Post-Traumatic Epilepsy: Clinical, Neurophysiological and Neuroimaging Study

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ABSTRACT

Post-traumatic epilepsy (PTE) is a recurrent seizure disorder due to traumatic injury of the brain. There is controversy regarding the precise mechanism by which epilepsy may results from traumatic brain injury. Mesial temporal lobe sclerosis (MTS) is reported as a major risk factor for intractability of posttraumatic epilepsy. We aimed from this work to revise patients with post-traumatic epilepsy, to define risk factors, and assess the clinical, neurophysiological and neuroradiological characteristics. The frequency of mesial temporal epilepsy in contrast to neocortical epilepsy was also assessed in these patients. Twenty-three patients with post-traumatic epilepsy were included in this study. Clinical assessment, video EEG monitoring and MRI brain results were reviewed. We found that 14 patients (60.9%) with neocortical epilepsy (NCE), 8 patients (34.8%) of them had their trauma below or equal to 10 years and 6 patients (26.1%) had their trauma above 10 years old. We found also 8 patients (34.8%) with mesial temporal epilepsy (MTE), 5 patients (21.8%) had their trauma below or equal to 10 years and 3 patients (13%) had their trauma above 10 years. There was one patient (4.3%) with mixed neocortical and mesial temporal epilepsy. Of these patients, 6 had temporal lobectomy with successful post-operative results and the diagnosis of mesial temporal sclerosis was pathologically definite in 5 patients. We concluded that MTS could occur in patients with PTE in young or old ages. Detection of MTS is mandatory for all patients with PTE as resective surgeries of these patients gave a good outcome for the control of their intractable epilepsy. (Egypt J. Neurol. Psychiat. Neurosurg., 2007, 44(2): 737-749)

INTRODUCTION

Post-traumatic epilepsy (PTE) is defined as recurrent seizure disorder due to injury to the brain following trauma¹. It is an established consequence of head injury and its incidence is highest among young adults as they are more prone to head injury²⁻³. PTE accounts for 20% of symptomatic epilepsy in the general population and 5% of all epilepsy patients referred to specialized epilepsy centers⁴⁻⁵. In military series, the incidence of PTE is much higher (up to 50%), as these studies also include many patients with penetrating head injuries⁶.

PTE is classified as immediate seizures (less than 24 hours after injury), early seizures (less than 1 week after injury) and late seizures (more than a week after injury)⁷. The incidence of immediate seizure is 1-4%, early seizures 4-25% and late seizures 9-42% in civilian head injuries⁸⁻⁹.

Definitions for severity of head injury vary, but one of the most established paradigms is that proposed by Annegers et al., in which head injury is classified as mild, moderate, or severe. Mild injuries are defined by lack of skull fracture and a period of posttraumatic amnesia or loss of consciousness that is 30 minutes or less. Moderate injuries may or may not be associated with skull fractures, but there is a period of 30 minutes to 24 hours of posttraumatic amnesia or loss of consciousness. Severe injuries are characterized by brain contusion, intracranial hematoma, or 24 hours or more of either unconsciousness or posttraumatic amnesia⁹.
It was found in some studies that mesial temporal lobe epilepsy may result from traumatic brain injury (TBI) and occurs mainly in young children, while neocortical epilepsy occurs later in life. This may be because of the vulnerability of the developing brain to trauma\(^9,10\). In up to two-thirds of patients, late post-traumatic seizures are generalized or focal with secondary generalization, and often both seizure types may coexist\(^{11,12}\).

The incidence of subclinical seizure activity is much higher than that of overt seizures and is even higher in penetrating brain injuries than in non-penetrating injuries. In one series, the reported incidence of combined non-convulsive seizures and overt seizures was 22%, of these the incidence of non-convulsive seizures was about 52%\(^{13,14}\).

