Partial epilepsy in children
Clinical, electroencephalographic and radiological evaluation

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ABSTRACT

Objective: This study was designed to evaluate children with focal epilepsies whether cryptogenic or secondary, with their semiological features, etiological factors, electroencephalographic and neuro-radiological patterns as well as laboratory abnormalities, which might be of value in management, identifying outcome of these epilepsies as well as planning for their preventive measures. Patient and Methods: This study was carried on 25 patients (14 males and 11 females), their ages ranged from 3 to 15 years (mean 10.6±3.9), their duration of illness ranged from 2 weeks to 14 years (mean 5.8±4.7 years), their frequencies of seizures in the last month of illness ranged from 0 to 12 fits (mean 7.2±4.5), 2 cases had complex partial seizures (CPS), 4 cases had simple partial seizures (SPS), 19 cases were secondarily generalized, 18 patients were under treatment with antiepileptic drugs (AEDs). Patients were submitted to the following thorough history taking including history of seizures with their descriptions and antiepileptics used, full clinical and neurological examination. Conventional inter-ictal electroencephalographic examination, neuro-radiological assessment using C.T scan and MRI brain, and lastly laboratory investigations at three levels (routine, blood and urine amino acids, and TORCH screen). Results: The results of this study showed that: history of perinatal problems 10 patients, head trauma 3 patients, fever with hospital admission one patient and febrile convulsions in 2 patients, delayed developmental milestones (7 patients). Mental deterioration and learning problems (10 patients). Positive family history reported by 8 patients (32%) and abnormal past history in 8 patients (32%). Regarding clinical examination: abnormal general examination was observed in 2 patients (8%) while abnormal neurological examination in 8 patients (32%), in the form of motor and language deficits. Regarding EEG findings: Abnormal EEG reports in (20 patients) (80%), mainly spike and wave (10 patients) (19.5%), predominantly, temporal focus. Regarding radiological results: C.T brain was abnormal in 5 patients (20%) while MRI brain was abnormal in 8 patients (32%), the abnormalities included brain atrophy, mesial temporal sclerosis, A.V.M, encephalomalacia and brain infarction. Laboratory findings: normal routine laboratory investigations and amino acids level were observed in all patients, while TORCH screen showed that 4 patients had positive results either for rubella, toxoplasma or both. Lastly, the etiology of partial epilepsy in children was identified in 14 cases (56%). Conclusion: Perinatal insults (especially birth injury and difficult labour), and head trauma were common antecedents and etiological factors of childhood focal epilepsy. Motor and language deficits, mental deterioration and learning problems were represented in a relatively high percentage of children with focal epilepsy. Seizure semiology, ictal onset and EEG focus are valuable criteria to diagnose partial epilepsy. Temporal focus on EEG was the predominant type of foci in children with partial epilepsy. MRI was the most valuable neuroimaging examination to detect lesions. Combining EEG, CT and MRI help to ensure the diagnosis, focus localization and demonstration of the pathology of possible etiology. . (Egypt J. Neurol. Psychiat. Neurosurg., 2004, 41(1): 235-249).
**INTRODUCTION**

The overall incidence of childhood epilepsy from birth to 16 years is approximately 90 in 100,000 children per year\(^1\).

Berg\(^2\) reported that roughly half of all epilepsy begins during childhood and adolescence.

In developing countries, there is a high incidence of preterm and abnormal births. During infancy and early childhood, meningitis, tuberculosis, neurocysticercosis, and head trauma are common. These situations can be expected to increase the prevalence of secondary (symptomatic) epilepsy and of intellectual handicap in the child population\(^3\).

Previous neuroradiological studies have demonstrated that approximately one third of children with epilepsy have CT abnormalities\(^4\), however, CT may fail to detect abnormalities in up to 40% of patients with epileptogenic structural lesions, such as small tumours or developmental malformation\(^5\).

In children or young adults who present with complex partial seizures, the objective of MR imaging is to depict mesial temporal sclerosis, as well as other temporal or frontal lobe lesions\(^6\).

The aim of this study is to evaluate and throw light on childhood focal epilepsies with their semiological features, etiological factors, EEG, neuroradiological and laboratory patterns, to help in identifying whether it is cryptogenic or secondary to intracranial pathology. This might be of value in the management of cases and their outcome as well as it might have preventive measures aiming to reduce the prevalence of childhood focal epilepsy.

