

Childhood Temporal Lobe Epilepsy in the population of Algiers

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Abstract:

Background: The semiology of temporal lobe epilepsy in children can be profoundly affected by age and brain development. Children with temporal lobe epilepsy are more likely to demonstrate specific semiology. Our aim was to analyze clinical presentation, EEG and radiological assessment of childhood temporal lobe epilepsy in the population of Algiers.

Materials and Methods: We retrospectively analyzed 32 patients with temporal lobe epilepsy. We took into account familial and personal medical history, clinical examination, EEG, radiological assessment based on cerebral magnetic resonance imaging and evolution.

Results: 32 children with temporal lobe epilepsy. 18,78 % with malformations non progressive tumors on brain imaging. 21,87% with a pathological history. 59,37% with no abnormalities on brain imaging and no pathological history.

Conclusion: Patients with significant antecedents are expected to have high risk of continued seizures of childhood temporal lobe epilepsy in the population of Algiers.

Key Word: Temporal lobe epilepsy; childhood; clinical features

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I. Introduction

The semiology of temporal lobe seizures in children has been the subject of much research. As in adults, children with temporal lobe epilepsy (TLE) are more likely to demonstrate specific semiology when their seizures appear from specific parts of the temporal lobe (mesial, lateral or insular). However, unlike adults, the semiology of temporal lobe epilepsy (TLE) in children can be profoundly affected by age and brain development. This changing semiology may present a challenge for physicians trying to identify and effectively treat children with temporal lobe epilepsy (TLE).

The etiologies of temporal lobe epilepsy (TLE) in different cohorts of children have been little studied. Most of the data regarding the etiology of (TLE) in childhood come from studies of patients with seizures that can be treated surgically, particularly in older children undergoing temporal lobectomy. However, there is a large group of children with (TLE) who do not have any brain damage. These patients generally respond well to antiepileptic therapy and may present with a mild form of temporal epilepsy. Therefore, a classification based on the etiology of temporal lobe epilepsy (TLE) in children has prognostic implications. We recruited a cohort of children with temporal lobe epilepsy (TLE) and attempted to apply classification based on clinical findings, EEG (electroencephalography) and brain imaging.

II. Material And Methods

Study was carried out between January 2012 and December 2013 The inclusion criteria were the presence of two or more complex partial seizures (CPS) in the context of temporal lobe epilepsy (TLE), which appeared before the age of 15.

The clinical evaluation included a questioning (history, age of onset of seizures, type of seizures), a neurological examination. EEGs were recorded with conventional positions of the electrodes on the scalp, according to the international 10/20 system. Brain MRI was performed according to the epilepsy protocol.

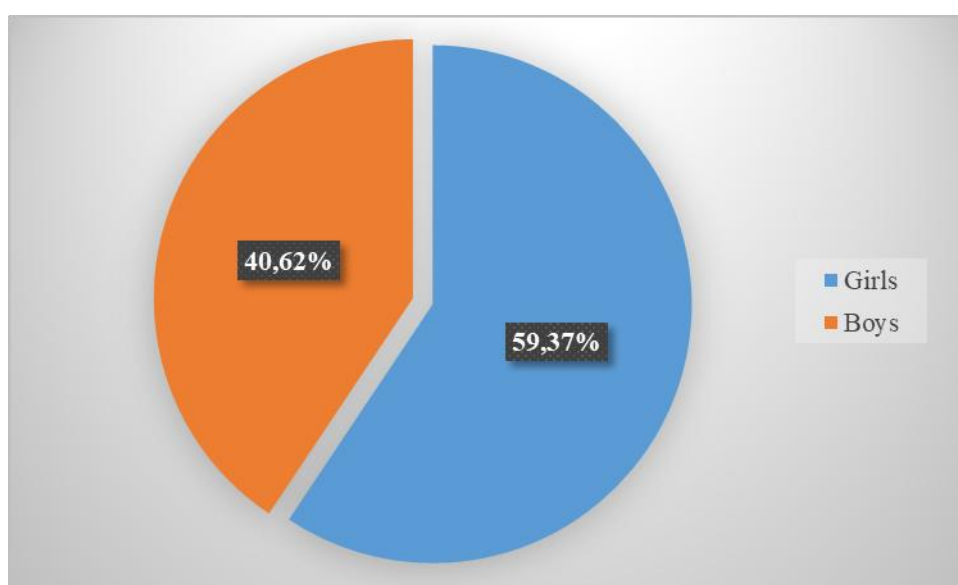
III. Results

We conducted a 2-year retrospective study between January 2012 and December 2013 that included 32 children with temporal lobe epilepsy (TLE) appearing before the age of 15, recruited from the district of Algiers.

