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Frontal Lobe Epilepsy in Children: Clinical, Electroencephalographic and Evolutive Aspects at the Neurology Department of Fann National University Hospital, Dakar, Senegal

Diop-Sène Marième Soda*, Nsounda Mandzela Annick, Mbaye Khalifa Ababacar, Mbodj Ahmadou Bamba, Diagne Rokhaya, Ka Mamadou, Saphou-Damon Michel Arnaud, Cissé Ousmane, Ba El Hadj Makhtar, Ndiaye Moustapha and Diop Amadou Gallo

Department of Neurology, Fann Teaching Hospital, Cheikh Anta Diop University, Dakar, Senegal

Abstract

Introduction: Frontal lobe seizures are one of the most frequent seizure types in children. Diagnosis and treatment could be difficult sometimes. In a context where there is no stereo-electroencephalography (SEEG), and epilepsy surgery is impossible.

Methodology: We conducted a descriptive study from January to July 2016 at the neurophysiology department of Fann University Hospital in Dakar. Children with frontal lobe epileptic abnormalities were involved in the study. They had a standard EEG and neurological and neuropsychological assessment.

Results: A total of 9 cases were collected and only 7 patients were included in the study. At the end of the study we found a good correlation between clinical signs and electrophysiologic findings. 4 out of 7 had had their first episode in preschool age, 6 out of 7 had nocturnal seizure, 5 out of 7 had 3 to 5 seizures each time. 4 out of 7 had seizures during EEG recording. EEG findings were fast rhythms, spike and spike and wave in frontal derivations. 5 out of 7 had neuropsychologic assessment and 4 patients had procedural memory; visio-spatial; planification; mental flexibility and inhibition impairment. We did not find the etiology in our patients. Evolution was good with antiepileptic treatment. All the patients were really improved.

Conclusion: Frontal lobe seizure are one of the most frequent seizure type in children. Diagnosis and treatment could be difficult sometimes. In a context where there is no stereo EEG, no epilepsy surgery, we need to have the most precise diagnosis, to offer more chance to our patient to benefit from treatment to improve prognosis and outcome in children with frontal lobe seizures.

Keywords: Frontal lobe epilepsy; Frontal lobe seizure; Electroencephalogram (EEG); Children; Senegal

Introduction

In the literature, it is recognized that almost half of all cases of epilepsy occur before the age of 15 [1]. Frontal lobe epilepsy is the second type of partial seizures after temporal seizures at the rate of 20 to 30% of partial seizures [2]. The critical semiology is very varied and surface electroencephalographic (EEG) recordings often fail in detection of irritative foci in the frontal lobe because of its large volume and deep localization of those foci, which often leads to misdiagnosis [3]. Detection of deep foci requires such means as stereo electroencephalography (SEEG) or functional imaging [4]. Evolution very often leads to refractory epilepsy, with repercussions on cognitive aspects and quality of life of patients [3].

In this work, we present observations of children with frontal lobe epilepsy by approaching the clinical, electroencephalographic and evolutive aspects of these seizures in our context.

Research Methodology

We conducted a prospective study at the EEG laboratory of the Neurological Clinic of the Fann Teaching Hospital during the period of January 2, 2016, to July 13, 2016, included. The study population consisted of any child who had an EEG in the Fann neurological clinic laboratory during the study period and in whom the EEG pattern showed focal epileptic abnormalities in the frontal regions. Children with frontal focal abnormalities whose follow-up was not done in the department or whose parents did not wish to participate in the study were excluded from the study. The Fann Neurology Clinic is in a national and international reference hospital. The EEG laboratory consists of three EEG devices held by four trained technicians under the supervision of neurologists specialized in neurophysiology and epileptology.

The recruitment EEG was a standard digitized EEG awake and/or sleep coupled with video recording. The electrodes were placed according to the 10/20 international system with a headset recording brain activity on 20 electrodes. The data were collected on a data collection sheet designed for the study and that considered different elements: anamnestic data, clinical elements, electroencephalographic aspects, and the evolutionary profile.

Observations

During the study period, 228 focal epilepsies were diagnosed; 86 were frontotemporal with an electro-clinical description that did not allow the diagnosis of a definite frontal origin. Nine children were relevant to our study, 7 of them were included. Below are the list of included patients.

***Corresponding author:** Marième Soda DS, Department of Neurology, Fann Teaching Hospital, Cheikh Anta Diop University, Dakar, Senegal, Tel: +00 221 77 631 14 97; E-mail: dmariemesoda@gmail.com

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Patient 1

Y.G. born on October 1, 2003, was followed since the age of 8 in psychiatry for behavioural disorders with sudden fear leading to escape, unmotivated fits of laughter. These episodes took place exclusively at bedtime. She received sodium valproate at the dose of 500 mg and phenobarbital 50 mg without effect, which motivated a consultation and follow-up in neurology.

In her personal history, we found fourth-degree consanguinity and a notion of epilepsy in a paternal cousin who had epilepsy for 3 years (from the age of 13 to 16). Pregnancy and childbirth had been unremarkable.

The patient had school retardation; she was enrolled in 4th grade student class. Her seizures occurred exclusively during sleep, they began with a contracture of the muscles of the whole body, then she began to cough and scream, then came a contact break, these events occurred about three times. During the day, occurred episodes of gestural automatism in hands-type manipulation of objects or people within reach staring at any point with eyes wide open; then she would start laughing. She did not respond to the entourage calls during this phase. The daily frequency of seizures was approximately 4-5 seizures during each episode. These episodes occurred once or twice a month.

The examination was without particularity. At the neuropsychological examination, the copy of Rey's figure was well made, the trail making test B was well executed, the test of labyrinths showed a procedural memory and the development of normal strategies. She had no problem with encoding but problems of restitution and did not present any problem of attention. The go-no-go test was well executed.

Awake-EEG was normal, sleep-EEG found spike-type anomalies, slow spike-waves, and fast- spike-waves at front polar electrodes and at F8, these anomalies predominated on the right and diffused to the temporal, ipsilateral and contralateral region. During the recording, there were 5 seizures with clinical manifestations and 4 subclinical seizures. These seizures were preceded by a flattening of electro-genesis, lasted on average 25 to 30 seconds and were followed by pseudo-rhythmic slow wave puffs. At the video-EEG the seizures occurred during the slow sleep, marked by eye-opening, gestural automatisms like manipulation of the bottom of the T-shirt, rubbing of the right hand by the left hand, followed by a grinning with deformity of the mouth, feeling of smiling and finally opening of the eyes, there was a deviation of the look on the right, a right nystagmus with ocular revulsion. At the end of the seizure, the patient rubbed her eyes and face and then plunged back into sleep. During the seizure, she was pressing her legs together. This behaviour was stereotyped and strictly repeated according to the same chronology (Figures 1 and 2).