**PATIENTS AND METHODS**

**Patients:**

This study was carried out on 25 patients (14 males and 11 females) selected from Kasr El-Aini Epilepsy Clinic, Cairo University. Their ages ranged from 3 years to 15 years (mean age 10.6±3.9).

All patients were presented with focal epilepsy or focal epilepsy with secondary generalization, (evidence of focal onset from either clinical or EEG data). Duration of illness ranged from 2 weeks to 14 years (mean 5.8±4.7 years).

**Methods:**

All patients were submitted to the following:

I. **History Taking:**

The study took in consideration the following items in the interpretation of seizure description:

1. Personal history: Age, sex and handedness. Children were classified according to age into preschool children (from 3 years to 6 years) and school children (above 6 years up to 15 years).
2. Prenatal history: (pregnancy) History of medical maternal disease, fever, drug intake or exposure to irradiation.
3. Natal history: History of obstructed or prolonged labour, birth injury or usage of sedation or anaesthesia.
4. Neonatal history: History of apnea or cyanosis, delayed cry, twitching or convulsions, fever or jaundice.
5. Vaccination history.
6. Developmental history (physical and language): It was considered as delayed if there were delayed milestones including head support, sitting, standing, walking, speech or teeth eruption.
7. Family history: Consanguinity and history of similar disease.
11. History of anti-epileptic drugs (AEDs) received and duration of treatment.

II. Seizure Description:
Thorough history taking of epilepsy was done from the patients, their mothers and their relatives to describe the seizures in detail. History taking included the following items: seizure frequency (number of seizures in the last month), seizure type (focal or focal with secondary generalization), aura, ictus description, post-ictus state and history of status epilepticus.

III. Clinical Examination:
A. Complete general physical and medical examination. (Head and cranium, facies, stature, abdomen, chest and heart).
B. Neurological examination according to standardized neurological sheet, Neurology Department, Cairo University.

IV. Neurophysiological Assessment:
Conventional inter-ictal EEG:
All patients had their EEG Carried out at the Neurophysiological Department of Kasr El-Aini Hospital.
All EEGs were carried out under normal standard conditions (sedative premedication was given to most of the children), lying supine, completely relaxed in a quiet room. Hyperventilation for (3) minutes (only the awake children) together with intermittent photic stimulation were used as provocative methods.

The EEG tracing were analyzed carefully as regards: background activity, presence of generalized slowing or spike and wave, presence of focal slowing or spike and wave, or focal changes with secondary generalization.

V. Radiological Assessment:
A. CT scan of the brain: All patients performed CT brain.
B. MRI assessment of the brain: All patients performed MRI brain to detect any lesions that might not be detected by CT.

VI. Laboratory Profile:
Three levels of laboratory profiles were performed:
1. Level 1: all patients submitted to the following lab. tests: CBC, blood sugar, liver function tests, kidney function tests, electrolytes (Na⁺, K⁺, Ca++, Mg++).
2. Level 2: 13 patients submitted to qualitative blood and urinary amino acids analysis.
3. Level 3: 7 patients submitted to TORCH screen.

RESULTS

1. Personal History:
In the present study, the ages of the patients ranged from 3 years to 15 years (mean age 10.6±3.9). Preschool children were 6 patients (24%), and their ages ranged from 3 years to 6 years (mean 4.9±1.4). School children were 19 patients (76%), and their ages ranged from 7 years to 15 years (12.4±2.4). Fourteen patients (56%) were males and 11 were females (44%). 22 patients (88%) were right handed and 3 (12%) were left handed.
No significant difference between patients with focal epilepsy and patients with...
focal epilepsy with secondary generalization as regards age or sex distribution (P>0.05).

The duration of illness ranged from 2 weeks to 14 years (mean 5.8±4.7 years).

2. **Prenatal History:**
   Prenatal history was abnormal in 2 patients (8%) in the form of history of drug intake (medication for renal colic, for few days) and normal in 23 patients (92%).

3. **Natal History:**
   Natal history was abnormal in 6 patients (24%) in the form of history of birth injury (torsion of umbilical cord), and history of prolonged labour, and normal in 19 patients (76%).

4. **Neonatal History:**
   Neonatal history was abnormal in 8 patients (32%) in the form of cyanosis, delayed cry and convulsions and normal in 17 patients (68%).

5. **Vaccination history:**
   All patients received routine vaccinations at scheduled times.

6. **Developmental History:**
   Developmental milestones were delayed in 7 patients (28%) and normal in 18 patients (72%).