The diagnosis of temporal seizures was supported by the presence of temporal focal inter-critical electrical abnormalities in all the children.

Of the 32 children with temporal lobe epilepsy (TLE), 40.62% (13 cases) were boys and 59.37% (19 cases) were girls, aged 6 months to 15 years (Figure 1).

Figure no 1: Distribution of patients by sex



In terms of neurological examination: 3 patients with neonatal anoxo-ischemic encephalopathy had tetraspasticity. These 3 children were mentally retarded. Electroencephalographically: Interictal EEGs, including sleep recordings, were performed in each child. Focal temporal interictal abnormalities, such as focal slow wave activity, focal epileptiform activity were present in all patients. The abnormalities were unilateral in 27 patients and bilateral in five patients. Brain imaging (MRI) was abnormal in 6 children (18.75%).

Arachnoid cysts were found in three children, focal cortical dysplasia in one, hippocampal sclerosis in one case and ganglioglioma in one. These patients had no history of head trauma, meningitis, neonatal distress, or stroke. None of them had progressive symptoms suggesting increased intracranial pressure, and all responded to antiepileptic therapy. A temporal tumor was revealed in one case with histology supporting ganglioglioma.

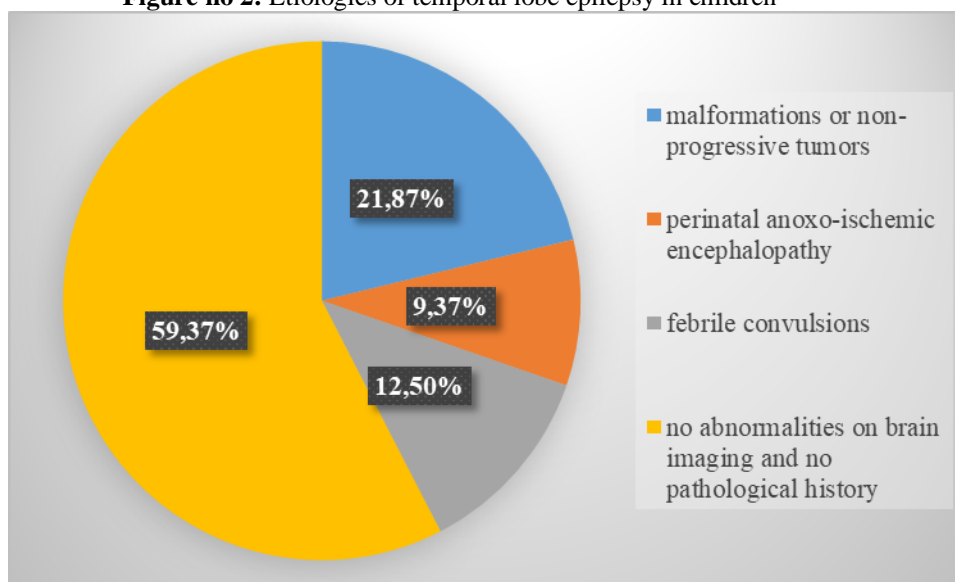
Classification according to probable etiology (Figure 2):

Group 1 consisted of six patients (18.75%) with malformations or non-progressive tumors on brain imaging.

Group 2 consisted of seven children (21.87%) with a pathological history before the onset of temporal lobe seizures (TLE). Three children with perinatal anoxo-ischemic encephalopathy and four others with complex febrile convulsions.

Group 3 included 19 children (59.37%) with no abnormalities on brain imaging and no pathological history.