The patient was treated with carbamazepine 400 mg daily. Favourable evolution was noted marked by a decrease of the frequency of seizures because she was having one to two outbursts per episode, the episodes of seizures were spaced of 2 to 3 months, the EEG of control realized twice showed no anomaly.

Patient 2

M.D. born on March 24, 2002 was followed since the age of 5 for nocturnal seizures occurring exclusively during sleep. He was having about five a day. These seizures were generalized tonic-clonic, preceded by cries approximately once a week. He was put on sodium valproate 500 mg, in the absence of improvement, clonazepam was added and sodium valproate was gradually replaced by phenobarbital 50 mg. After a few days, the frequency of seizures was reduced to one seizure a day.

He was the first child of 3 uterine siblings. In personal antecedents, pregnancy and childbirth without peculiarities were found. Acquisitions were made at 1 year for walking and 3 years for language. He was late in learning the verses at Koranic school. There was no notion of fever or cerebral infection in the years before the seizures. In the family antecedents, 1st-degree consanguinity was found in the parents; one paternal aunt and 2 first cousins were epileptic.

The neurological examination and the examination of other systems

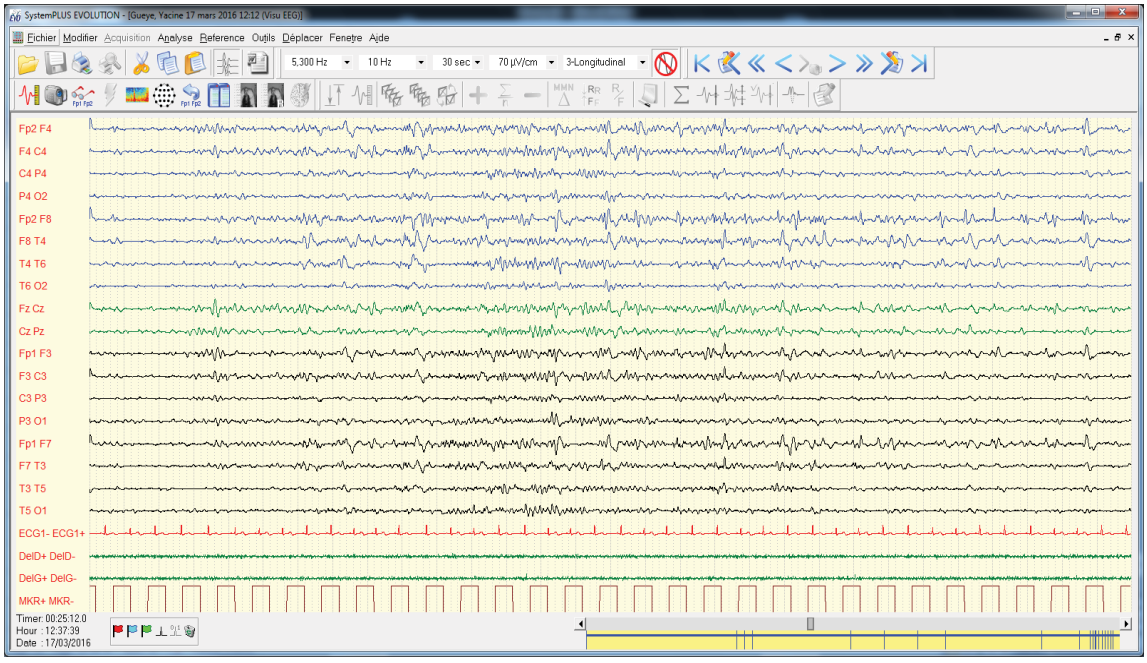

Figure 1: Inter-critical EEG showing spike and spike-wave trains in right frontal regions diffusing in left frontal regions.

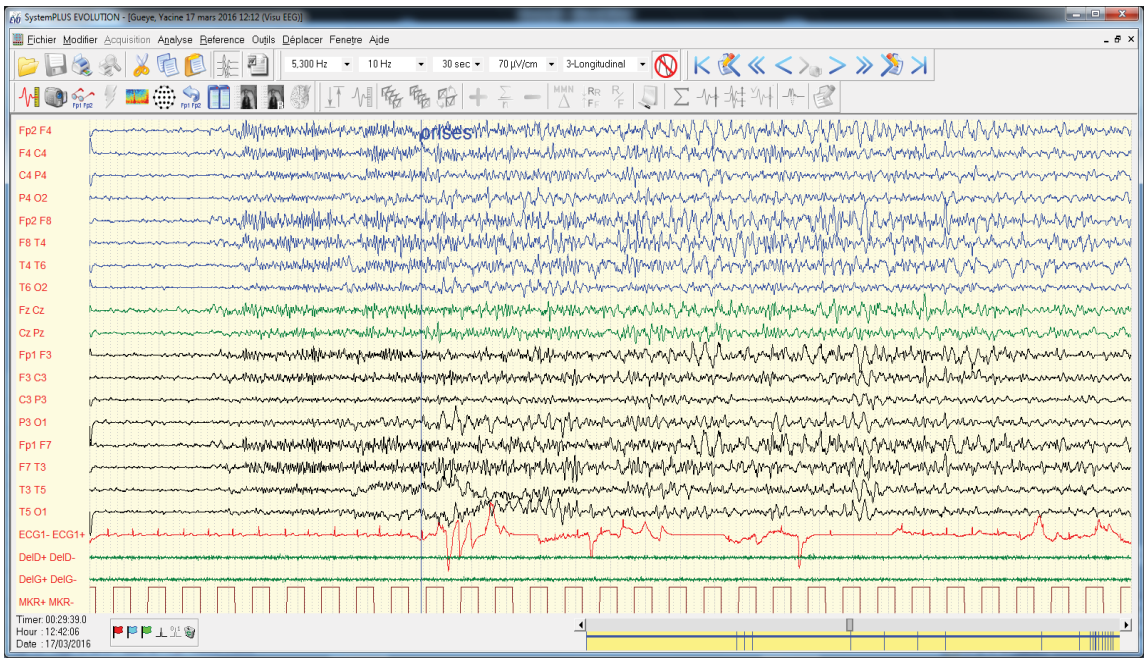

Figure 2: Critical EEG showing fast spike-waves discharges in bilateral frontal regions.

were without particularity. The neuropsychological examination was difficult to carry out because the child was not educated in French and had speech difficulties. However, at the memory level, a well-done encoding was found but the restitution was difficult; semantic memory disorders, procedural memory disorders, were noted. He had disturbances of visuospatial perception and difficulties of concentration.

The brain scan performed in 2007 was normal (report attached but no snapshot). The sleep-EEG showed an unstructured macro-architecture with the impossibility of identifying the different stages of sleep. There was a rough sketch of K complexes and poorly drawn and asynchronous sleep spindles. On this background rhythm, there were numerous bursts of spikes, generalized spike-waves preceded by rapid rhythms in the anterior regions, the anomalies came in generalized bursts with predominance in the bi-frontotemporal regions. These anomalies were increased by sleep. The video-EEG showed episodes of elevation of the right upper limb then left with tonic contracture and extension of the trunk ("position of the fencer"). At the end of the recording, we saw the appearance of numerous discharges of poly spike-waves concomitant to myoclonic jerks of the upper limbs. At the beginning of the seizure, the child jolted awake and displayed a sense of bewilderment, these events were concomitant with fast rhythms bursts in the bi-frontal regions. (Figures 3 and 4).