7. **Family History:**
   Positive family history has been reported by 8 patients (32%), 6 patients of them had a positive consanguinity, one patient had a family history of epilepsy (His cousin had falling attacks “atonic fits”?) and one patient had positive history of both consanguinity and epilepsy (His uncle had post traumatic fits) in the family. Negative family history had been reported by 17 patients (68%).

8. **Past History:**
   Past history was abnormal in 8 patients (32%), 3 of them had a past history of head trauma, 2 patients had an infantile hemiparesis, 2 patients had febrile convulsions, and one patient had fever and admitted in a fever hospital.

   Features of history of studied patients were demonstrated in table (1).

9. **Education level,** history of scholastic mis-achievement or history of intellectual and behavioural deterioration (Table 2).

   Regarding preschool children, 3 of them had intellectual and behavioral deteriorations (as recorded by their caregivers) while the other 3 showed normal intellectual development.

   Regarding school children, 10 of them were educated, (only 2 of them showed intellectual deterioration while 8 had normal development) and 9 were not educated (5 of them showed abnormal intellectual development while 4 were normal).

10. **AEDs received:**
    Out of the 25 patients, 7 patients (28%) received no medication, 9 patients (36%) received monotherapy and 9 patients (36%) received polytherapy (Table 3).

    Out of the 9 patients who received monotherapy, 6 patients of them (66.7%) were on VPA, 3 patients (33.3%) were on CBZ.

    Out of the 9 patients who received polytherapy, 5 patients (55.6%) were on CBZ and VPA, 2 patients (22.2%) were on CBZ and PHT, one patient (11.1%) was on VPA and PHT and one patient (11.1%) was on CBZ and BZ.

    The duration of treatment ranged from weeks to 14 years (mean 4.1±4.4 years).
Regarding response to treatment of patients receiving treatment, 4 patients (22.2%) had a good seizure control, 8 patients (44.5%) had a fair control while 6 patients (33.3%) had bad control (intractable), all of them were taking more than one drug and had more than one type of seizure (Fig. 1).

II. Seizure Description:
A. Seizure frequency:
The frequency of fits ranged from 0-12 seizures in the last month of illness (mean 7.2±4.5).
B. Types of seizures:
Partial seizures were observed in 6 patients (24%) [4 of them (16%) had simple partial seizures while 2 (8%) had complex partial seizures]. On the other hand, partial seizures with secondary generalization were observed in 19 patients (76%) all of them had generalized tonic clonic convulsion (Table 4).
C. Aura:
Auras were observed in 8 patients (32%), absent in 17 patients (68%). Observed auras were in the form of lip smacking, automatism, eye blinking, deviation of angle of mouth and electric sensation of hand.
D. Post-ictal manifestations:
were mainly lassitude, fatigue, sleep, headache.
F. History of status epilepticus:
It is positive in 8 cases (32%).
E. Other types of seizures:
Myoclonic seizures were observed in 4 cases (16%) and absence seizures were observed in 3 cases (12%).

III. Examination:
A. General examination:
Abnormal findings were recorded in 2 patients (8%) in the form of protruded tongue with increased salivation, mental subnormality and stunted growth.
B. Neurological examination:
Abnormal findings were recorded in 8 patients (32%) in the form of hemiparesis and dysphasia.

IV. Neurophysiological Assessment:
Results were as follow:
Normal EEG reported in 5 patients (20%) and abnormal records were reported in 20 patients (80%).
Those with abnormal EEG had the following changes (Table 5):
1. Background activity: It was normal in 15 patients (75%) and abnormal in 5 patients (25%).
2. Generalized slowing was recorded in 3 patients (15%).
3. Focal slowing: was recorded in 3 patients (15%).
4. Focal spike and wave: was recorded in 19 patients (95%), 5 (26.3%) had right sided focus, 8 (42.1%) left focus and 6 (31.6%) had bilateral foci.
The origin of the focus was fronto-temporal in 10 patients (52.6%), centrotemporal in 4 patients (21%), temporal in 3 patients (16.8%), centrontempo-temporal in one patient (5.3%) and centro-parietal in one patient (5.3%).
So temporal origin of focus is present in 18 patients (94.7%) of those having focal spike and wave (Table 6).
5. Secondary generalization: was detected in the EEG of 11 patients (55%) of those with abnormal EEG and 11 patients (57.9%) out of 19 patients with focal spike and wave.