Much less is known about the characteristics of TBI, which is associated with increased risk of seizures. However, certain risk factors have been consistently identified, placing TBI patients at significant risk of developing post-traumatic epilepsy. These risk factors include duration of loss of consciousness, missile injuries, intracerebral hemorrhage, diffuse cerebral contusions, prolonged (3 days) post-traumatic amnesia, acute subdural hematoma with surgical evacuation, early post-traumatic seizures and depressed skull fracture\(^{15,16,17,12}\). Brain contusions and subdural hematomas are the strongest risk factors for late seizures and this increased risk persists for up to 20 years\(^2\). Individuals with bilateral or multiple cerebral contusions have increased risk of developing seizures due to large amount of tissue destruction\(^{12}\). Patients with multiple post-traumatic intracranial surgeries have also high rates of late PTE\(^{12}\).

We aimed from this work to revise patients with post-traumatic epilepsy, to define risk factors, assess the clinical, neurophysiological and neuroradiological characteristics. The frequency of mesial temporal epilepsy in contrast to neocortical epilepsy was also assessed in these patients.

### PATIENTS AND METHODS

**Patients:**

They were selected from patients attending the Epilepsy Unit, Neuroscience Department, Saudi German hospital Jeddah (SGHJ). Patients were included if they had moderate or severe head injury that preceded the onset of epilepsy and was of sufficient magnitude to result in prolonged loss of consciousness (30 minutes). Amnesia, hospitalization or neuroradiologic evidence of traumatic brain injury\(^{12}\).

Patients must not have in their past history other risk factors for epilepsy or family history of epilepsy.

**Clinical assessment:**

All patients were subjected for detailed history and neurological examination. Detailed history of the head trauma, with revising previous hospital records of trauma, hospitalization, ICU admission, surgeries done and onset of seizures.

Semiology of seizures was revised with family members, attending hospital staff and prolonged video recordings.

Neurological examination was done and compared with hospital files at time of trauma.

Anti-epileptic medications, types, doses and response of patients were revised. Intracranial surgeries done whether as a management of traumatic brain injury or as an epilepsy surgery, were all revised.

**Neurophysiological study:**

All patients underwent prolonged surface interictal EEG recording using the international 10-20 system of electrode placement (Machine). Patients were subjected for continuous video EEG monitoring for ictal and interictal changes. Detailed descriptions of seizure semiology and focus detection were done.

**Neuroradiological study:**

All patients underwent magnetic resonance imaging (MRI) of the brain. T1-weighted sagittal
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and gradient echo axial images were obtained. T1- and T2-weighted and Fluid attenuated inversion recovery (FLAIR) coronal images were obtained at a 3mm-slice thickness through the region of hippocampus. Mesial temporal sclerosis (MTS) was defined by the finding of hippocampal atrophy, T2-weighted hyperintensity, or bright FLAIR signal of hippocampus.18

**Pathological study:**

Pathological examination was done for en bloc resections of either temporal or frontal lobectomy. Criteria for mesial temporal sclerosis were identified.19 These criteria consisted of neuron loss in CA1 and CA4 and the presence of associated reactive gliosis.

**RESULTS**

Twenty-three patients were included in the study. There were twenty males (87%) and three females (13%) (Graph 1). The mean age was 30.5 years, the youngest was 9 years old and the eldest was 46 years old. The mean age of trauma was 12.7 years. The youngest age of trauma was 3 months while the eldest age of trauma was 30 years (Table 1).

Patients with their trauma below or equal to 10 years were 13 patients (56.5%), while patients with their trauma above age of 10 years were 10 patients (43.5%) (Table 1).

The exact time of starting seizures could not be known of some of the patients as patients had management of head trauma at different hospitals with lacking of some information. But we could differentiate between 2 groups of patients. Patients had onset of seizures within the first week (early seizures, 16 patients, 69.6%) and patients with onset of seizures after the first week (late seizures, 7 patients, 30.4%). In the latter group, time of onset of seizures ranged between 6 months (patient no. 3) and 13 years (patient no. 23) (Graph 2).