The patient received phenobarbital 50 mg and carbamazepine 400 mg daily. The clinical course was favorable under treatment marked by a decrease in the frequency of seizures.

Patient 3

M.N. born on March 23, 2005, was followed for the management of generalized tonic-clonic seizures occurring since the age of 5 years. He received phenobarbital at a dose of 50 mg daily but with poor compliance with treatment. He was the first of four siblings. Pregnancy, birth and the neonatal period had gone smoothly. Parents reported a delay in learning Koranic verses compared to students of his age. In the family history, there was first-degree consanguinity in the parents but there was no notion of epilepsy in the family.

The usual seizure was described as a generalized tonic-clonic seizure that occurred several times during the day, at a frequency of 2 to 3 episodes a day when he was not taking his treatment; when intake were regular, the frequency of seizures decreased to one seizure every 2 weeks or less.

The neurological and other systems examination was without particularity. The neuropsychological examination found a reserved child and lacking self-confidence which gave poor results to the tests. He was not educated in French and this was a hindrance to performing some neuropsychological examinations. The copy of Rey's figure had very altered shapes and proportions showing disturbances of visuospatial perception, the trail making test was not done because there is a problem of understanding the instructions, the failure to the labyrinth test showed an inability to plan, reflecting a deficit of procedural memory. The "go-no-go" test showed a deficit of inhibition and attention. The test of the 3 words found disorders of concentration. There were no coding issues and the restitution was good.

The awake-EEG found bursts of bilateral frontal spikes, discrete, accentuated by Hyperpnea (HPN). These anomalies propagated to the adjacent temporal electrodes and had a clear tendency to diffusion. The video-EEG recorded clonic movements of the lower jaw and eyelids followed by a swallowing motion; this phenomenon occurred many times and was concomitant with these spike-wave bursts (Figures 5 and 6).

The patient was given phenobarbital 50 mg daily, in the evening for 2 months. The clinical evolution was favourable, marked by the interruption of the seizures. A control EEG was planned.

Patient 4

D.D, born on June 02, 2004 was followed for generalized tonic-clonic seizures evolving for 6 months under phenobarbital 100 mg since the beginning of the seizures. He was the 2nd child of a uterine

Figure 3: Inter-critical EEG showing puffs of bi-frontal spike-waves with diffusion sometimes in the right temporal region.

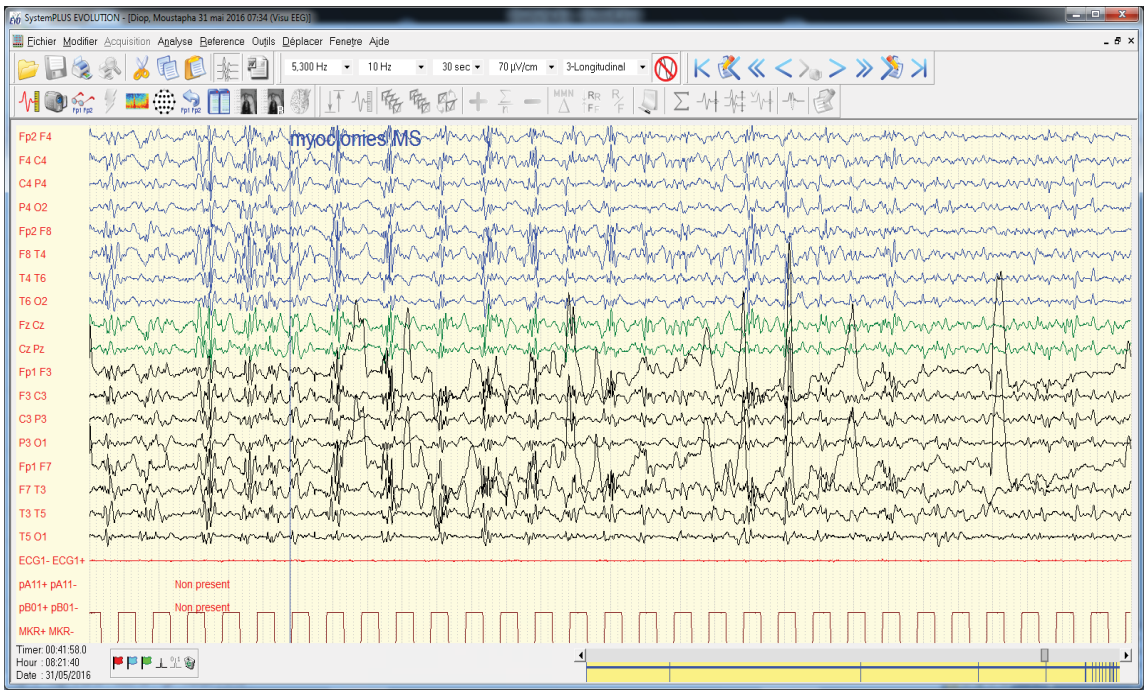

Figure 4: Critical EEG showing generalized spike-wave puffs and discharges of poly-spikes - maximum waves in bilateral frontal regions concomitant with colonies.

family of 3 children. Pregnancy and childbirth had gone smoothly. In his personal history, there is no history of cerebral infection or head trauma; he was educated at the French school and was late. In her family history, there was no parental consanguinity; her maternal aunt had convulsive seizures that improved at the age of 20 years.

During episodes of seizure the patient manipulated his hands mechanically; these gestural automatisms were followed by clonic movements of the head, then came a generalized tonic-clonic seizure. He has several crises a day including 2 or 3 at night.

The neurological and other systems examination was without particularity. The neuropsychological examination showed good procedural memory, but the development of strategies assessed with the labyrinth-test was poor; his planning capabilities were limited despite good information consolidation because the instructions were followed. The Stroop test was well done so he did not have any inhibition problems. He was unable to do the trail making test B due to planning difficulties and this reflected a low mental flexibility.

