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Epilepsy in Children and Imaging: About Thirty Cases

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ABSTRACT

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Keywords Epilepsy, Childhood, Diagnosis, Imaging. Epilepsy is a chronic condition that often begins in childhood and is characterized by the occurrence of spontaneous and recurrent epileptic seizures. The aim of our study is to identify the different etiologies of epilepsy in children and to highlight the importance of imaging in the etiological diagnosis, in the extension assessment and in the therapeutic management of epilepsy. Our study was carried out over a 5-year period from January 2014 and 2019, and involved 30 cases in the pediatric radiology department of the Rabat Children's Hospital. Seizures and loss of consciousness dominated the clinical picture. The etiological diagnosis in the majority of our series was based on CT scan and brain magnetic resonance imaging. The evolution under treatment was favourable for all our patients.

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Introduction

Epilepsy is considered to be a chronic neurological condition often encountered in the paediatric population and manifests itself as generalized and/or partial seizures.

Imaging, particularly MRI, is the key examination for the lesion in question because of its superiority over CT scanning.

Patients et Méthods

Retrospective study, based on a series of 30 cases admitted for epilepsy, collected in the pediatric radio department - Children's Hospital - Rabat - over a period of 5 years from January 2014 to February 2019.

In our series, 18 cases benefited from a cerebral CT scan, while cerebral MRI was performed in only 8 patients.

Results

The mean age of the patients was 7 years (Figure 1). Generalized seizures were represented in 60% of cases, followed by loss of consciousness in 38% of cases and altered consciousness in 12% of cases (Figure 2). In our series, brain CT scan was performed in 18 cases; it was normal in 08 cases and showed biventricular hydrocephalus in 5 cases, cerebrovascular accident in 4 cases and giant cell astrocytoma in 1 case (Figure 3). A cerebral MRI was performed in 8 cases, showed a temporal lobe signal abnormality in 03 cases (figure 6), tuberous sclerosis in 1 case (Figure 7) and a malformative syndrome in 1 case after symptomatic and etiological treatment.

Discussion

Epidemiology

Epilepsy affects about 0.5 to 1% of children under 16 years of age.

The age of the child, the context and the semiology of the epileptic seizure are elements of orientation towards a focal or multifocal lesion.

Perinatal hypoxo-ischemic lesions and malformations (including phacomatoses) each account for nearly 40% of epileptogenic lesions in children (2,5).

Pathophysiology

An epileptic seizure is a stereotypic, paroxysmal, transient disorder of consciousness, motor skills, behaviour and/or sensation, resulting from abnormal cortical neuronal discharges (2,3).

It is therefore a non-specific clinical sign indicating acute cerebral dysfunction. This may be the result of a brain injury. **Clinic**

It takes very diverse clinical forms, particularly in relation to the age of the child.

Several classifications of epileptic seizures and epileptic syndromes have been proposed (1,2,4).

These classifications are based on the partial or generalised nature of the epileptic seizures.

Differential diagnosis

Various conditions may be confused with epilepsy:

Meningitis

The subarachnoid hemorrhage.

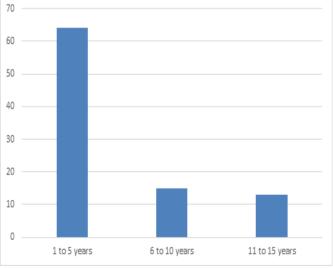


Figure 1. Age distribution

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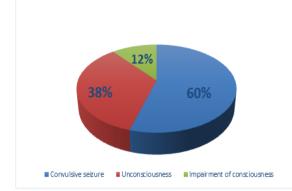


Figure 2. Distribution of clinical signs

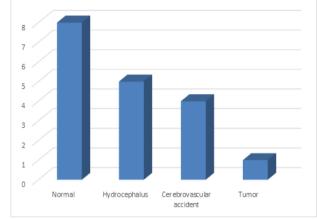


Figure 3. Brain CT scan results

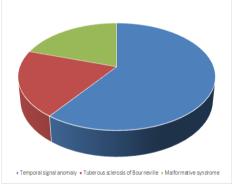


Figure 4. Brain MRI results

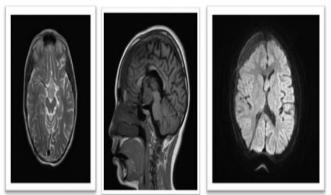


Figure 5 :Cerebral MRI showing a polylobed V3 tissue mass of intermediate T2 signal with diffusion restriction in relation to tumor residue associated with a right chronic subdural hematoma in a pediatric patient undergoing surgery for pilocytic astrocytoma who is currently having seizures.