Patients had five types of head trauma. Road traffic accident (RTA) in 14 patients (60.9%), falling from height in 3 patients (13%), falling to ground in 3 patients (13%), direct blunted head trauma in 2 patients (8.7%) and firearm injury in one patient (4.4%) (Graph 3).

The seizure semiology, EEG findings and MRI results of all patients were summarized in Table 2.

Mesial temporal epilepsy (MTE) was diagnosed according to: (1) seizure semiology (2) Ictal EEG change showing onset in temporal lobes (3) Radiological evidence of atrophy of hippocampus and T2 signal shortening on high resolution MRI or both.20,21 These criteria of MTE is widely accepted and is correlated pathologically.20

We found 14 patients (60.9%) with neocortical Epilepsy (NCE), 8 patients (34.8%) of them had their trauma below or equal to 10 years and 6 patients (26.1%) had their trauma above 10 years old. We found also 8 patients (34.8%) with mesial temporal epilepsy (MTE), 5 patients (21.8%) had their trauma below or equal to 10 years and 3 patients (13%) had their trauma above 10 years. There was one patient (4.3%) with mixed neocortical and mesial temporal epilepsy and he had his trauma above 10 years of age (patient number 9), (Table 3 and Graph 4).

Six patients had temporal lobectomy and one patient had frontal lobectomy and another patient had aspiration of frontal cyst. Surgical outcome according to Engel’s classification22 showed successful results as 3 patients had class 1 (Sizure free, or few residual auras after withdrawal of antiepileptic drugs) and 5 patients had class 2 (Rare seizure, fewer than three complex partial seizures per year) (Table 4).