The awake-EEG found a focus of slow waves in the right frontal

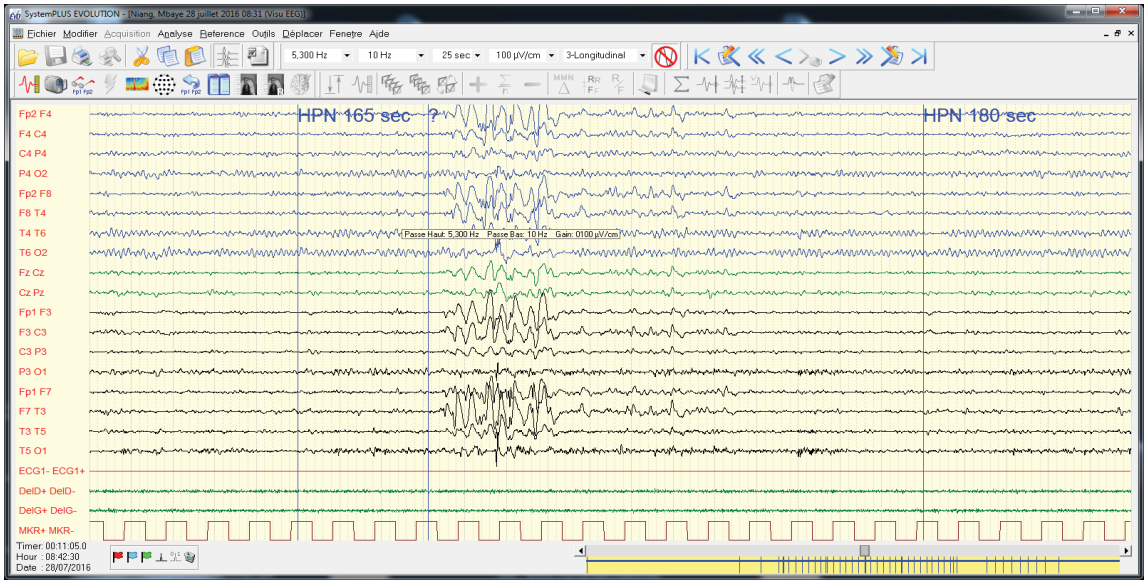

Figure 5: Inter-critical EEG showing slow waves interspersed with spikes in bilateral frontal regions.

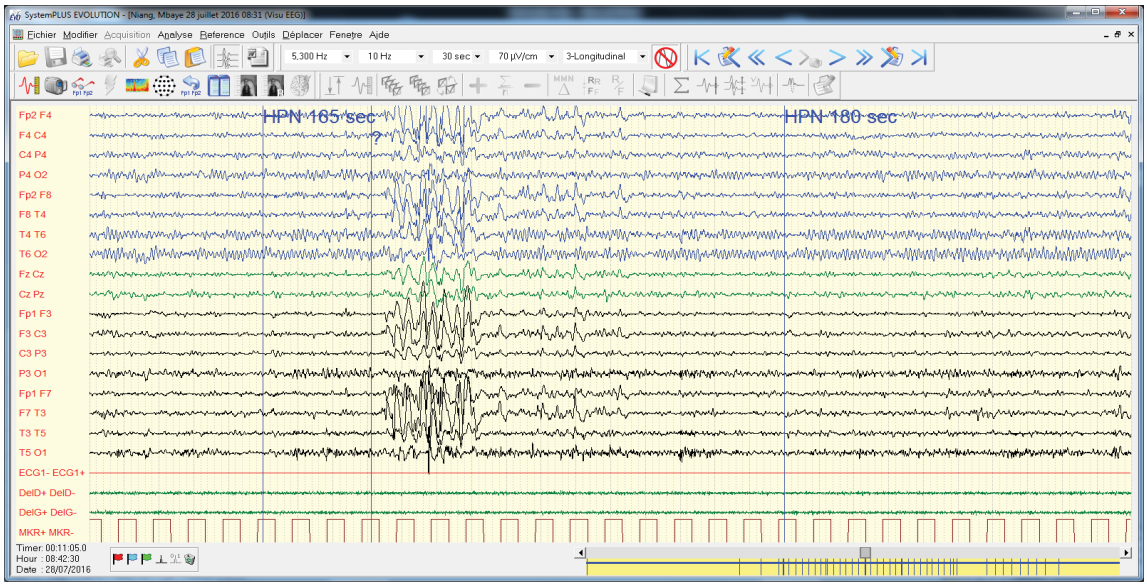

Figure 6: Critical EEG with pseudo-rhythmic puffs of bilateral frontal spike-waves diffusing in the temporal regions.

leads. The sleep-EEG showed abnormalities of slow spikes and degraded spike-waves in the right frontal derivations, these anomalies diffused to the contralateral hemisphere. The video-EEG is unremarkable (Figures 7 and 8).

The patient was given phenobarbital 100 mg a day in the evening for a month. A single night-time seizure occurred throughout the month. The evolution was favourable under treatment marked by an interruption of the seizures.

Patient 5

M.C.D. born on March 09, 2010 had his first seizure at the age of 4; it was a single episode and was not treated at this time. He had redone another 3 after 2 years of calm, which motivated his consultation in neurology where he was followed since. He had frequent episodes of nocturnal fright during sleep, accompanied by loss of urine and escape from the room. In his antecedents, he was the 2nd of a uterine family of 4 children. Pregnancy and childbirth had gone smoothly. There was no notion of neonatal infection or head trauma. He was having normal schooling. In family history, parental consanguinity was not found. There was a notion of familial epilepsy: his father had generalized tonic-clonic seizures treated with phenobarbital at the age of 6 with a resolution of seizures in adolescence; his older brother had seizures similar at 6-7 years, these seizures lasted 2 years during which he was treated with sodium valproate.

During the crisis, the patient screamed, had clonies of the head before doing a GTCS (generalized tonic-clonic seizure). These seizures

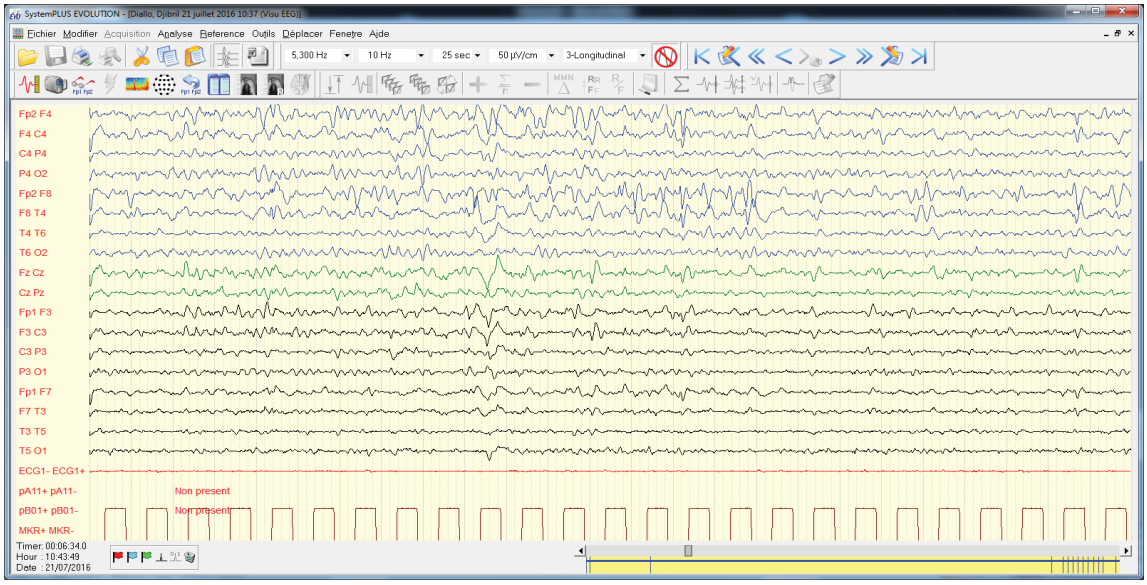

Figure 7: Awake-EEG showing a focus of slow right frontal waves.