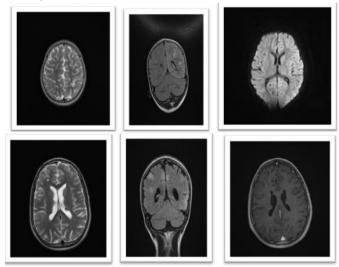


Figure 6. Cerebral MRI showing cortical and subcortical signal abnormalities in hyper T2 signal and left parieto-temporal Flair without diffusion translation suggesting laminar necrosis in a 5 year old child followed for EME.

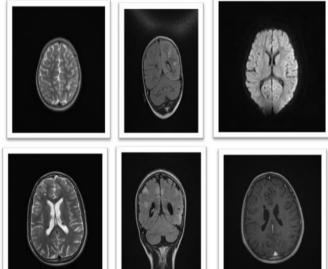


Figure 7. Cerebral MRI showing non-enhanced hypoT2 subependymal nodules after PDC injection with hypoT2 cortical-sub cortical peripheral signal abnormalities possibly related to Bourneville's tuberous sclerosis in a 14year-old boy followed for epilepsy with intellectual disability.

Imaging means

a. Magnetic resonance imaging (MRI)

Represents the key examination to detect the lesion involved when the clinic suggests a symptomatic seizure. Its sensitivity for detecting abnormalities related to epilepsy is estimated to be approximately 90%.

The diffusion-weighted sequence is of interest in the acute phase of hypoxo-ischemic, traumatic and metabolic lesions.

MRI can also monitor the impact of prolonged epileptic seizures (such as cortical laminar necrosis) on the cerebral parenchyma and helps in the development of prognosis (7,8). **b. Brain scan**

His epileptology indications have greatly diminished since the advent of MRI scans (figure 8).

The CT scan is reserved for emergency situations or as a second line of investigation, after MRI, to look for calcifications (6,7).

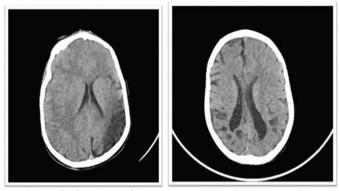


Figure 8. Cerebral CT scan showing bilateral parietooccipital sequelae with right cortical hemiatrophy in a 4year-old child admitted for epilepsy with psychomotor retardation.

c.Functional Imaging

Positron Emission Tomography (PET)

- ≻ Single Photon Emission Tomography (SPECT)
- ≻ NMR Spectroscopy.
- ≻ Functional MRI.

Etiological diagnosis

A. Genetic factors

Genetic factors are unquestionably present in many epilepsies, although it is not always possible to assess their significance (1,4).

A special case is that of genetic diseases: phacomatoses.

Bourneville's tuberous sclerosis is responsible for a high percentage of West's syndrome. In newborns and infants, MRI is very efficient in the detection of subependymal and intraparenchymal tubers that are not yet calcified (figure 7,9). In the Sturge-Weber syndrome, T1-weighted MRI after injection and T2-weighted MRI is good for assessing the extension of leptomeningeal angioma, which is highly epileptogenic and associated with a choroidal angioma and a cutaneous planar angioma with homolateral trigeminal topography(1,6).