Pathological results confirmed mesial temporal sclerosis in five of six patients with temporal lobectomy (neuron loss in CA1 and CA4 and the presence of associated reactive gliosis).
<table>
<thead>
<tr>
<th>Patient No./Sex/ Age (yrs)</th>
<th>Age of trauma (Years)</th>
<th>Type of trauma</th>
<th>Type of brain injury</th>
<th>Age of seizures (Years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/M/20</td>
<td>2</td>
<td>RTA</td>
<td>Rt. temporal contusion</td>
<td>2</td>
</tr>
<tr>
<td>2/M/34</td>
<td>14</td>
<td>RTA</td>
<td>Lt. frontal &amp; temporal contusion</td>
<td>14</td>
</tr>
<tr>
<td>3/M/25</td>
<td>10</td>
<td>RTA</td>
<td>Lt. frontal &amp; temporal contusion</td>
<td>10</td>
</tr>
<tr>
<td>4/F/19</td>
<td>3</td>
<td>Falling to ground</td>
<td>Multiple cerebral contusions</td>
<td>13</td>
</tr>
<tr>
<td>5/M/36</td>
<td>7</td>
<td>Falling from height</td>
<td>Multiple cerebral contusions</td>
<td>7</td>
</tr>
<tr>
<td>6/M/23</td>
<td>5</td>
<td>RTA</td>
<td>Bilateral frontal &amp; temporal contusions</td>
<td>5</td>
</tr>
<tr>
<td>7/M/35</td>
<td>3 months</td>
<td>Falling to ground</td>
<td>Bilateral frontal contusions</td>
<td>6</td>
</tr>
<tr>
<td>8/M/39</td>
<td>6</td>
<td>RTA</td>
<td>Lt. frontal contusion</td>
<td>6</td>
</tr>
<tr>
<td>9/M/34</td>
<td>23</td>
<td>RTA</td>
<td>Multiple cerebral contusions</td>
<td>23</td>
</tr>
<tr>
<td>10/M/24</td>
<td>19</td>
<td>RTA</td>
<td>Multiple cerebral contusions</td>
<td>19</td>
</tr>
<tr>
<td>11/F/20</td>
<td>8</td>
<td>RTA</td>
<td>Multiple cerebral contusions</td>
<td>8</td>
</tr>
<tr>
<td>12/M/20</td>
<td>6</td>
<td>RTA</td>
<td>Rt. extradural hematoma, evacuated</td>
<td>6</td>
</tr>
<tr>
<td>13/F/9</td>
<td>2.5</td>
<td>Falling from height</td>
<td>Multiple cerebral contusions</td>
<td>2.5</td>
</tr>
<tr>
<td>14/M/46</td>
<td>21</td>
<td>Direct blunted head trauma</td>
<td>Brain concussion, brain edema</td>
<td>21</td>
</tr>
<tr>
<td>15/M/39</td>
<td>19</td>
<td>RTA</td>
<td>Rt. Intracerebral hemorrhage, operated</td>
<td>19</td>
</tr>
<tr>
<td>16/M/35</td>
<td>22</td>
<td>Direct head trauma</td>
<td>Rt. Intracerebral hemorrhage</td>
<td>22</td>
</tr>
<tr>
<td>17/M/46</td>
<td>6</td>
<td>Falling from height</td>
<td>Rt. Parietal contusion</td>
<td>6</td>
</tr>
<tr>
<td>18/M/37</td>
<td>17</td>
<td>RTA</td>
<td>Fracture skull and spine</td>
<td>18</td>
</tr>
<tr>
<td>19/M/37</td>
<td>30</td>
<td>Falling to ground</td>
<td>Fracture skull, biparietal contusions</td>
<td>30</td>
</tr>
<tr>
<td>20/M/31</td>
<td>8</td>
<td>RTA</td>
<td>Bifrontal contusions</td>
<td>8.5</td>
</tr>
<tr>
<td>21/M/13</td>
<td>7.5</td>
<td>RTA</td>
<td>Bifrontal contusions</td>
<td>8.5</td>
</tr>
<tr>
<td>22/M/35</td>
<td>25</td>
<td>Fire arm injury</td>
<td>Rt. Occipital injury</td>
<td>28</td>
</tr>
<tr>
<td>23/M/45</td>
<td>30</td>
<td>RTA</td>
<td>Multiple cerebral contusions</td>
<td>44</td>
</tr>
</tbody>
</table>

(M: Male, F: Female, RTA: Road traffic accident, Rt.: Right, Lt: Left)

Graph (1): Number of male and female patients.

π: females, 3, 13%
♂: Males, 20, 87%
Graph (2): Patients with early and late seizures.

(RTA: Road traffic accident, FFH: Falling from height, FTG: Falling to ground, DT: Direct head trauma, FAI: Fire arm injury).

Graph (3): Type of head trauma.

Graph (4): Number of patients with MTE and NCE below and above 10 years of age.
Table 2: Clinical data, EEG findings and MRI results.