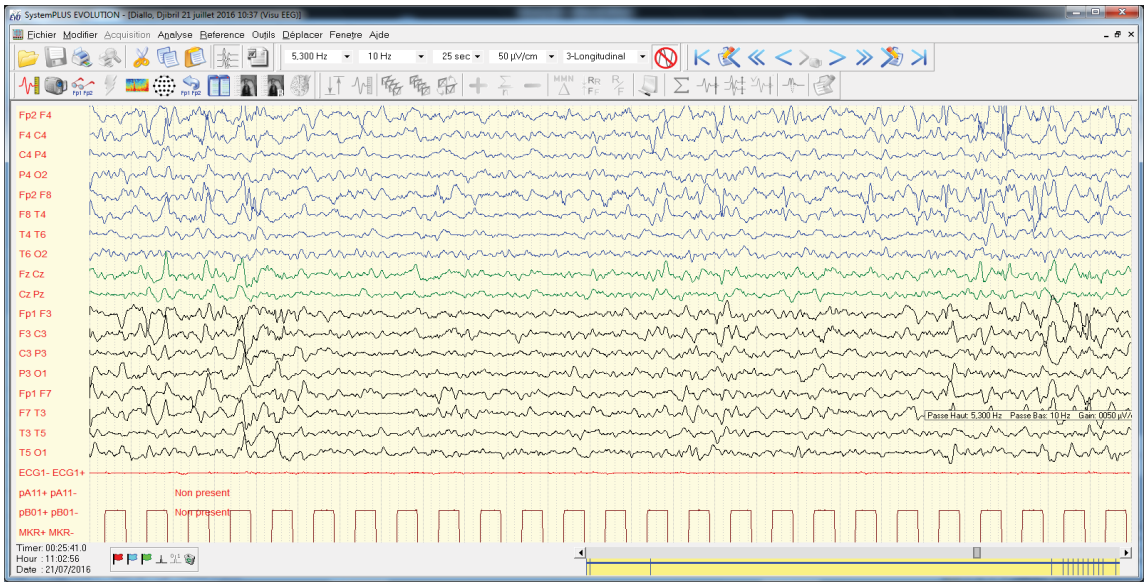

Figure 8: Sleep-EEG with spikes and spike-waves in the right frontal region. [EEG: Electroencephalogram].

occurred on average 3 times a day, at waking time.

The neurological examination and the examination of the other systems were without particularity. The neuropsychological examination showed the test of labyrinths: at the beginning of the test, setting up a strategy to get out of the labyrinth; then he forgot the instructions he had to repeat over and over again. It went through the walls which reflected a deficit of procedural memory and the development of strategies. He had disturbances of visuospatial perception with a distorted copy of the proposed geometric figures. He had inhibition difficulties highlighted by the "go-no-go" test. The encoding was deficient. Semantic and episodic memories were not evaluated.

The awake-EEG showed a bi-frontal irritative focus whose abnormalities tended to diffuse at the central and temporal electrodes, these anomalies were more marked on the right.

At the sleep-EEG, bursts of spike-waves occurred in the centrotemporal frontal regions (Figures 9 and 10).

The patient received phenobarbital 50 mg daily. The clinical course was good under treatment marked by the interruption of seizures.

Patient 6

D.S. born April 11, 2005, was followed for epilepsy since the age of 5 years. She was doing 3 to 5 seizures a day especially at night during sleep. She followed a traditional treatment and then, in the absence of improvement, was taken to the hospital for better care.

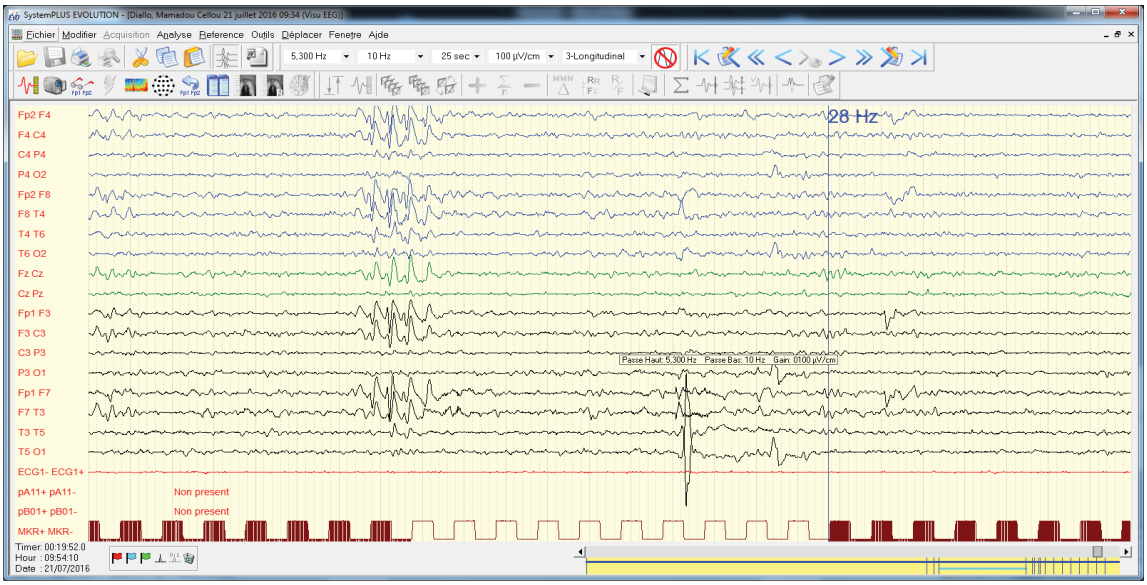

Figure 9: Awake-EEG showing bilateral frontal spike-waves.