V. Radiological Results: (Table 7)
A. CT scan of the brain:
Normal CT results were recorded in 20 patients (80%) and abnormal CT was recorded in 5 patients (20%).
Two patients (40%) had unilateral brain atrophy, one patient (20%) had
bilateral occipital encephalomalacia, one (20%) had temporo-parietal post-encephalitic encephalomalacia, and one (20%) had unilateral cerebral infarction.

B. MRI:
Normal results were recorded in 17 cases (68%) and abnormal results were recorded in 8 cases (32%).
Two patients (25%) had unilateral mesial temporal sclerosis, two patients (25%) had unilateral AVM, two patients (25%) had unilateral brain atrophy, one patient (12.5%) had unilateral occipital encephalomalacia and one patient (12.5%) had post-encephalitic encephalomalacia.

No statistical significant difference between patients with focal epilepsy and patients with focal epilepsy with secondary generalization as regards CT or MRI findings (P>0.05).

VI. Lab. Profile: (Table 8)
Level 1 lab profile (Routine):
CBC, liver function tests, kidney function tests, serum glucose, electrolytes (Na**, K**, Ca**, Mg**).
Results were within normal ranges according to patient ages:
Level 2 (Aminoacids):
Qualitative plasma aminoacids and urinary amino acids: All the group of patients had negative results.
Level 3 (TORCH screen):
Results of the group submitted to TORCH screen were as follow: 3 cases (42.8%) had negative results, 4 cases (57.1%) had positive results.
Out of the 4 cases having positive results, 2 cases had positive rubella (IgG), one case had positive toxoplasmosis and one case had positive rubella and toxoplasmosis (IgG).

VII. Etiology: (Fig. 2)
Of the 25 cases, etiology was identified in 14 cases (56%).
Out of the 14 cases with identified etiology, 5 cases (20%) had birth injury, 2 cases (8%) were post-traumatic, 2 cases (8%) had congenital malformation (AVM), 2 cases (8%) had MTS, 2 cases (8%) had unilateral brain atrophy and one case (4%) was post-encephalitic.

Table 1. Features of history in children with focal epilepsies.

<table>
<thead>
<tr>
<th>History</th>
<th>Abnormal history (or positive)</th>
<th>Normal history (or negative)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Prenatal history</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Natal history</td>
<td>6</td>
<td>24</td>
</tr>
<tr>
<td>Neonatal history</td>
<td>8</td>
<td>32</td>
</tr>
<tr>
<td>Developmental history</td>
<td>7</td>
<td>28</td>
</tr>
<tr>
<td>Family history</td>
<td>8</td>
<td>32</td>
</tr>
<tr>
<td>Past history</td>
<td>8</td>
<td>32</td>
</tr>
<tr>
<td>Intellectual, behavioral or scholastic mis-achievement</td>
<td>10</td>
<td>40</td>
</tr>
</tbody>
</table>
Table 2. Demonstration of abnormal mentality and behavior or scholastic mis-achievement.

<table>
<thead>
<tr>
<th>Patients</th>
<th>Normal</th>
<th>Abnormal</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>Preschool children</td>
<td>3</td>
<td>12</td>
<td>3</td>
</tr>
<tr>
<td>School children</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Educated</td>
<td>8</td>
<td>32</td>
<td>2</td>
</tr>
<tr>
<td>Non-educated</td>
<td>4</td>
<td>16</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>15</td>
<td>60</td>
<td>10</td>
</tr>
</tbody>
</table>

Table 3. Pattern of medication in patients with partial epilepsy.

<table>
<thead>
<tr>
<th>Anti-epileptic drugs received</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
</tr>
<tr>
<td>No medication</td>
<td>7</td>
</tr>
<tr>
<td>Monotherapy</td>
<td>9</td>
</tr>
<tr>
<td>Polytherapy</td>
<td>9</td>
</tr>
</tbody>
</table>

Fig. (1): Response to anti-epileptic drugs.

Table 4. Demonstration of types of seizures.