<table>
<thead>
<tr>
<th>Pat. No.</th>
<th>Seizure Semiology</th>
<th>EEG Findings</th>
<th>MRI Results</th>
<th>Conclusion (Type of epilepsy)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Epigastric aura, Lt. UL automatis, Rt. dystonia</td>
<td>Rt. Temporal slowing</td>
<td>Rt. Inferior temporal sclerosis (Cortical sclerosis)</td>
<td>Rt. Temporal neocortical</td>
</tr>
<tr>
<td>3</td>
<td>Epigastric aura, Lt. motor &amp; Lt. adverse, GTC</td>
<td>Rt. Fronto-temporal epileptic activity</td>
<td>Rt. Fronto-temporal encephalomalacia</td>
<td>Rt. Fronto-temporal neocortical</td>
</tr>
<tr>
<td>4</td>
<td>Vertigo, Lt. adverse, GTC</td>
<td>Bilateral temporal epileptic activity, more Rt.</td>
<td>Rt. Hippocampal atrophy &amp; sclerosis*</td>
<td>Rt. MTE</td>
</tr>
<tr>
<td>5</td>
<td>Expressive dysphasia, Rt. Motor &amp; adverse, GTC</td>
<td>Left anterior temporal epileptic activity</td>
<td>Lt. Mesial temporal sclerosis*</td>
<td>Lt. MTE</td>
</tr>
<tr>
<td>7</td>
<td>Fear, oral automatis, GTC</td>
<td>Lt. ant temporal &amp; frontal epileptic activity</td>
<td>Lt. frontal cyst</td>
<td>Lt. Fronto-temporal neocortical</td>
</tr>
<tr>
<td>8</td>
<td>Lt. motor</td>
<td>Lt. frontal slowing</td>
<td>Lt. frontal cyst</td>
<td>Lt. frontal neocortical</td>
</tr>
<tr>
<td>9</td>
<td>Epigastric, Rt. UL dystonia, GTC</td>
<td>Lt. temporal epileptic activity</td>
<td>Rt. Frontal encephalomalacia, Lt. mesial temporal sclerosis*</td>
<td>Rt. Frontal neocortical &amp; Lt MTE</td>
</tr>
<tr>
<td>10</td>
<td>GTC</td>
<td>Rt. Frontal epileptic activity</td>
<td>Lt. Frontal encephalomalacia</td>
<td>Rt. Frontal neocortical</td>
</tr>
<tr>
<td>11</td>
<td>Vertigo, oral automatis, Rt. Adverse, GTC</td>
<td>Normal</td>
<td>Rt. Hippocampal sclerosis* &amp; cystic lesion</td>
<td>Rt. MTE</td>
</tr>
<tr>
<td>13</td>
<td>Attacks of lost consciousness</td>
<td>Rt. Temporal epileptic activity</td>
<td>Lt. Mesial temporal sclerosis*</td>
<td>Rt. MTE</td>
</tr>
<tr>
<td>14</td>
<td>GTC</td>
<td>Generalized dysrhythmia</td>
<td>Lt. Mesial temporal sclerosis*</td>
<td>Rt. MTE</td>
</tr>
<tr>
<td>16</td>
<td>Left motor, Rt. dystonia</td>
<td>Rt. Fronto-temporal epileptic activity</td>
<td>Rt. Frontal hematoma (resolved)</td>
<td>Rt. Frontal neocortical</td>
</tr>
<tr>
<td>18</td>
<td>Chest compression, GTC</td>
<td>Rt. Fronto-temporal epileptic activity</td>
<td>Rt. Frontal gliosis</td>
<td>Rt. Frontal neocortical</td>
</tr>
<tr>
<td>20</td>
<td>Rt. Motor, GTC</td>
<td>Lt. frontal epileptic activity</td>
<td>Lt. frontal encephalomalacia</td>
<td>Lt. Frontal neocortical</td>
</tr>
<tr>
<td>21</td>
<td>Lt. motor, GTC</td>
<td>Rt. Frontal epileptic activity</td>
<td>Bifrontal encephalomalacia</td>
<td>Bifrontal neocortical</td>
</tr>
<tr>
<td>22</td>
<td>Vertigo, flashes of light, GTC</td>
<td>Rt. Occipital epileptic activity</td>
<td>Rt. Occipito-parietal encephalomalacia</td>
<td>Rt. Occipito-parietal neocortical</td>
</tr>
<tr>
<td>23</td>
<td>Rt. adverse, post-ictal confusion, GTC</td>
<td>Lt. temporal epileptic activity</td>
<td>Lt. Temporal sclerosis*</td>
<td>Lt. MTE</td>
</tr>
</tbody>
</table>

(GTC: Generalized tonic-clonic convulsions, Rt: right, Lt: Left, MTE: Mesial temporal epilepsy)
Table 3. Number of patients with Mesial temporal epilepsy (MTE) & Neocortical epilepsy (NCE).

