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Partial seizure disorder co morbidity factors, causes and associated brain structural lesion, hospital based study.

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Abstract

Backgrounds: Partial epilepsy is the most common seizure disorders encountered in patients with epilepsy. Partial seizure was defined as sudden excessive, rapid and localized electrical discharges by gray matter from some part of the brain certain structural & metabolic abnormalities in the brain with predictable and lower the epilepsy those whose seizure threshold are not reached by routine stresses and trauma.

Objectives: To identify the cause of partial seizure and to clarify the association of these causes and age of the patients.

Material and Methods: A prospective cohort study, we take all patient referred for neurological consultation in Al- Kahdemyia Teaching Hospital, Baghdad Teaching Hospital & Al-Yarrnok Teaching Hospital for the period from (November 2013-May 2015).

The patients with partial seizure and partial with secondaly generalized epilepsy were enrolled in this study. This was secured through a detailed history, elaborate examination, generalized and of the nervous system, 106 patients who presented with partial seizure, the age of them ranged between 6-73 years, 52 males and 54 females.

Result: The abnormal neuroimaging occurred in (61%) of patients. Tumors occurred in about (19.7%) of patients most of them below 40 years of age while infractions is about (25.5%) of patients above this age.

(83.7 %) of CPS patients had temporal lobe foci and (16.2%) in frontal lobe, while (49%) of SPS patients had frontal lobe foci, (22%) frontoparietal and (13%) had partial lobe foci.

(75.4%) of patients with SPS and (35.1%) with CPS had brain lesion.

Conclusion: Infraction is common cause of partial seizure in patients above the age of 40 and below this age was a tumor. Partial seizure is associating mostly with brain lesion.

Incidence of brain lesion (structural) was decrease in association with long duration of partial epilepsy.

Keywords: Partial epilepsy, partial seizure, SPS, CPS, brain lesion.

Introduction

In contemporary society, the Frequency and impollance of epilepsy can hardly overstated.

Epiepsy can be defined as an intermittent derangement of then nervous system due to "an excessive and disorderly discharged of cerebral nervous tissue on muscles". This was postulatein1870, of Hughlings Jackson. [The Eminent British Neurologyist], and modem electro physiology offers no evidence to the contrary.

The word epilepsy was derived from Greek words meanings" To seizure upon" or a" Taking hold of". Our predecessors referred to it as the "Falling sickness" or the falling evil(1).

Partial (focal or localization related) epilepsy the most common seizure disorder encountered in patients with epilepsy. These seizure are focal at onset that is emanating from localized region of the brain. Patient with partial epilepsy may have seizures that are refractory to AED mediation. The financial burden for these patients includes the cost of medical care and often the loss of employment.(2)Seizure is a paroxy small event due to abnormal, excessive, hyper synchronus discharge from an aggregate of central nervous system(CNS) neurons. Approximately 5-10% of population will have atleast one seizure during their lifetime, with highest incidence occurring in early childhood and late adulthood. (3) Epilepsy a group of disorders characterized by recurrent seizure.(4)

Intracellular recordings show bursts of rapid action potential firing, with reduction of the transmembrane potential (Paroxysmal depolarization shift). It is likely that both reduction in inhibitory systems and excessive excitation play a pan in the genesis at seizure activity.

Cells undergoing repetitive epileptic discharges undergo morphological and physiological changes which make them more likely to produce subsequent abnormal discharges (Kindling) (6).

In partial seizure there is paroxysmal depolarization of membrane of a local group of neurons, which correspond stem porally to the finding of a focal spike and wave complex on the EEG. (7-9.)

Focal seizure may spread from one lobe to another or from one hemisphere to another via association and commissural fibers, or it may spread via projection fiber to the thalamus and there by become generalized seizure, the focal symptoms are called an aura and provide evidence of the site of seizure origin (11). Seizure threshold: The point at which a seizure will occur. For every person there is a stimulus or combination of stimuli that may elicit the seizure. People are considered free of epilepsy when there seizure threshold has not reached by routine stresses and trauma. (12) Certain structural and metabolic abnormalities in the brain will predictably lower the epilepsy threshold. (13) The data from several studies suggest that there is a small increase in the incidence of epilepsy in family members patients with partial on set seizure and these are not inherited in a Mandelian fashion. It appears that increase inpaliialon set seizure be best explained by inheritance of a greater susceptibility to epilepsy. The mechanism of it is not yet known. May be related to alteration in the extent of glial cell proliferation, which respond to cortical injury. (14)

