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Post. Traumatic Epilepsy in Al-Nasiriya City

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بِسْمِ اللهِ الرَّحْمنِ الرَّحِيْمِ

قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ

CONTENT

We would like to express our great thanks to Dr Haider Mukhlif Prof. in Neurosurgery for his sound supervision, patience and guidance throughout the execution of this study , to his we are so in debt

Dedicated to :

To God Almighty our Creator, our source of inspiration, wisdom, knowledge and understanding.

For those sharing the difficult time and offering continuous support, our family.

We dedicate this work with our love.

Abstract

Objective:

This study to alent the neurosurgeon and clinician of the expected occurrence of the epilepsy past the injury and how to follow the patient

Methods:

cases of post traumatic epilepsy were reported during sixteen years 2005_2021 500 this study is retrospective study from patients who were admitted to the neurosurgical department of the AL_hussain teaching hospital we depended on clinical assessment , brain CT scan , EEG , all patients were treated by anticonvulsant agent

Results :

we found post traumatic epilepsy tend to occur in those patient who were suffering from brain injury in epileptic region which including frontal, pareital and temporal cortex . Those patients had gliosis tissues in their brain cortex that means brain scar. .Tegretol is best anticonvulsant agent to control the post brain traumatic epilepsy

Conclusion :

■Focal epilepsy is occasionally occur post brain trauma

■main factor which disturbance the cerebral cortex discharge is cortex gliosis which is revealed in brain CT scan

■Diagnosis should be done by clinical assessment, EEG , brain CT sacn and brain MRI

■There is certain area of cerebral cortex which is including frontal , pareital and temporal cortex what is called epileptic region.

Key words:

post brain traumatic epilepsy, Brain CT sacn , EEG , Epileptic region , Brain gliosis (scar) Tegretol.

Introduction

An epileptic seizure is a transient occurrence of sign and symptoms due to abnormal excessive or synchronous neuronal activity in the brain(1) Epilepsy is a disorder on brain characterized by unenduring predisposition to generate epileptic seizure and by the neurological ,cognitive psychological and social consequences of this condition (1).the widely accepted operational definition Epilepsy requires that an individual have at least two unprovoked seizure on separate days ,generally 24hours apart . An unprovoked seizure refer to A seizure that occurs in the absence of an acute brain insult or systemic disorder (2)

Two unprovoked seizure rarely occur in isolation and are associated with high risk of individual experiencing more seizure, in some cases ,such as when the seizure occurs in the sitting of potentially epileptogenic brain insult. Such as an episode of encephalitis or a traumatic brain injury, it is possible to recognize the specific from of Epilepsy at its ear list presentation. In such cases the diagnosis of Epilepsy can be made after the very first unprovoked seizure (2)

Posttraumatic Epilepsy refers Epilepsy that developed after (TBI).most investigators agree that (PTE) is to be distinguished from repeated seizure in early stage following TBI ,while the brain is acutely traumatized in flamed ,and re tab locally disrupted. There fore common set of definitions adopted by many researcher is following(1) immediate seizure, usually defined as those occurring with in 24h after injury(2) early seizure which occur less than 1week after injury (3). Late seizure which occur more than a week after injury .

Since the risk of recurrence after a single late POST traumatic seizure is over 70%, most investigators consider a single late post _traumatic seizure as being sufficient for diagnosis of PTE(3)

The occurrence of seizures after head injury is a recognized complication of TBI and has been demonstrated to worsen functional outcome significantly [5]. Burns and Hauser performed an epidemiologic review of TBI and determined that the incidence of TBI is between 180 and 250 per 100,000 per year [6]. Other studies, using data from the CDC, report a higher incidence of TBI based on emergency room visits, hospitalizations and deaths [7]. The percentage of TBI patients who develop PTE is not known. It is estimated that TBI is an etiological factor in up to 20% of .symptomatic epilepsies in the general population [8]

Patient and Methods:

cases of post traumatic epilepsy (PTE) which were studied during (16) years period 2005_2021 . This study is retrospective and the data were collected during the study including history clinical assessment, brain CT scan , EEG, brain MRI which are investigations for follow up

Pathophysiology

The mechanism by which trauma to brain tissue leads to recurrent seizures is unknown because there are so many different types of head insults and the excitatory cascade is a series of complex processes. Cortical lesions with cortical dysfunction seem important in the genesis of the epileptic activity. Early seizures are likely to have a different pathogenesis than late seizures; early PTS are thought to be .a nonspecific response to the physical insult

The PTE kindling model of epilepsy postulates that iron deposition from extravasated blood leads to damage by free radicals, and the accumulation of glutamate leads to .damage by excitotoxicity

New information suggests that inflammation and immune system alteration may be contributing to the development of seizures and epilepsy. The TBI that leads to PTE in humans is probably the best model for studying epileptogenesis, but even then it is difficult to do so. This offers an opportunity to intervene with therapy to decrease the development of PTE

Injury_ related factors that increase the risk of PTE are as follows:

■Sever trauma

■ Penetrating head Injury

■Intracranial hematoma

■Linear depressed skull fracture

■ Hemorrhagic contusion

■ History of prior TBI as tend to be cumulative

■Focal neuroimaging or electroencephalographic abnormalities in the acute post injury period

Results

Table 1

Type of the brain injury (table1) had shown that in 60% of cases the Penetrating brain Injury was PTE while Those patients who were suffering from non-penetrating brain injury were only 200 case in percentage 40%. So the penetrating brain injury cases more brain tissues more injury and more bleeding and disturbance of the brain metabolic state.