<table>
<thead>
<tr>
<th></th>
<th>NCE</th>
<th></th>
<th>MTE</th>
<th></th>
<th>Mixed NCE &amp; MTE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>Below, equal 10 ys</td>
<td>8</td>
<td>34.8%</td>
<td>5</td>
<td>21.8%</td>
<td>---</td>
</tr>
<tr>
<td>Above 10 ys</td>
<td>6</td>
<td>26.1%</td>
<td>3</td>
<td>13%</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>14</td>
<td>60.9%</td>
<td>8</td>
<td>34.8%</td>
<td>1</td>
</tr>
</tbody>
</table>

Table 4. Drug treatment and surgical intervention of all patients.

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Drug treatment</th>
<th>Surgical Intervention</th>
<th>Outcome after surgery (Engel's class.)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Surg. TBI</td>
<td>Epilepsy Surg.</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>CBZ, PHT, LTG</td>
<td>Rt. Temporal lobectomy</td>
<td>Class 2</td>
</tr>
<tr>
<td>2</td>
<td>PHT, CBZ</td>
<td>Lt. Temporal lobectomy</td>
<td>Class 1</td>
</tr>
<tr>
<td>3</td>
<td>CBZ, VPA, LTG</td>
<td>Rt. Ant. Temporal lobectomy</td>
<td>Class 2</td>
</tr>
<tr>
<td>4</td>
<td>LTG, CBZ</td>
<td>Rt. Temporal lobectomy</td>
<td>Class 1</td>
</tr>
<tr>
<td>5</td>
<td>VGT, CBZ, LTG, VPA</td>
<td>Lt. Temporal lobectomy</td>
<td>Class 1</td>
</tr>
<tr>
<td>6</td>
<td>CBZ, VPA, TPA</td>
<td>Lt. Temporal lobectomy</td>
<td>Class 2</td>
</tr>
<tr>
<td>7</td>
<td>PHT, VPA, LTG</td>
<td>Drainage of cyst</td>
<td>Class 2</td>
</tr>
<tr>
<td>8</td>
<td>VPA, CBZ, LTG</td>
<td>Rt. Frontal lobectomy</td>
<td>Class 2</td>
</tr>
<tr>
<td>9</td>
<td>PHT, TPA, VPA, CBZ</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>10</td>
<td>CBZ, TPA</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>11</td>
<td>CBZ, VPA</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>12</td>
<td>LTG, CBZ, CZM</td>
<td>Evacuated Extradural H.</td>
<td>-</td>
</tr>
<tr>
<td>13</td>
<td>PHT</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>14</td>
<td>PHT</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>15</td>
<td>CBZ, VPA</td>
<td>Operated cerebral H.</td>
<td>-</td>
</tr>
<tr>
<td>16</td>
<td>VPA, TPA</td>
<td>Operated cerebral H.</td>
<td>-</td>
</tr>
<tr>
<td>17</td>
<td>CBZ, LMG</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>18</td>
<td>CBZ, TPA</td>
<td>Fixation of fracture spine</td>
<td>-</td>
</tr>
<tr>
<td>19</td>
<td>CBZ, PHT</td>
<td>-</td>
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</tr>
<tr>
<td>20</td>
<td>LTG</td>
<td>-</td>
<td>-</td>
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<tr>
<td>21</td>
<td>CBZ</td>
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<tr>
<td>22</td>
<td>CBZ</td>
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<td>-</td>
</tr>
<tr>
<td>23</td>
<td>PHT</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Fig. (1): Patient with Rt. Temporal sclerosis and lobectomy.

Fig. (2): Patient with Lt temporal sclerosis and lobectomy.

Fig. (3): DEEG showed left temporal focus.
Fig. (4): Patient with Right frontal encephalomalacia

Fig. (5): Patient with right frontal and left temporal encephalomalacia.

Fig. (6): Patient with extensive right parietal encephalomalacia.
DISCUSSION

In this study, we reviewed twenty-three patients with post-traumatic epilepsy who had their head trauma at any age.