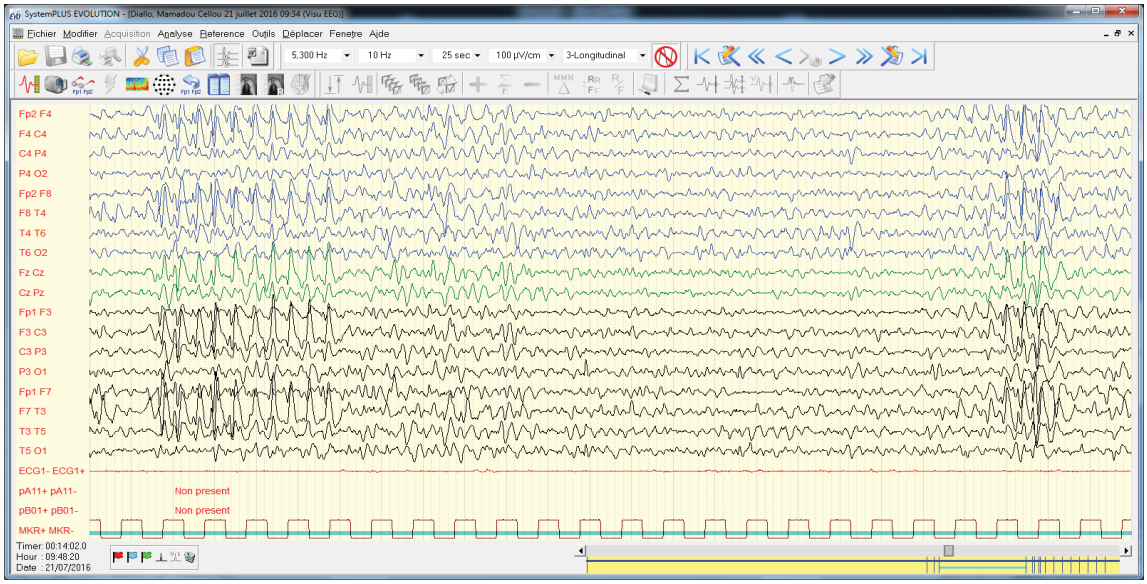

Figure 10: Sleep-EEG with wide spike-waves in the bilateral frontal regions.

She was the first child of a sibling of 3 children. The pregnancy went well until 33 weeks of amenorrhea. The delivery was premature in a context not specified by the parents. She stayed in neonatology for 2 or 3 weeks before being given to her parents. She had no personal history of cerebral infection, fever, or head trauma. She was late in schooling. In family history, there was no notion of epilepsy in the family. There was no parental consanguinity.

The seizures occurred at night beginning with a cry, then an expression of fright registered on the face, she said "I see lions", and then fled running. These seizures occurred 3 to 5 times a day. The neurological exam and other systems were unremarkable. The neuropsychological examination was not performed.

The realized sleep-EEG showed the presence of physiological figures of sleep. At this rate, there were persistent outbursts of spikes in the bilateral frontal regions (Figure 11).

The patient was receiving sodium valproate at a dose of 500 mg daily. The clinical course since taking this treatment showed a clinical improvement because seizures were less frequent, 3 seizures, and more spread out (one episode of seizures every 2 or 3 months).

Patient 7

M.M. born August 12, 2009 was followed since the age of 4 years for epilepsy. He made his first episode at the age of 4. Seizures were all-body clonic movements with head version and facial fright, without loss of contact; these seizures were very frequent, about fifteen attacks in the night. He received sodium valproate 500 mg daily and carbamazepine

400 mg for 3 years without improvement. He was the third child of a uterine family of four children. There was no parental consanguinity. Pregnancy and childbirth had gone smoothly. In his personal history, there was no brain infection in childhood or brain trauma. He was out of school because of the seizures. No notion of epilepsy in the family.

The patient had seizures of diffuse clonies, accompanied by a version of the head, and a frightening expression on his face. Episodes were frequent in the range of 15 to 20 seizures a day at night or waking in the morning. The neurological examination and the examination of the other systems were without particularity. The neuropsychological examination was not performed.

The awake-EEG showed slow waves bursts at the left hemisphere predominant in the frontal derivations. On this background rhythm, came a critical discharge made of a diffuse micro-voltage rapid activity that started in the left frontal region. It was followed by an activity masked by the seizure artifact in which momentous spikes and rhythmic spike-waves were distinguished more marked in the left hemisphere, this critical activity was followed by a diffuse slowing background, more marked and persistent in the left. Sleep ensued with the post-critical phase, marked by a slowing down of the line and the appearance of vertex points, on mixed rhythms where slow activities always predominated on the left. The child then went into stage II and K complexes appeared. (Figures 12 and 13).

At the video-EEG: the attack began with an elevation of the right upper limb followed quickly by that of the left upper limb, then a tonic and tonic-clonic rotation of the trunk, upper limbs and head and eyes towards the left, with an expression of terror on the face. It was associated with a lateral sway of the lower limbs, a reaction of automatic gripping of the stroboscope stem, then secondarily a progressive generalized muscular relaxation, the child entered a kind of apathy after having had some oral and labial automatisms, then went back to sleep.

The patient received carbamazepine 400 mg daily and phenobarbital 50 mg daily. The evolution was favourable under treatment marked by a decrease in the frequency of seizures; at a rate of a nocturnal crisis every month or every 2 months.

Discussion

Clinical aspects

In our study, most patients had their first attack in preschool-age; for three of them, it was at the age of 5 years, for another at the age of 4 years. These results agreed with those of Laskowitz et al., who found seizures onset age below 7 years for 45 to 55% of their patients [5].

Six out of seven patients had nocturnal attacks during sleep. This trend was confirmed by Fogarasi et al., who found a nocturnal predominance of frontal seizures in children [6]. Only one patient described frequent seizures during the day. But his father described frequent nocturnal awakenings because he often leaves his room with frightened air and urine-stained pants. This child was likely to have manifestations that went unnoticed during his sleep; the indication of a Holter-EEG associated with polysomnography was posed [7]. According to Panayiotis et al., [8], this nocturnal trend associated with the wide clinical variety of frontal seizures, related to the complexity of this lobe, explained the difficulty and / or delay of clinical diagnosis of frontal seizures.

Five out of seven patients described frequent episodes of seizures ranging from 3 to 5 seizures per episode. These results were consistent with those found in a study of frontal epilepsies [4,6], which revealed a high frequency of seizure episodes in patients with frontal epilepsy. For only one of our patients, we observed only one episode of 3 seizures which could be explained by the possibility that it makes more subtle events that passed unnoticed in the eyes of the parents.

Seizures were recorded during the EEG in 4 of the 7 patients retained. Three-quarters of our patients had short electro-clinical seizures lasting less than one minute. This was consistent with the results of Fogarasi et al., [6]. For the others, it was impossible to specify the duration of the crisis.