Table 2

Age incidence (table2) 'has. Shown that in30% of the cases the age was above 25 years the aged group of cases 25 years. There were high (PTE) incidence developing (PTE) that is mean the incidence (PTE)is more in the children and young aged.

Table 3

The most common site of the (PTE) was frontal cortex 45% the next lobe was temporal Cortex 30% then the Parietal cortex 20% the last lobe was the occipital cortex 5%.

Table 4

Had shown the onset of (PTE) at time of accident 40%, where as Those patients were suffering with in First to weeks 10%. while The patient got (PTE)after two months 50%.

Table 5

Had shown the role and an efficiency of the anticonvulsant agent, there was the Tegretol 5% 200mg which was the more effective medication To Control the (PTE) in comparison with Depakin 10%, and phenytoin 40%.

Table 6

That had shown the relationship between the Severity of brain injury which depended on the length of amnesia when there is prolonged amnesia that is means more brain tissue damage which is associated with high incidence of (PTE).So when the amnesia more than 24h post traumatic amnesia was 80%. While PTA was less than 24 h, the Percentage was 20%.

Table 7

That had shown the remission rate of PTE was below 5 years period, 70% while those patients got remission beyond 5 years 30% the remission of PTE was depending on the Severity of TBI and. Anticonvulsant medications which is used .

Table 1/ Type of brain injury which is related to PTE

Type of brain injury	**No of patients**	**%**
Penetrating injury	300	**60**
Non Penetrating injury	200	**40**
Total	500	

Table 2/Age incidence

Age incidence	No of patients	%
Above 25 years old	150	30
Below 25 years old	350	70
Total	500	

Table 3/ site of brain injury

Site	No of patients	%
Frontal cortex	225	45
Temporal cortex	150	30
Pareital cortex	100	20
Occipital cortex	25	5
Total	500	

Table 4 / Time of onset

Time of onset	No of patients	%

Immediate	200	40
First two weeks	50	10
After tow months	250	50

Table 5 / Efficiency of anticonvulsant in controlling PTE

Medication	No	%
Tegretol	250	50
Depakin	50	10
Phenytoin	200	40
Total	500	

Table 6/ Amnesia PTA which is related to PTE

	No	%
PTA is less than 24h	400	80
PTA is more than 24h	100	20
Total	500	

Table 7/Remission period of PTE

	No	%
Below 5 years	350	70
Above 5 years	150	30

Discussion

PTE is common complication of the traumatic brain injury in our locality, post traumatic Epilepsy (PTE) is a form of acquired epilepsy that results from brain damage Caused by physical trauma.(1)

A person with (PTE) suffers repeated post traumatic seizure (PTs) seizures that results from (TBI) more than a Week after the initial injury (2)

In our study shown how to predict who will develop epilepsy after TBI (traumatic brain injury) and who will not (3)

however, the likelihood that Person will develop PTE Is influenced by Severity and type of injury, for example Penetrating injuries those that involve bleeding with in .brain carry a high risk (4)

.It is like in our study(Table 1) that shown 80% in Penetrating group of the TBI

The onset of PTE can Occur with in a short time of the physical trauma that Causes it (5)

while in our study which had shown mostly the onset can occur beyond two months Table (4).

people with head trauma may remain at a high risk for past traumatic seizures than general population even a decades after injury (6).as our study that had shown the Same a high risk of PTE(table 4 and table 7)

In our study we had found on brain CT scan extensive brain tissue gliosis what is called brain Scare is the important Cause of. PTE while other study's had shown that PTE may be caused by several biochemical processes that occur in the brain after trauma including over excitation of brain cells and damage to brain tissues by free radicals (8)

In our study antiepileptic drugs do not prevent the development PTE after head injury, but may be used to treat the condition if it dose occur. We saw the use of Tegretal 200mg is high effective to control the PT E then phenytoin (Epanutin) as shown in(table 5), other studies said when medication dose not work to control the seizures surgery may be needed(9).