Figure no 2: Etiologies of temporal lobe epilepsy in children



IV. Discussion

1. Semiology of temporal epilepsy (TLE) in infants and newborns aged 0 to 3 years :

Temporal lobe seizures that occur in infants and newborns are the most difficult to identify using only semiology. Unlike older children, infants and newborns are more likely to display epileptic semiology reminiscent of extratemporal seizures and generalized seizures. There is a relationship between ictal motor manifestations and age. Such manifestations include tonic, clonic, myoclonic, hypermotor seizures, and epileptic spasm. Despite their unilateral origin, such manifestations can appear bilateral and symmetrical; complicating attempts to lateralize the onset of seizures. They can also easily mimic frontal lobe seizures. The likely reason for the increased motor manifestations in temporal lobe epilepsy (TLE) in infants and newborns is the presence of focal pathology in the setting of incomplete myelination of the central nervous system. This is especially relevant when it comes to the limbic system, which is resistant to synchronization in an immature state. The occurrence of epileptic spasms in infants with pathology of the temporal lobe is probably secondarily to a rapid secondary generalization of focal-onset seizures through dysfunctional cortico-subcortical interactions (notably involving the thalamus, basal ganglia and other brainstem structures). This can lead to clinical and electrical semiological appearances, making it more difficult to identify the area with ictal onset.

One of the hallmarks of temporal lobe epilepsy (TLE) in adults is the onset of automatism, although automatisms can be seen in infants and newborns with temporal lobe epilepsy (TLE). They are generally simpler than those seen in older children and adults. These simpler automatisms include eating, gestural (groping of the hand) and blinking movements of the eyelids. This difference may be due to the limited repertoire of fine voluntary motor gestures in young children.

It is usually not possible for clinicians to properly assess auras at the onset of the attack. The first signs of such seizures in infants and newborns may be a cessation of behavior, or cyanosis of the lips.

2. Semiology of temporal lobe epilepsy (TLE) in preschool and school age children (3 to 6 years old) :

Unlike infants and newborns, preschoolers and school-aged children with temporal lobe epilepsy (TLE) have lateralized motor manifestations that are best developed with seizures. This age group is more likely to exhibit dystonic posture, head rotation, eye / mouth deviation.

In preschool children, more complex automatisms can be observed. These included eating automatisms observed in the youngest (0-3 years), looking around and / or clapping of the hands.

However, more complex automatisms are even less likely to be observed in this age group compared to older children (> 6 years) and adults. They are also less likely to lateralize properly on the ipsilateral hemisphere than in older children (50% vs. 100%). Children with temporal lobe epilepsy (TLE) are more likely to notice auras early in the seizure than their younger counterparts. This can still be difficult to assess (given the subjective nature of these symptoms). On auras, it may be more useful for clinicians to ask parents about the behaviors associated with such a phenomenon. For example, a child who cries or runs regularly to a parent, the crisis can be felt, like a fear attack, this is frequently localized at the level of the mesial temporal region. Conversely, children who experience a sensation of a stereotypical unilateral earplug at the onset of seizures may have an auditory aura localized to the contralateral superior temporal gyrus.

3. Semiology of temporal lobe epilepsy in older children (age > 6 years) and adolescents :

From the age of 6, children with temporal lobe epilepsy (TLE) show roughly the same seizure semiology as their adult counterparts. Some older children with temporal lobe epilepsy (TLE) report prodromes that last for several hours (or even several days) before the seizure begins. These prodromes can include headaches, irritability, insomnia, personality changes and / or a feeling of impending doom. However children with (TLE) are less likely to suffer from such prodromes compared to those with generalized tonicoclonic seizures. The rest are common in older children with TLE, particularly the best known is that reported by older children; is a sensation of localized or ascending epigastric heaviness (viscero-sensitive manifestations). The other auras are olfactory, gustatory, somatosensory, auditory, visual, visceral (oropharyngeal, abdominal, genital and retrosternal), or psychic (already seen, never seen, and / or dream state)

The seizures associated with (macropsia, micropsy, macrocausasia, microcausasia) originate from the lateral temporal region. Hallucinations of smell and taste typically arise from the uncus. Emotions such as fear, strangeness or embarrassment come from the amygdala

Compared to children 0 to 6 years old, older children and adolescents with TLE are more likely to have automatisms with their seizures. These automatisms include oro-alimentary automatisms (lip strokes and swallowing) and gestural automatisms (groping and aimless movement).

When they involve only one extremity, automatisms represent a reliable sign of lateralization for the ipsilateral hemisphere. Children over the age of 6 may also have toned or dystonic posture of one limb, especially the arm. Such a posture can help clinicians properly lateralize the seizure in the contralateral hemisphere.