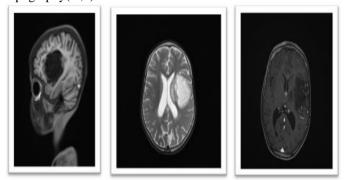


Figure 9. Cerebral MRI showing a left temporo-frontoinsular cortical lesion in T1 hyposignal, T2 hypersignal, and no enhancement after injection in favor with DNET in

an 8-year-old child admitted for EME. B.Factors acquired

1.Pre- and perinatal factors

Prenatal causes include cerebral malformations, strokes, CNS infections and intoxications occurring during intrauterine life (figure 10,11).

a.Malformations

Diffuse or localized anomalies of neuronal migration and gyration such as isolated or syndromic smoothencephaly, polymicrogyria, periventricular nodular heterotopias and agenesis of the corpus callosum are epileptogenic.

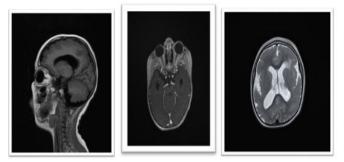


Figure 10. Cerebral MRI in sagittal T1 and axial T2 section with injection showing a thickened aspect of the cortex with rarefaction of the cortical grooves in favour of a pachygyria associated with anomalies of the

periventricular white matter in a 3 year old boy admitted for epilepsy with delayed motor acquisition.

b. Hypoxo-ischemic lesions of the newborn and older child

While transfontanel ultrasound remains the first-line examination in the newborn, MRI is the most effective examination for detecting areas of infarction in the acute and scarring stages (7,10).

2.Infections

- Cerebro-meningeal tuberculosis.
- ≻ Meningitis
- ≻ Measles.
- ≻HIV...

3.Head trauma

Seizures may occur early or late in the course of head trauma or later, achieving post-traumatic epilepsy, usually characterized by generalized tonic-clonic seizures from the outset (1,4,9).

4.Brain tumours

Brain tumours cause only about 1% of epileptic seizures in children. The most common tumours encountered in the context of epilepsy are low-grade tumours such as the dysembryoplastic tumour DNET, ganglioglioma, astrocytomr and oligodendroglioma (8,10).

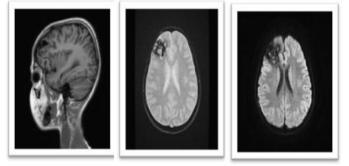


Figure 11 . Cerebral MRI showing on the right frontal level of a process formed of multiple serpiginous asignal structures realizing the salt-pepper appearance, with punctiform hypersignals in T1 and surrounded by a gradient echo asignal patch, more reminiscent of a cavernoma that bled in an 11-year-old girl admitted for epilepsy in an apyretic context.

5.Cerebrovascular diseases

Strokes, angiomatous malformations can cause seizures.

- 6.Toxic and/or drug-related factors
- > Acute lead poisoning, methanol are epileptogenic.
- > Epileptogenic metabolic changes such as disturbances in blood sugar control, hyponatremia and hypocalcemia.
- > Psychotropic drugs can trigger seizures through overdose or withdrawal.

7. Degenerative and metabolic disorders

Mesial or hippocampal sclerosis, which is less common in children than in adults, is characterized by temporal lobe atrophy secondary to neuronal loss, accompanied by a hyperintense T2 area indicative of a gliosis scarring response (7,12).

Leukodystrophies and other metabolic diseases are sometimes the cause of epileptic seizures.

Infarctions of an embolic or thrombotic nature may also manifest as epilepsy. Moya moya, most often encountered in the context of sickle cell disease (4,11).

Therapeutic management

The therapeutic attitude is based on daily background treatment with the aim of reducing the number of attacks.

An anticonvulsant seizure treatment may be combined in some cases.

Hygienic measures and therapeutic education remain essential for a better life for the child and his environment. **Conclusion**

Imaging is the technique of choice for detecting, establishing the nature and assessing the extension of brain lesions causing epilepsy.

Magnetic resonance imaging can also monitor the impact of prolonged seizures on brain parenchyma and assist in prognosis development.

Conflicts of Interest. The authors do not declare any conflict of interest.

Contributions by authors

All the authors have contributed to this work. All authors have read and approved the final version of the manuscript. **References**

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