Three of our patients had gestural automatism during seizures. Another presented oral automatisms. This observation agreed with those found in the literature [6,9]. According to Fogarasi et al., there was less gestural automatism in the paediatric population than in

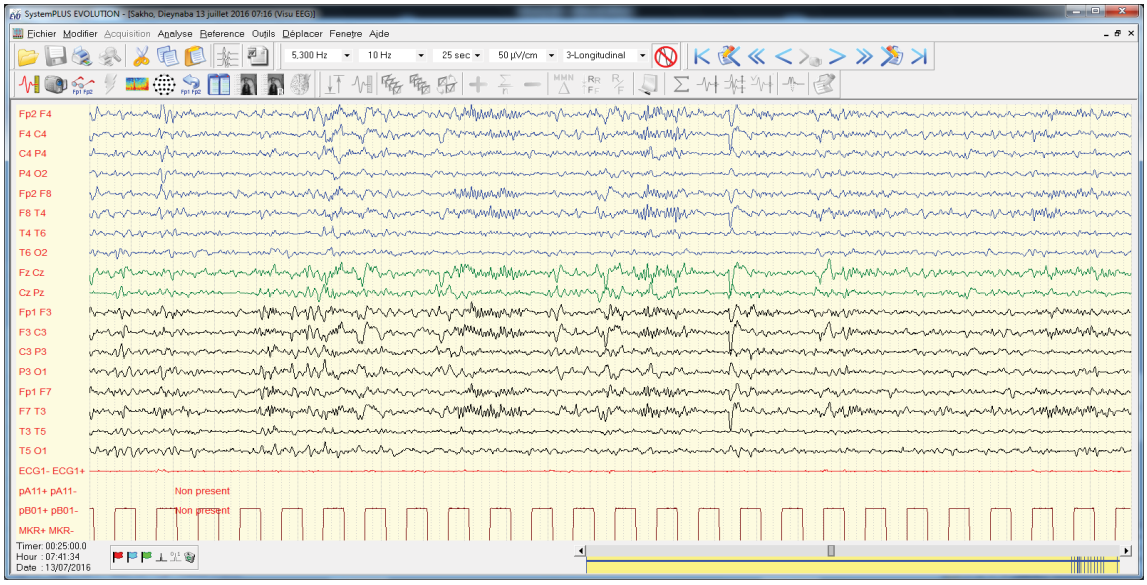

Figure 11: Sleep-EEG showing peaks in bilateral frontal regions.

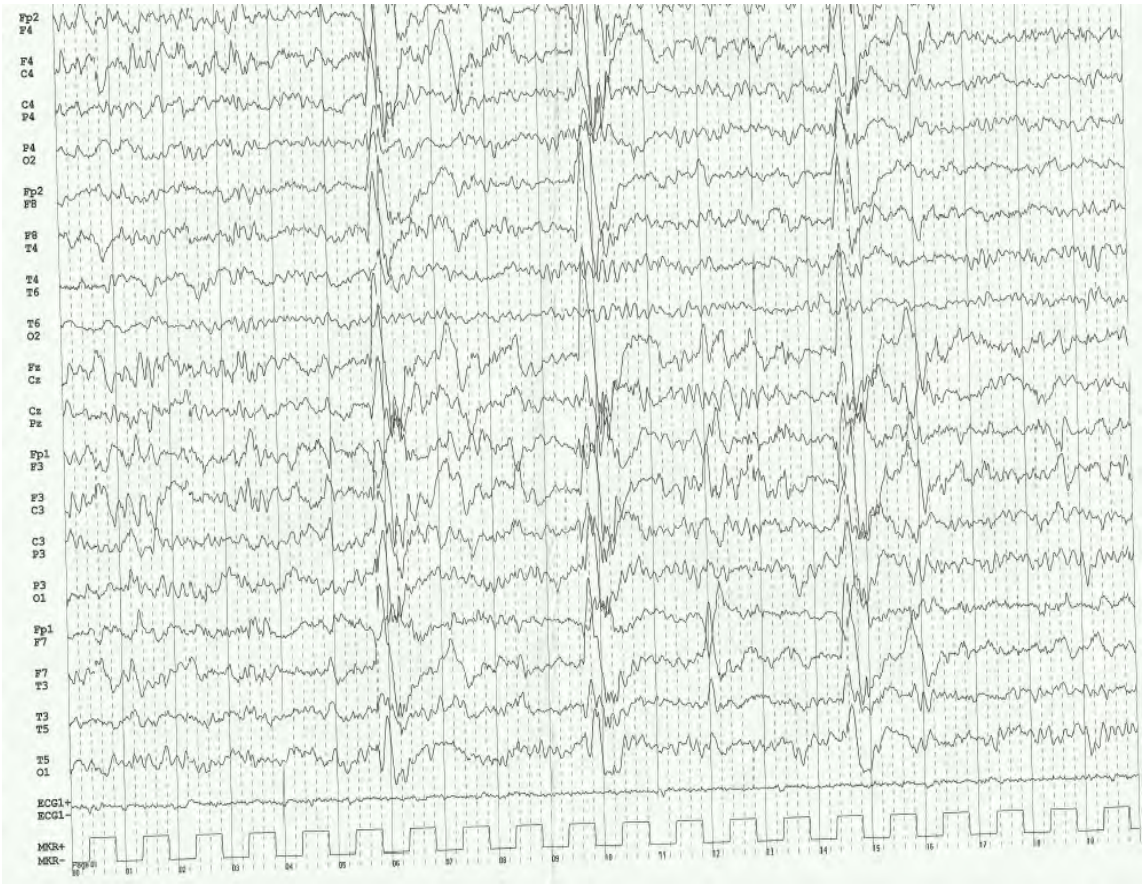

Figure 12: EEG of patient 7 in February 2015 showing high voltage spikes in bilateral frontal regions.

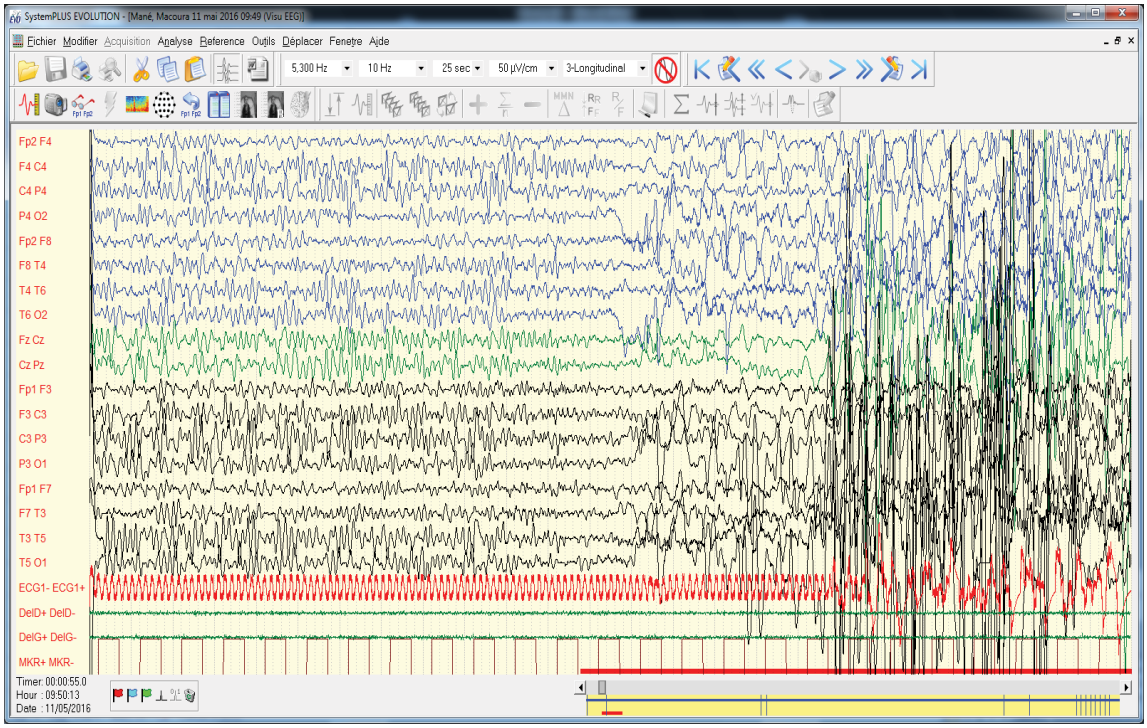

Figure 13: Critical EEG showing diffuse fast activities predominant in bilateral frontal regions and critical activity masked by the crisis artifacts.

