection is suspected (meningeal signs: Kernig positive sign and/or a Brudzinski positive sign and/or neck stiffness)
- changed consciousness for more than one hour after cessation of FS
- the source of the infection is not clearly determined
- rash develops in child
- bombing of anterior fontanel
- tachycardia that persists even after normalization of body temperature
- signs of moderate to severe respiratory distress, such as tachypnoea, obstruction, low oxygen saturation (<92% in the air), and recessions of the thoracic wall
- the child's age is less than 18 months
- there is a risk of recurrence
- parents or carers are unable to provide regular monitoring shortly after FS.(1)

Complications. FS can be extremely frightening and emotional traumatizing for parents. These may create the impression that a child may die during the crisis and brain damage is inevitable. Also, these may have concerns about the risk of developing epilepsy after febrile access. The risk of developing epilepsy would be 1% for the simple FS, compared to the incidence in the general population of 0.5%. The risk of developing epilepsy in complex FS is around 4-6%, being correlated with their number. Other risk factors include: short duration of fever (<1 hour) before access, seizure initiation up to 1 year of age or after 3 years, multiple FS, neuropsychological compromise, aggravated ereccololic anamnesis, epileptiform changes in EEG.(2,3)

Encephalopathy is a rare complication of febrile seizures. Recent studies have shown that mutations in SCN1A and SCN2A sodium channels can lead to a predisposition to severe convulsions. Within the increased frequency of convulsions, changes may occur in hippocampal neural circuits and in temporal sclerosis. They can also cause damage to white matter maturation, reorganization of neuroplasticity and microstructure.(1)

It is known that children with FS will not have any cognitive problems or neurological deficits in the future. A cohort population study in Rotterdam has shown that there is no association between FS and the risk of developing cognitive problems. In the case of recurrent FS, frequent association with retardation of speech development acquisitions (OR 3.22) was demonstrated. In a study in Sweden, a combination of autistic spectrum disorders, learning difficulties, attention deficit hyperactivity disorder and high frequency fever ($p < 0.001$) (2,3) have been shown to be associated in 27092 children.

Some FS studies may increase the risk of developing Tourette's syndrome. A retrospective study of 1,586 patients in Taiwan showed an increased incidence in the cohort of FS patients (28.5 versus 13.9 per 10000 persons per year).

Children with FS have an increased risk for atopic pathologies such as allergic rhinitis and bronchial asthma. Also, an increased prevalence of stress hyperglycaemia in children with FS has been demonstrated. Very rarely FS can be associated with pulmonary edema.

Prognosis. Prognosis is usually favorable. It is usually an age-dependent syndrome that disappears after the age of six. Approximately one-third of children who have had a FS may have recurrence during childhood, and less than 10% will have more than 3 recurrences. The trend of repetition of crises would be in 75% of cases in the first year after first febrile access and 90% in the first two years. The risk factors for recurrence are: family history of positive FS (FS and epilepsy in first degree relatives), a first FS before the age of 18 months, the occurrence of the first FS episode less than one hour after the onset of fever, FS at a body temperature of less than 38 °C, frequent febrile illnesses, multiple febrile accesses during the same infection, complex FS, retention in psychomotor development. Convulsions will be repeated in 4% of children without risk factors, and up to 80% of children with risk factors described above. Clinicians and parents/careers are often concerned about the reappearance of FS, especially with regard to the risk of epilepsy. Simple FS may slightly increase the risk of epilepsy but do not have adverse effects on behavior, school performance, or cognition. The risk of developing epilepsy is further increased in children with a history of complex FS. It is important to know the risk factors for the recurrence of FS to counsel parents or caregivers of the child and to administer rescue antiepileptics to children at increased risk of recurrence.(11,12)

Treatment. Emergency treatment includes Lorazepam iv 0.05-0.1 mg/kg or Diazepam 0.1-0.2 mg/kg. The Cochrane Revolution of 2018 has shown that Lorazepam has the same efficacy as Diazepam in stopping acute tonic-clonic seizures (OR 1.04) and both drugs have the same respiratory depression rate. In a randomized, double-blind study on a group of 273 epilepticus pediatric patients, 140 received 0.2 mkg/kg of Diazepam and 133 children received Lorazepam 0.1 mg/kg. Elimination of epileptic status after 10 minutes was assessed in 101 of 140 children (72.1%) in the group of children receiving Diazepam and in 97 of 133 children (72.9%) who received Lorazepam. When the intravenous route of administration is not available, Diazepam can be administered i/rectally 0.5 mg/kg, 0.5 mg/kg or intranasally 0.2 mg/kg, and Midazolam can be administered orally 0.2 mg/kg, or nasal 0.2 mg/kg.(2,3,11,12)

The febrile status epilepticus rarely can be discontinued and usually several anti-epileptic remedies are needed to stop it. Initial treatment consists of administering Diazepam 0.2 mg/kg or Lorazepam 0.1 mg/kg iv. If the seizures continue, more than 5 minutes repeat the same iv dose. Further, if access continues, Phenobarbital 20 mg/kg or Phenytoin (Phosphenitine) 20 mg/kg is administered. And if the convulsions persist it can be supplemented with a dose of Phenytoin of 5-10 mg/kg. Another option would be the administration of valproic acid 20-40 mg/kg or Leviteracetam 20-60 mg/kg iv.(2,3,11,12)