We found large percentage of male patients (87%) as compared to female patients (13%) (Graph 1). This could be explained by the large number of males who are more prone to RTA than females.

The most common type of head trauma in our study was RTA (60.9%), followed by falling from height and falling to ground, each 13% then direct head trauma 8.7% and fire arm injury, 4.4% (Graph 3). This showed the magnitude of road traffic accidents in civilians as a major risk for post-traumatic epilepsy.

Early seizure were high in our study as we found 16 patients (70%) with their seizures in the first week, while only 7 patients (30%) had their seizures late, even up to 13 years of trauma (Pt. No. 23) (Graph 3). In some studies the incidence of immediate seizure was 1-4%, early seizures 4-25% and late seizures 9-42% in civilian head injuries. Other studies showed higher percentage as our study as they found approximately 80% of individuals with TBI had their first seizure within the first 12 months post injury and more than 90% by the end of the second year. Some patients were followed up to 15 years and found increased risk especially in penetrating head injuries to reach 50%.

Several studies searched for the percentage of mesial temporal lobe epilepsy and sclerosis in patients with post-traumatic epilepsy. Many studies found higher percentage in patients with head trauma at young age (Below 5 years) and others found that MTE could occur also in patients with head trauma occurred at older ages.

In our study, we found 14 patients (60.9%) with neocortical Epilepsy (NCE), 8 patients (34.8%) of them had their trauma below or equal to 10 years and 6 patients (26.1%) had their trauma above 10 years old. We found also 8 patients (34.8%) with mesial temporal epilepsy (MTE), 5 patients (21.8%) had their trauma below or equal to 10 years and 3 patients (13%) had their trauma above 10 years. There was one patient (4.3%) with mixed neocortical and mesial temporal epilepsy and he had his trauma above 10 years of age (patient number 9). So, by addition of this patient, we had 9 patients with mesial temporal epilepsy and sclerosis (39.1%) (Table 3, Graph 4).

Of these patients, 6 had temporal lobectomy with successful post-operative results and the diagnosis of mesial temporal sclerosis was definite in 5 patients (Table 4).

Several groups have studied patients who underwent anterior temporal lobectomy (ATL) as a therapy for refractory epilepsy. Mathern et al., studied 259 patients who underwent ATL from 1961 to 1992. They found that 26 (10%) of these patients had TBI as a major risk factor and 50%
of these patients had hippocampal sclerosis. They emphasized also that the mean±SD of these patients was 6.3±1.6 years.

Marks et al.\textsuperscript{24} described 25 patients with PTE who were examined in Yale University from 1982 to 1992, 21 of whom treated surgically. They found seventeen patients with mesial temporal lobe epilepsy (MTE) and eight with neocortical epilepsy (NCE). Fourteen of the patients with MTE were treated surgically with ATL. Of these, 6 (35%) had hippocampal sclerosis confirmed pathologically and they had excellent postoperative outcomes. Again, these researchers emphasized that all patients with hippocampal sclerosis had their head trauma younger than 5 years (mean age 3.4 years). Patients with NCE were significantly older at time of head trauma (mean age 18.25 years).

Another surgical series describe 102 patients who underwent ATL at the university of Michigan from 1990 to 1996. Twenty-nine (28.4%) had head trauma as a cause, of which, 20 (69%) had hippocampal sclerosis identified pathologically. But this study didn’t find correlation between MTS and age of head trauma\textsuperscript{25}.

These earlier reports focused on highly selected patients who were prepared for resective epilepsy surgery.

Our results were compatible with these previous results and one recent retrospective case series, which studied presence of temporal lobe sclerosis in adult patients with intractable epilepsy following TBI. They found that 35% had foci in the mesial temporal lobe while 48% had neocortical foci\textsuperscript{10}. So, this supports the findings that TBI can lead to hippocampal sclerosis in adults as well as in children.