One study said the use of Phenytoin and Depakin which were widely used to control the (PTE)(10) but because of the complication and its Side-affects. We used the Tegretol Medication as anticonvulsant Medication

Some studies that had been shown or suggested in the period between a brain injury and onset of epilepsy brain-cells may form new synapses and axons undergo apoptosis or necrosis and experience altered gene expression (25) in addition damage to particularly vulnerable area of the Cortex such as the hippocampus may give rise to PTE blood that gathers in the brain after injury may damage brain tissue and there by cases epilepsy (8) the products that Results from break down of hemoglobin from blood may be toxic to brain tissue(8) the iron" hypothesis " hold that PTE is due to damage by Oxygen free radicles the formation of which is catalyzed by iron from blood

To be diagnosed with PTE ,a person must have a history of head trauma and no history of seizures prior to the injury (30) witnessing a Seizures is the most

effective way to diagnose PTE (12)Electroencephalography EEG is a tool used to diagnosis a seizure disorder but a large portion of people with PTE may not have .abnormal

epileptiform" EEG findings indicative of epilepsy (19).in one study about a fifth of " people who had normal EEG Three months after an injury later developed PTE. while EEG is not useful for predicting who will develop PTE it can be useful to localized the epileptic focus to determine Severity and to predict whether person will suffer more ,seizures if they stop taking antiepileptic Medication (15)

Magnetic resonance imaging (MRI) is performed in People with PTE, and, TC scanning can be used to detect brain lesion if MRI is unavailable (15) how are its frequently not possible to defect the epileptic focus using neuroimaging (20)

In our study that has shown the diagnosis of PTE, seizures must not be attributable to anther obvious cause Seizure that occur after head injury are not necessarily due to epilepsy or even to the head trauma' like any one also, TBI survivors may suffer seizures due to factors including imbalances of fluid or electrolytes epilepsy from other Causes, hypoxia (insufficient)Oxygen and ischemia(in sufficient) blood flow to brain withdrawal from alcohol is anther potential cause of seizures. That these factors must be ruled outs cases of seizures in people with head injury before diagnosis of PTE can be made

There was relationship between the remission of PTE and age of the patients who were developing PTE in children and young aged patients had early _remission less than 5 years period. In comparison with middle and old aged patient

In our study said the period of Time between an injury and development of PTE varies and t is not uncommon for an injury is followed by latent period with No recurrent seizures (18), the longer a person goes without developing seizures. the lower the chances are that epilepsy will develop (11)

At least 80 90 % of people with PTE have first seizure with two years of the TBI (15)People with no Seizures within 3 years of the injury have only 5% chance of developing PTE (19)However one study found that head trauma survivors are at risk for PTE as many as 10 years after moderate TBI and over 20 years after severe TBI (12)

The gustily of how long person with PTE remains at high risk for seizures than the general population controversial (12) About the half of PTE Causes go in to remission but cases that occur later, may have a Smaller chance of diagnosis (17).

Conclusion

1. PTE are uncommon in the traumatic brain injury one suspects PTE especially when the patient develops the seizures.

2. Diagnosis should be achieved by clinical assessment EMG brain CT Scan and MRI.

3. Follow up the patients who develop the seizures although they got a remission of epilepsy.

4. Medical treatment is of important to control the PTE but No help up to prevent PTE.

5. There are still many question need to be answered in this field and this stimulate further researchers and studies like Medical treatment to prevent treat PTE.

6. Brain scar (brain tissues gliosis) play major role to induce PTE.

7. PTA (post traumatic amnesia) has significant in onset and prognosis of PTE.

8. We can map the region of the brain is including fronto parito temporal region what is Called epileptic area of the Cortex in which the brain injury will occur leads to (PTE).

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حدوث نوبات الصرع بعد إصابات الرأس

الخلاصة

تقرير عن 500 حالة صرع تعقب إصابات الرأس المختلفة والمتباينة الشدة والنوعية، سجلت خلال ستة عشر سنة (٢٠٠٥ - ٢٠٢١) هذه الدراسة أصيلة ومحدودة للمرضى الذين أدخلوا شعبة الجراحة العصبية في مستشفى الحسين التعليمي في مدينة الناصرية استندت الدراسة على العلامات السريرية والفحوصات الشماعية وخاصة الأشعة المحورية الطبقية CT Scan brain كل المرضى عولجوا تحفظيا طبيا ما عدا الحالات الجراحة مثل كسر الجمجمة المنخسف وإصابات الرأس المفتوحة تم علاجها جراحيا brain wound حالات أو مضاعفات نوبات الصرع ونتائجها نوقشت وقورنت بالأرقام العالمية. حدوث نوبات الصرع التي أعقبت إصابات الرأس كانت متقاربة نسبيا مع الدراسات والنتائج العالمية وقد ثم تحديد منطقة وقد شملت قشرة الدماغ للفص الأمامي والجداري والصدغي وهي المنطقة الأكثر مسؤولة عن احتمالية حدوث نوبات الصرع إذا ما أصيبت بضرر نسيجي. و بيَّنت دراستنا أهمية أثر التليف النسيجي في قشرة الدماغ وسُميت ب brain scar.