<table>
<thead>
<tr>
<th></th>
<th>Partial</th>
<th>Partial with secondary generalization</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Simplex</td>
<td>Complex</td>
</tr>
<tr>
<td>No. of cases</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Percentage</td>
<td>16%</td>
<td>8%</td>
</tr>
</tbody>
</table>
**Table 5. Different electroencephalographic abnormalities.**

<table>
<thead>
<tr>
<th>EEG changes</th>
<th>Patients with abnormal EEG (20)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
</tr>
<tr>
<td>I. Background:</td>
<td></td>
</tr>
<tr>
<td>a. Normal</td>
<td>15</td>
</tr>
<tr>
<td>b. Abnormal</td>
<td>5</td>
</tr>
<tr>
<td>II. Generalized slowing</td>
<td>3</td>
</tr>
<tr>
<td>III. Focal slowing</td>
<td>3</td>
</tr>
<tr>
<td>IV. Focal spike and wave</td>
<td>19</td>
</tr>
<tr>
<td>V. Secondary generalization</td>
<td>11</td>
</tr>
</tbody>
</table>

**Table 6. Demonstration of origin of EEG focus.**

<table>
<thead>
<tr>
<th>Origin of focus</th>
<th>No. of patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fronto-temporal</td>
<td>10</td>
<td>52.6</td>
</tr>
<tr>
<td>Centro-temporal</td>
<td>4</td>
<td>21</td>
</tr>
<tr>
<td>Temporal</td>
<td>3</td>
<td>15.8</td>
</tr>
<tr>
<td>Fronto-temporo-central</td>
<td>1</td>
<td>5.3</td>
</tr>
<tr>
<td>Centro-parietal</td>
<td>1</td>
<td>5.3</td>
</tr>
</tbody>
</table>

**Table 7. The distribution of radiological abnormalities.**

<table>
<thead>
<tr>
<th>Radiological Findings</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>C.T Findings:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unilateral brain atrophy (central and cortical)</td>
<td>2</td>
<td>40</td>
</tr>
<tr>
<td>Bilateral occipital encephalomalacia</td>
<td>1</td>
<td>20</td>
</tr>
<tr>
<td>Temporo-parietal postencephalitic encephalomalacia</td>
<td>1</td>
<td>20</td>
</tr>
<tr>
<td>Unilateral cerebral infarction</td>
<td>1</td>
<td>20</td>
</tr>
<tr>
<td>MRI Findings:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unilateral MTS</td>
<td>2</td>
<td>25</td>
</tr>
<tr>
<td>Unilateral AVM</td>
<td>2</td>
<td>25</td>
</tr>
<tr>
<td>Unilateral brain atrophy</td>
<td>2</td>
<td>25</td>
</tr>
<tr>
<td>Bilateral occipital encephalomalacia</td>
<td>1</td>
<td>12.5</td>
</tr>
<tr>
<td>Postencephalitic encephalomalacia</td>
<td>1</td>
<td>12.5</td>
</tr>
</tbody>
</table>

**Table 8. The distribution of lab results.**

<table>
<thead>
<tr>
<th>Laboratory investigations</th>
<th>No. of submitted patients</th>
<th>Negative results</th>
<th>Positive results</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Level 1 (Routine)</td>
<td>25</td>
<td>25</td>
<td>100</td>
</tr>
<tr>
<td>Level 2 (Amino acids)</td>
<td>13</td>
<td>13</td>
<td>100</td>
</tr>
<tr>
<td>Level 3 (TORCH)</td>
<td>7</td>
<td>3</td>
<td>42.8</td>
</tr>
</tbody>
</table>
DISCUSSION

Partial epilepsy constitutes between 37 and 66% of childhood epilepsies, depending on the considered age-range and the modality of recruiting.

Focal childhood epilepsies are either idiopathic or symptomatic. Dalla Bernardina et al. reported the important criteria for diagnosing idiopathic partial epilepsies in childhood which include absence of neurological or intellectual deficit, family history of epilepsy, onset after 18 months of life and seizures are usually brief and rare with a good response to treatment.

On the other hand, aetiological symptomatic partial epilepsy in children include diverse causes which may be congenital malformation, disorders of neuronal migration, traumatic parenccephaly, vascular, inflammatory, tumors or may occur without any obvious definable causes.

The age distribution of patients in the present study ranged from 3 to 15 years. In other studies on childhood epilepsy, ages of patients ranged from 3 months to 16 years in Harvey series, one year to 16 years in Berg series, and from 16 months to 12 years in Brockhaus and Elger series.

In the present study, sex distribution was 56% male and 44% females, 46% males and 54% females in Harvey series, 50.1% males and 49.9% females in Berg series, 41.4% male and 58.6% females in Brackhous and Elger series and 49.4% male and 50.6% females in Berg series.