Parents need to be educated about the benign nature of seizures and about the fact that they are an age-dependent phenomenon. Most children with CF do not require hospitalization. Hospitalization is recommended only in children with severe and or prolonged infections.(2)

Table no. 3. Ordinary medical treatment of the child with FS

M	Do	Adm	Freq	Max dose	When to use it
P	15 mg/ kg	Oral, rectal or intra-venous adminis-tration (IV)	Every four to six hours	Five in 24 hours	For pyrexia in children with FS
I	5-10 mg/ kg	Oral	Every six to eight hours	Four in 24 hours	For pyrexia in children with FS, unless they are de-hydrated

D	0,25 - 0,5 mg/ kg	IV or intra-venous Rectal	A second dose can be adm. ten minutes after first	Only two doses of benzo-diaze-pines should be used irrespective of the agent selected and given alone or in combi-nation	For a child with active seizures whose crises lasted more than five minutes
L	0,1 mg/ kg	IV	A second dose may be given ten minutes after the first dose	Only two doses should be used	For a child with active seizures, if the crisis lasted more than five minutes
Mi	0,15-0,2 mg/ kg	IV	A second dose can be given 10 minutes after the first dose	Only two doses should be used	For a child with active seizures whose crises lasted more than five minutes
0,9% NaCl	20 ml/ kg	IV	During resusci-tation	More than two doses are rarely needed	In children with shock, for example, in febrile illness due to gastroenteritis

Legend: M = medication; Do = dose; Adm = administration; Max = maximum; Freq = frequency; P = paracetamol; I = ibuprofen; D = diazepam; L = lorazepam; Mi = midazolam

Prevention. The Cochrane Revolution of 2017 has shown that daily dosing of valproic acid 10-15 mg/kg or fenobarbital 5-8 mg/kg for children less than 2 years and 3-5 mg/kg for children over 2 years may be effective in the prevention of recurrences.(2,3) Adverse reactions of antiepileptic medication were noted in 30-40% of patients with chronic anti-epileptic therapy. Adverse reactions of valproic acid have been noted: headache, nervousness, insomnia, alopecia, renal toxicity, pancreatitis, gastrointestinal disturbances, thrombocytopenia and hepatotoxicity. The adverse effects of Phenobarbital were: vertigo, appetite disorders, nausea, vomiting, sleep disturbances, attention deficit hyperactivity disorder.(12) Thus, adverse effects of Phenobarbital and valproic acid outweigh their benefit. Based on the above, it was decided by consensus that continued prophylaxis with antiepileptic remedies is not recommended for children with CF. It is also considered that their continued use would not reduce the risk of epilepsy. A randomized clinical trial of a 145 group of children aimed at comparing the efficacy of continuous Fenobarbital treatment and the intermittent administration of Diazepam during febrile arrest showed a recurrence rate of 23% for Fenobarbital and 15% administration of Diazepam.(11) Adverse effects were more rarely encountered in intermittent therapy with Diazepam and were characterized by: somnolence, ataxia, irritability, respiratory depression. They can sometimes mask the symptoms of an intracranial infection (e.g. meningitis). Some studies have been performed on the intermittent administration of Clobazam and Leviteracetam to prevent febrile access. The current consensus does not recommend the routine administration of anticonvulsant treatment intermittently. It can be recommended in cases of high parental anxiety about recurrence of febrile access, prolonged or multiple seizures, and those at high risk of recurrence.(3)

Controlled studies on administration of antipyretic remedies during febrile illness have shown their ineffectiveness in preventing FS. A study of antipyretic remedies (acetaminophen, ibuprofen and diclofenac) to prevent the onset of FS has been performed.(12) Thus, there was no statistically significant difference between the study group and the control group (OR 0.9, 95% CI) in the prevention of FS. There is also no evidence of the effectiveness of physical remedies to reduce fever for FS recurrence.

CONCLUSIONS

FS are the most common types of seizures encountered in the pediatric population, being diagnosed in 2-5% of children aged 6 months to 5 years. Parts of the FS are simple (up to 95%) and only a small number of them are complex. Most children have an excellent prognosis because FS is often benign, but few children develop long-term health problems because complex FS have a higher risk of recurrence and development of epilepsy. Approximately one third of children who had a FS show a recurrence of access during their childhood. FS is an age-dependent phenomenon, which disappears after 6 years, typically. The FS diagnosis is clinical and it becomes important in such cases to exclude intracranial infections, especially after a complex FS. The patient's conduct consists in controlling the symptoms and treating the cause of the fever. Parents and caregivers need to be properly informed by health professionals about child behavior and FS prognosis. To avoid abuse of diagnostic tests and treatments, pediatricians and neuroscientists should be properly informed about the FS in a child.(2,3)

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