However, care should be taken not to temporarily assume a temporal localization with such a posture, as it can also originate from the frontal lobe. When the seizures occur with loss of consciousness, it is thought that there is bilateral involvement of the limbic regions. Complex partial seizures of the temporal lobe can last for several minutes which is usually longer than seizures that originate from extra-temporal regions (e.g. frontal). These seizures are more susceptible to secondary generalization. Children with complex partial seizures may present post-stroke with confusion, disorientation, fatigue, headaches, and continued automatisms. Such behaviors can last from a few minutes to a few hours. However, they tend to be shorter than similar post-critical behaviors exhibited by adults.

4. Abdominal epilepsy :

Abdominal semiology of TLE is more common in children than in adults, these attacks are usually accompanied by altered consciousness, abdominal pain and vomiting. The duration of abdominal pain is limited to 10-15 minutes, such a diagnosis can be difficult for clinicians, requiring prolonged video-EEG monitoring.

5. Vegetative signs of temporal lobe epilepsy (TLE) in children :

The most frequently observed vegetative signs in temporal lobe seizures (TLE) in children are tachycardias. These tachycardias can be seen in up to 98% of temporal lobe seizures in children. This is especially true if they are of right hemispherical origin. Ictal bradycardia is rare in children and occurs in less than 4% of controlled seizures.

One of the most dramatic vegetative disturbances is hypoxemia and / or apnea that can occur during temporal lobe seizures in children. Half of the children have a quarter of their seizures captured in surveillance units with desaturations <90%. Cardiac hypoxemia is not specific to TLE and can be seen in secondarily generalized partial seizures. However, in young children 2 to 6 years old, apnea attacks may be the only manifestation of temporal lobe epilepsy. Such hypoxemia / apnea could theoretically exacerbate carotid chemoreceptor-induced bradycardia, causing additional respiratory suppression.

Harvey et al [1] suggested classifying temporal lobe epilepsies (TLE) into three groups based on etiology. These groups were designated TLE with structural abnormality, TLE with pathological history, and (TLE) cryptogenic. We applied this classification to a cohort of 32 children with newly diagnosed temporal lobe epilepsy TLE, and we obtained results very similar to those described by Harvey et al [1].

Group 1 with epilepsy and abnormal development of the temporal lobe includes children with malformations or tumors. Harvey et al chose the designation of the term developmental abnormalities based on the ancient, non-progressive, apparently benign nature of these lesions. These children presented extra hippocampal structural lesions in the temporal lobe. The coexistence of extra hippocampal lesions and hippocampal sclerosis is considered a double pathology. Proton magnetic resonance spectroscopy studies revealed a decrease in median temporal N-acetyl-aspartate / creatine ratios in 90% of patients with structural lesions of the extra-hippocampal temporal lobe (dysplasia, tumor, vascular malformation, calcification), providing evidence of dual pathology. The involvement of the median temporal structures could therefore constitute an important pathogenic factor in patients with an abnormality in the development of the temporal lobe. Arachnoid cysts have been considered to be arachnoid malformations. Although an association between

the focus of the epileptic seizure and the location of the arachnoid cysts is only found in a minority of cases. Seizure semiology and interictal EEG have suggested a link between the hypoplastic temporal lobe, partially replaced by a cyst, and the epileptic focus. Studies on the relationship between temporal lobe and hippocampus morphology, as well as epileptogenesis in patients with arachnoid cysts are not yet available. The hippocampal malformation has been described as a cause of familial febrile seizures and hippocampal sclerosis. Further research may shed light on a relationship between the onset of seizures and hippocampal temporal dysgenesis in patients with temporal arachnoid cysts. Cortical dysplasia is a well-known cause of epilepsy. Cortical dysplasia has been observed in a child. In our study we found a case of ganglioglioma.

Group 2 with pathological history, including complex febrile convulsions in four cases, perinatal anoxo-ischemic encephalopathy in three cases. A strong association between hippocampal sclerosis and complex febrile seizures has been reported in pathological studies and MRI. MRI can reliably detect children with hippocampal sclerosis. Head trauma at birth, hypoxia, encephalitis and meningitis can predispose to hippocampal sclerosis and temporal lobe epilepsy (TLE). Therefore, it is very likely that a seizure-causing injury, with or without febrile convulsions, is necessary to generate hippocampal sclerosis and temporal lobe epilepsy (TLE). In our patients a pathological history probably potentiated the lesions of the hippocampus.