As regards type of seizures in the present study, 4 cases (16%) had SPS, 2 cases (8%) had CPS and 19 patients (76%) developed 2ry generalization. It was 16.3%, 47.3% and 52.9% respectively in Berg series.
Seizure frequencies in the last month of illness ranged from 0 to 12 fits which was nearly equal to frequency detected by Harvey et al.\textsuperscript{10} and highly lower than frequencies detected by Brackhouse and Eleger\textsuperscript{12}.

Features of ictal and post-ictal manifestations in this study were nearly in accordance with that reported by many authors\textsuperscript{10,12,14}, who observed that automatism, lip smacking, head turn, occasional autonomic manifestations, fear, vocalization and focal tonic and/or clonic convulsions were the main ictal onset manifestations, on the other hand; confusion, lassitude and sleep were the main post-ictal features.

O’Dohoe\textsuperscript{15}, mentioned that despite the many unusual types of seizures in childhood, tonic-clonic convulsions were the commonest epileptic manifestation of childhood. This occurred in 19/25 patients (76%) of our series, half of Berg series\textsuperscript{11} but (14.3%) of Harvey series\textsuperscript{10}.

Neurologic deficits were found in 32% of our cases in the form of motor and language deficits. Higher results were found by Bauer et al.\textsuperscript{16} (59%) and El-Melegi\textsuperscript{17} (80%). In controversy, 11.1% only were found in Harvey series\textsuperscript{10}.

Regarding prior antecedents and etiological factors of focal epilepsy in children, El-Melegi\textsuperscript{17} and Mulligan et al.\textsuperscript{18} concluded that seizures are most likely to occur in infants with perinatal problems especially delayed initiation of respiration, prolonged cyanosis, delayed cry, or birth injuries. This was in accordance with the results of this study which reported, 10 patients (40%) with perinatal problems. Again, perinatal problems were found in 30% of Scarpa and Carassini series\textsuperscript{19}, 13% of Mayanagi series\textsuperscript{20}, 21% of Murakami series\textsuperscript{21}, but only 1.6% of Harvey series\textsuperscript{10}.

Despite advances in diagnostic capabilities, the unknown etiologic category remain larger than any known etiology for all age groups\textsuperscript{22}.

Associated developmental neurologic disorders (e.g. cerebral palsy and mental retardations, head trauma, birth injuries, fever, and cerebrovascular disease are the most commonly identified causes\textsuperscript{22}).

Children who have experienced febrile seizures are at increased risk of developing later unprovoked seizures compared with children without history of febrile seizures\textsuperscript{23-25}. Although the risk of unprovoked seizures after febrile seizures is only a few percent, it is several times higher than what is seen in the general population\textsuperscript{24,25}.

In the present study, 2/25 of patients (8%) had a past history of febrile convulsions. This is nearly like what is found by Scarpa and Carrassini\textsuperscript{19} (10%) and Berg\textsuperscript{2} (13.9%) who concluded that no specific association with focal epilepsy and febrile seizures. In controversy, higher results were recorded by others\textsuperscript{10,20,25,26}.

As regard head trauma, in the present study, 3/25 of patient (12%) had a past history of significant head trauma. In other reports post-traumatic epilepsy was noted to be ranged from 1.6% to 10.9%\textsuperscript{10,17,19,20}.

Late post-traumatic epilepsy is defined as seizure that develop one week or more after head injury. The first seizure usually occur within the first year after the injury in more than 50% of cases. However, in about 25% of the patients, epilepsy is delayed for more than 4 year\textsuperscript{24}.

Jennett and Van de Sando\textsuperscript{26} have found that EEG studies were unhelpful in predicting the possibility of late epilepsy. However, EEG abnormalities were commoner in those who developed late epilepsy, and this only reflects the greater degree of brain damage in these patients. There are also patients who develop EEG abnormalities but never have any seizures, whereas some patients develop
post-traumatic epilepsy and have normal EEG\textsuperscript{27}.

In the present study, EEG abnormalities were found in the 3 cases with post-traumatic epilepsy, in the form of unilateral spike and wave (temporal, centro-temporal and fronto-temporal).

Regarding family history of epilepsy only 2 patients had positive family history of epilepsy (8%). This was supported by Casetta et al.\textsuperscript{(28)} who reported that by the age of 25 years, nearly 9% of children of mothers with epilepsy and 2.4% of children of affected farthers, develop epilepsy.

Moreover, Casetta et al.\textsuperscript{(28)} concluded that personal history of febrile convulsions and family history of epilepsy in the first degree relation were independent risk factors for development of focal epilepsy in children.