The pathogenesis of temporal lobe sclerosis was studied by many workers. In humans, direct injury to hippocampus from TBI is uncommon. Courville\textsuperscript{26}, examined the brains of 108 patients who had fatal TBI and found contusions in the hippocampus in only 11 (10.2%). Other studies have found neuronal loss primarily in CA1 subfield of the hippocampus, which was frequently bilateral. They presumed that hippocampal sclerosis and MTS resulted from diffuse secondary effects of TBI\textsuperscript{27,28}.

Because of the retrospective nature of these studies and our study, we can’t exclude the possibility that these patients had pre-existing, clinically silent hippocampal sclerosis, and that epilepsy was expressed only after injury. This explanation is unlikely as hippocampal sclerosis is rare in non-elderly patients who do not have temporal lobe epilepsy\textsuperscript{26,29}. It is possible however, that patients who develop head injury have genetic predisposition to hippocampal injury. Epidemiologic support for this possibility is the studies showed increased family history for epilepsy in patients with PTE\textsuperscript{30,31}.

This was studied in animal models for post-traumatic epilepsy. Several animal studies documented clear anatomical changes in the hippocampus and other brain structures together with increased excitability of specific networks\textsuperscript{31-37}.

More recent study demonstrated an increase in the excitability of CA1 pyramidal cells in response to stimulation, 3 months after fluid percussion injury in animal models\textsuperscript{38}.

Other animal models of direct cortical injury showed epileptiform potentials arising from area V of cortex\textsuperscript{39,40}.

More recent work on animal models showed that following injury, spontaneous partial seizure originate from the neocortex at site of injury. Then seizures became chronic and progressive in course (electrographically and behaviourally). By follow-up, they found progression of the phenotype from neocortical (at site of injury) to a predominance of mesial temporal seizures at later time points\textsuperscript{41}.

So, the Importance of our work is that MTS could occur in patients with head trauma in young or old ages. Detection of MTS, clinically, neurophysiologically and radiologically is mandatory for all patients with PTE as resective surgeries of these patients gave a good outcome for the control of their intractable epilepsy.

So, we recommend detailed study of patients with post-traumatic epilepsy, for proper seizure localization and for detection of mesial temporal sclerosis as the source of epileptic activity. Resective surgeries of temporal lobe sclerosis gave successful results for those patients with intractable epilepsy.
REFERENCES

يعتبر مرض الصرع الناجح عن الأصابات المخية بأنه زمرة صرعتية متكررة نتيجة للأصابات الشديدة بالمخ. ويختلف العلماء في تحديد أسباب حدوث الصرع في هؤلاء المرضى ولكن وجد أن تكاليس القص الأذيني من أهم العوامل التي تزيد من احتمال حدوث الصرع.

وقد كان الهدف من هذا البحث هو الدراسة المستقبليّة لؤلؤة المرضي من الناحية الأكليتية والفيزيولوجية وكذلك دراسة المخ باستخدام الرنين المغناطيسي. وتتم دراسة 3 أشياء. أولها مراقبة مريض يعانون من مرضي الصرع نتيجة للأصابات المخية ووجود أنها 4 أمراض (20.0%) يعانون من الصرع نتيجة لاصابة القشرة المخية و18 مرضي (40.8%) يعانون الصرع نتيجة لاصابة القص الأذيني وأن مريض واحد (3.3%) يعاني من نوعي الصرع معا.

ولقد تم استئصال القص الأذيني نتيجة من هؤلاء المرضى وثبت تحسن حالاتهم. وثبت بالدراسة البيولوجية دلت تكاليس القص الأذيني في خمسة مرضى منهم.

ولذا فإن تشخيص الإصابة بنكتنف القص الأذيني من أهم الأسباب اللازمة لعلاج هؤلاء المرضى حيث أن العلاج الجراحى يساعد على تحسن حالاتهم بدرجة كبيرة.