adolescents and young adults, and this reflected the immaturity of cortical connections and myelination of the frontal lobe in the paediatric population. All our patients had frontal seizures with motor manifestations. Their critical descriptions were strongly in line with those found in the literature, which originated in the above-mentioned regions [9-11], which also allowed us to locate the critical focus. According to Bonini et al., who had identified "frontal groups of epilepsy" according to the signs and made it possible to direct towards precise locations thanks to the stereo-EEG: the patients of our study were therefore in group 3 for patients 1, 4 and 7; in group 1 for patients 3 and 5, in group 2 for patient 2 and in group 4 for patient 6.

Electroencephalographic characteristics

It was often difficult to distinguish generalized seizures from partial seizures by visual inspection of the semiology of seizures, especially in children [12]. But we must also note that the critical focus in frontal epilepsy is often difficult to locate at standard EEG due to the deep or basal situation of this critical focus that surface electrodes have difficulty capturing, the depth of the frontal lobe, the complexity of the functional organization and the many integrated circuits that it supports [13].

In our study the inter-critical EEG found at the awake state a normal background rhythm for most patients and a well-organized sleep for all those who realized it. On these background activities were grafted spikes, slow spikes-wave and rapid spike-waves in the frontal regions, these anomalies diffused in the contralateral hemisphere, giving bi-frontal lesions; there were also slow-wave foci in the frontal branches, sometimes large spikes in the frontal regions. These abnormalities sometimes diffused in the central and temporal regions. These results agreed with those found by Beleza et al., [14]. The epileptic abnormalities recorded in inter-critical EEGs were bifrontal in our patients and consistent with those described by Yu HJ et al., [15]. This phenomenon was explained by the fact that epileptic anomalies of the frontal lobe could rapidly diffuse into the opposite hemisphere via the corpus callosum and give this bilateral synchrony. The morphology of the inter-critical anomalies of the EEG of our patients was thus in agreement with the description of Kellinghaus et al., which found fast paroxysmal activities, isolated spikes or isolated poly-spikes, complexes of spikes-waves in the frontal regions [16].

For 4 of the 7 patients in the study, seizures were recorded during the EEG. The patterns observed corresponded to those reported by Beleza et al., who found rapid anomalies of the epileptic type in 36% of cases, delta rhythmic activities in 26% of cases and flattening in 14% of cases. These activities corresponded to a critical focus in the dorsolateral regions, the central gyrus, and the premotor and prefrontal cortex. Muscle artefacts contaminating critical EEG patterns were also described by the same team in 20% of cases and involved the frontal mesial area; slow wide critical waves and electrical decrement (29%) reflecting rapid activity (33%) are characteristic of this region.

In general, the lateralization of the critical EEG is minimal (25%) and for 75% the pattern is not lateralized or localized. The spread of frontal seizures was found in neighbouring areas in about 80% of cases in the 2004 Kellinghaus et al., study [4]. Mesial region seizures spread faster than those in the orbital or dorsolateral region [4]. Lateralization was also found at the EEG for 4 out of 7 patients and not for all patients. Yu HJ et al., found accurate lateralization of the epileptic focus for 19% of their patients who all had frontal epilepsy [15]. Precise localization studies of focal frontal foci were done using SEEG or functional imaging because of the difficulty of locating the frontal foci due to the volume and depth of this lobe. Our study found a good electro-clinical concordance in all our patients from the surface EEGs realized and thus allowed to confirm the diagnosis of frontal epilepsy in all our patients.

Neuropsychological performances

The preliminary neuropsychological examination was performed in 5 of our patients. The tests were normal in a single patient who had no cognitive impairment. The types of abnormalities found in our series were consistent with the neuropsychological deficits found in the literature concerning frontal epilepsies in children [17]. However, other neuropsychological assessments are needed to confirm these preliminary results, as the results of neuropsychological tests in frontal epilepsies may vary over time due to seizure frequency [18]. The abnormal results in neuropsychological tests reflected cognitive disorders and their repercussions on school performance and probably explained the school delay in these patients [19]. The high frequency of seizures led to the de-schooling of one of our patients. We live in a socio-cultural context where epilepsy is considered "transmissible", as a factor of social exclusion; in this context, parents estimated at 52% according to Ndiaye et al., [20], that it was not necessary to educate an epileptic child. School eviction was the rule in epileptic seizures. The main feature of frontal epilepsy was the high frequency of seizures and was therefore very disabling in our context for patients; school performance was disrupted due to absenteeism related to seizures but also significant cognitive impairment that resulted from it [21].

Etiologies

Frontal lobe epilepsy was linked to multiple etiologies, the most common were tumors and focal cortical dysplasia [22]; no etiology was retained in our patients. The high cost of cerebral magnetic resonance imaging (MRI) and the unavailability of genetic testing limited etiologic research in our context.

Therapeutic and Evolutionary Aspects

Most of our patients were on antiepileptic monotherapy: phenobarbital for 3 of them, one patient was receiving carbamazepine, and another was receiving sodium valproate. For all of them, it was a decrease in the frequency of seizures and for one of them a seizure interruption for 2 months.

Two patients were on antiepileptic combination therapy. These patients had a decrease in seizure frequency. Epilepsy is called refractory when there is inadequate control of seizures despite well-conducted combination therapy and good adherence for 1 to 2 years; our patients under antiepileptic combination therapy for more than 2 years of polytherapy did not notice the cessation of seizures. The drug resistance of the frontal epilepsies in these patients became obvious and a possible indication of surgery considered. This agreed with the results of Matricardi and Beleza et al. who reported that 25% of patients referred for refractory epilepsy surgery had frontal lobe epilepsy [23,24].

Conclusion

Frontal lobe epilepsy is an entity made of multiple and varied complex and simple motor seizures. It also has a significant impact on the executive functions that it is important to emphasize on. The localization of these focal frontal critical foci constitutes a challenge in Epileptology. These seizures are described as resistant to antiepileptic drugs and very frequently leading to an indication of surgery.

Conflict of Interest

The authors declare that they do not have any conflict of interest related to this article.

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