In the present study, 20/25 of patients (80%) had abnormal EEG. This is nearly in agreement with El-Koureshi\textsuperscript{29} results who detected EEG abnormalities in 8% of cases, and lower than others\textsuperscript{17,30,31} who found EEG abnormalities in 100% of their cases with partial epilepsy.

It is important to remember that about 10-40% of patients with epilepsy don’t show epileptiform abnormalities on routine EEG. So normal or non-specifically abnormal EEG never excludes the diagnosis of epilepsy\textsuperscript{27}.

In the present study, epileptic foci were recorded in EEG of 19 patients (76%), all of them showed spike and wave, 3 of them showed focal slowing. This was in agreement with some reports\textsuperscript{9,29,31} who detected epileptic foci in 78%, 47% and 75% of their patient’s EEG respectively, and lower than Scarpa and Carrassini\textsuperscript{19} who detected epileptic foci in 90% of their patient’s EEG. Moreover, temporal foci were the predominant type, in this study which goes with results of 2 other studies\textsuperscript{17,19}.

In the present study, CT abnormalities, were detected in 5 patients (20%), which was in agreement with many authors\textsuperscript{29,32,33}, who demonstrated focal lesions by CT scan in 23-26% of children with partial epilepsy and lower than that obtained by many investigators\textsuperscript{16,17,34-37}, who reported focal CT abnormalities in 35-76.6% of children with partial epilepsy.

In the present study, brain atrophy (8%) and encephalomalacia (occipital and temporoparietal) (8%) were the predominant CT abnormalities, one CT (4%) revealed unilateral old cerebral infarction. In Yang et al.\textsuperscript{38} focal atrophy and hemiatrophy were also the predominant CT abnormalities (other abnormalities were in the form of porencephalic cyst and brain tumours). Also, CT abnormalities in El-Melegi series\textsuperscript{17} were mainly in the form of atrophy and hemiatrophy. (Hydrocephalus with porencephalic cyst, brain abscess and subdural collections were detected by CT).

Diffuse cerebral atrophy was found in 14% of patients with focal epilepsy before the age of twenty\textsuperscript{36}. Hemiatrophy, focal atrophy, hydrocephalus, or cystic expansion of the ventricles and subarachnoid space are clearly shown by CT scan in epileptic children\textsuperscript{39}.

On the other hand, cerebral infarction and encephalomalacia were the predominant CT abnormalities in El-Koureshi study\textsuperscript{29}.

However, CT might miss common epileptogenic lesions which can be clearly demonstrated by MRI such as hippocupal sclerosis, cortical dysplasia, AVM and cavernous malformations\textsuperscript{40-43}.

MR imaging has been proved to be useful for the evaluation of seizures, and should be the first imaging examination used in the detection of underlying, potentially life threatening pathologic processes\textsuperscript{11}.

In the present study, MRI was the most valuable imaging examination for detection of
lesions that could not be detected by CT. MRI revealed (AVM) in 2 cases, one of them had normal CT, the other case had CT showing old infarct. Also MRI revealed (MTS) in 2 cases who had normal CT.

This was in agreement with many authors\textsuperscript{41-43} who reported that MRI is more sensitive than CT in detecting potentially epileptogenic lesions such as cortical dysplasia, hamartoma, cavernous malformations.

Moreover, MRI with its improved resolution and interpretations detect abnormalities correlating with the location of epileptogenic focus in the EEG in 22\% of children with focal epilepsies, with normal CT scan\textsuperscript{46}, supporting the results of this study about temporal focus in EEG and its involvement by organic lesion on MRI.

Temporal lobe changes are more evident using MRI techniques, unilateral temporal lobe atrophy is generally reported more often after MRI than CT scanning, owing to the superior imaging quality of MRI in the region of the temporal lobe, as it is close to the skull base with bone artifacts that makes the assessment of temporal lobe difficult on CT scanning\textsuperscript{20,41}.

The basic features of MRI changes in MTS are: decreased signal and atrophic change in T1 weighted images, and increased signal in T2 images\textsuperscript{21,26,42,43}. These features are nearly in agreement with MRI features of our 2 cases with MTS.

In cases with AVM; MRI is diagnostic in both the large as well as the small sized lesions and help to identify the feeding artery\textsuperscript{11}. Furthermore, MRI can be used to demonstrate haemosiderin in the brain in cases of post-traumatic epilepsy and so can assess its epileptogenic potentiality\textsuperscript{44,45}.