Group 3 included children with cryptogenic temporal lobe epilepsy (TLE), consisting of children in whom no aetiology is apparent in clinical history or brain imaging. Data from the literature suggest that the epileptic semiology in infants may be very different from that observed in children or adults. It is difficult to assess the weakening of consciousness, a defining characteristic of complex partial seizures. Brockhaus and Elger [2] found that younger children lacked complex auras and automatisms, and often had symmetrical motor phenomena, as in our study.

V. Conclusion

Patients with significant antecedents are expected to have high risk of continued seizures of childhood temporal lobe epilepsy in the population of Algiers.

References

- [1]. Harvey AS, Berkovic SF, Wrennall JA, et al. Temporal lobe epilepsy in childhood: clinical, EEG, and neuroimaging findings and syndrome classification in a cohort with new-onset seizures. *Neurology* 1997; 49:960-8.
- [2]. Brockhaus A, Elger CE. Complex partial seizures of temporal lobe origin in children of different age groups. *Epilepsia* 1995; 36:1173-81.
- [3]. Kuzniecky R, Ho SS, Martin R, et al. Temporal lobe developmental malformations and hippocampal sclerosis: epilepsy surgical outcome. *Neurology* 1999; 52: 479-84
- [4]. Cendes F, Andermann F, Gloor P, et al. Atrophy of mesial structures in patients with temporal lobe epilepsy: cause or consequence of repeated seizures? *Ann Neurol* 1993; 34:795-801.
- [5]. Raymond AA, Fish DR, Stevens JM, et al. Association of hippocampal sclerosis with cortical dysgenesis in patients with epilepsy. *Neurology* 1994 ; 44:1841-5.
- [6]. Mathern GW, Babb TL, Vickrey BG, et al. The clinical-pathogenic mechanisms of
- [7]. hippocampal neuron loss and surgical outcomes in temporal lobe epilepsy. *Brain* 1995; 118:105-18.
- [8]. Mathern GW, Babb TL, Mischel PS, et al. Childhood generalized and mesial temporal epilepsies demonstrate different amounts and patterns of hippocampal neuron loss and mossy fibre synaptic reorganization. *Brain* 1996; 119: 965-87.
- [9]. Jackson CD, Berkovic SF, Duncan JS, et al. Optimizing the diagnosis of hippocampal sclerosis using MR imaging. *AJNR Am J Neuroradiol* 1993; 14:753-62.
- [10]. Grattan-Smith JD, Harvey AS, Desmond PM, et al. Hippocampal sclerosis in children with intractable temporal lobe epilepsy: detection with MR imaging. *AJR Am J Neuroradiol* 1993; 161:1045-8.
- [11]. Cendes F, Andermann F, Dubeau F, et al. Early childhood prolonged febrile convulsions, atrophy and sclerosis of mesial structures, and temporal lobe epilepsy: an MRI volumetric study. *Neurology* 1993; 43:1083-7.
- [12]. Broglin D. (2001) Neuro-vegetative manifestations in temporo-limbic seizures. In: G Avanzini, A Beaumanoir, M Mira (Eds). *Limbic seizures in children*. John Libbey, London , pp. 61- 78.
- [13]. Broglin D. (2001) Neuro-vegetative manifestations in temporo-limbic seizures. In: G Avanzini, A Beaumanoir, M Mira (Eds). *Limbic seizures in children*. John Libbey, London , pp. 61- 78.
- [14]. Fogarasi A, Jokeit H, Faveret E, Janszky J, Tuxhorn I. (2002) The effect of age on seizure semeiology in childhood temporal lobe epilepsy. *Epilepsia* 43: 638- 643.
- [15]. Ray A, Kotagal P. (2005) Temporal lobe epilepsy in children: overview of clinical semiology. *Epileptic Disorders* 7: 299- 307.
- [16]. Wyllie E, Chee M, Granstrom ML, Del Giudice E, Estes M, Comari Y, Pizzi M, Kotagal P, Bourgeois B, Luders H. (1993) Temporal lobe epilepsy in early childhood. *Epilepsia* 34: 859- 868.