Generally children with focal epilepsies are relatively refractory to treatment than those with generalized epilepsies, this was in agreement with our results, as most of our patients receiving treatment were either fairly controlled in 2 patients (44.5\%) or with bad control in 6 patients (33.3\%). A fact that was supported by many authors\textsuperscript{3,14,19,23} who added that focal epilepsies in children secondary to a known focal lesions are more refractory than these without identified abnormality.

Furthermore, refractory response to treatment was associated with presence of neurological deficits, intellectual deterioration and radiologically positive CT or MRI brain\textsuperscript{3,19}.

\textbf{REFERENCES}

الملخص العربي
الصرع العضني في الأطفال

الهدف من هذا البحث دراسة وتقديم حالات الصرع العضني في الأطفال من ناحية معرفة الأسباب والمظاهر
الإكلينيكية ووصف النوبات المختلفة والعلاج المسبب ودراسة نتائج رسم المح كهربائي والأنشطة المقطعة والربين
المغناطيسي على النحو والفحصات العملية والتي يمكن أن تكون لها دور في العلاج والتخطيط لوسائل مترابطة
من هذا المرض.

وقد أشارت هذه الدراسة على خمسة وعشرين طفلاً (14 ذكر، 11 إناث) تراوحت أعمارهم بين 3 سنوات
إلى 15 سنة (5.10 + 3.9) ، بينما تراوحت مدة الإصابة بالمريض 0.40 إلى 14 سنة بمرحلة
قبد (5.8 + 4.7) وبلغ معدل حدوث النوبات السرعتي من صفر إلى 12 نوبة في الشهر الأخير من المرض
بمتوسط قدره (7.2 + 4.5) وكان جميع الأطفال مصابين بنوبات جزئية منهم 4 أطفال مصابين بنوبات جزئية
بسيطة، وطلق مصابين بنوبات جزئية مركبة، وتسعة عشر طفلاً مصابين بنوبات جزئية متضوورة إلى نوبات عامة
(عامة ثانية).

وقد تم إجراء الفحوصات والبحث التالية على هؤلاء المرضى: تناول التاريخ المرضي وإجراء فحص إكلينيكي
شمل، رسام المح كهربائي المعتاد (في الفترة بين النوبات)، فحص للنحو بالأنشطة المقطعة بالكمبيوتر، فحص للمخ
بالربين المغناطيسي، وفحوصات عمومية.

وقد أظهرت نتائج البحث إصابات وخلع فترة الولادة وفترة المحتوي بها وظل المتبقي في 10 حالات
(40%)، تأخر في مراحل النمو في 7 حالات (28%)، تأخر في القرارات العقلية أو تأخر في التعليم الدراسي في 10
حالات (40%)، إصابة الرأس (3 حالات)، تشنجات حرارية (حالتين)، حمى (حالة واحدة).

وتراوح عدد مرات حدوث النوبة بين صفر إلى 12 نوبة في الشهر وهي جزئية في 6 مرضى (24%) وجزئية مع
الانتشار الكافي في 19 حالة (76%) وفترة العلاج تراوحت بين أسباعية إلى 14 سنة مع استجابة متغيرة بالنسبة
للذاتية المضادة للصرع. وقد اتضح أن الفحص الإكلينيكي العام غير طبيعي في حالتين (68%) وأن فحص الجهاز
العصبي غير طبيعي في 8 حالات (32%) (العصابات الحركية ولغوية)، نتائج رسام المح كهربائي غير طبيعي في 20
حالة (80%)، نتائج الأنشطة المقطعة غير طبيعية في 5 حالات (20%)، نتائج الرنين المغناطيسي غير طبيعية في 8
حالات (32%)، الفحوصات العملية كانت طبيعية. ما عدا فحوصات المستوى الثالث، حيث وجدت 4 حالات
موجبة، ومن هنا يتضح أن من العوامل الكبيرة الحدث السرعتي للصرع العضني في الأطفال هو عل واسباب
الولادة وظل المتبقي في الرأس وأرتفاع درجة الحرارة، كثرة حدوث الإصابات الحركية واللغوية والعقلية
وصعوبات التعليم الدراسي للأطفال المصابين بالصرع العضني، ضعف إنتاج رسام المح كهربائي والأنشطة
المقطعة والربين المغناطيسي لعدة تحديد موقع الوراء العصبية التي قد تفيد قطعاً في العلاج وقد تكون قابلة للتدخل
الجراحي.