Pathologic Possibilities:

Patients presenting with PSs who are under 20 years of age are not likely to have an anatomically progressive focal lesion (15).Seizures before the age of 20 years are rarely associated with tumours (16). If the patient is between 20-30 y of age the risk of a Progressive lesions goes up slightly (17). From 30 to 60 years, the incidence of primary brain tumor peaks (18)., reaching approximately 15 percent in patients with PSs. After the age of 60, the incidence of tumours starts to fall off again as a vascular cause becomes more likely (19-22).

P.E may represent a clinical sign due to ischemic cerebrovascular disease (23). Incidence of mild brain injuries was 4 times high in patients with PE (23,-25). Approximately 30% of patients with PE have mass lesions like neoplasms or vascularmal formations as the cause of their seizure disorders (26). Aura (the functional onset of seizure activity before an alteration in consciousness) occur in more than 50% of patients with CPS, which may be useful in localizing the onset of seizure (27).Unilateral automatism, head version, tonic phenomena, dystonic posturing, unilateral grimacing, post-ictal paresis and unilateral clonic movements and forced vision manifested by maximal deviation of the head, neck and trunk are a reliable indicators of the lateralization of the onset of seizure in patient with PE (28-31)

The incidence of structural abnormalities was higher with increasing age of the onset of seizure and declined with long duration of hi story of epilepsy (32). About epileptogenic foci, related to the brain lobes, reveal that 22.5%. Frontal, 42.5% central sensori -motor, 27% temporal, 5.6% fronto-temporal and 6.3% parietal and other posterior collex (33).The evaluation of the patients with a probable PS includes a variety of diagnostic and clinical evaluation in addition to the history and physical neurological examination. These include EEG and neuro- imaging pictures.

The most common ly used as a diagnostic test in assessment of patients with PE(34,35). A yield between 60 and 93percent has been claimed forth is procedure when multiple recordings are used or when recordings are made after24 hours period of sleep deprivation (41, 42). Sphenoidal electrodes both the anterior and

Posterior ones improve the yield in patients with mesial temporal sclerosis who seroutine EEG are normal (36, 37). Because of the limitation so finterracial EEG recording, prolonged monitoring can be performed and correlated with the video - recorded seizure (38, 39), a normal interictal wake and sleep EEG does not exclude a PS disorders, and may also be false positive.(2)

Patients and Methods

We take all patient referred for neurological consultation in Al- Kahdemyia Teaching Hospital Baghdad Teaching Hospital & Al- Yarrnok Teaching Hospital for the period from (November 20013-May 2015).The patients with partial seizure and partial with secondaly generalized epilepsy were enrolled in this study; this was secured through a detailed history, elaborate examination, generalized and of the nervous system. All patients were evaluated by an interview questioner from patient them self or their relatives. Those patient were examined, investigated and treated by a senior (neurologist) in the above mentioned centers. The patients were examined clinically to look for any neurological deficits and checking for other systems of the body and define any abnormality specially patients with cerebrovascular disease and tumor. All patients had 16 channel EEG recordings (by use of Nihon Kohden Corporation : 432 1 F), some times more than one recording is needed with emphasis on activating procedure like hyperventilation, Each recording is for 20 minutes . Some of the EEGs were done in a private clinics; quality control over the recordings was exercised heavily. All patients had neuroimagings like brain spiral CT (Somatotom Plus 4- Siemens, Version C 1 OB) with and without contrast when needed. MRI (Gyroscan NT 1.5 tesla power. Philips Medical System) was done in patients with no abnormalities revealed by CT and also for the patients with suspicion of tumours or alleriovenous malformation. A11 these reported nueroimagings were done and bv radiologists of the centers mentioned above .Other investigations, like blood sugar, blood urea, electrolytes, and complete blood picture were done.

Results

One hundred six patients were collected from those who visited neurological department of Al-Yarmok teaching hospital, Al-Kadhemia teaching hospital /Baghdad Teaching Hospital with symptoms and sign goes with pm1ial epilepsy. The age of the patients ranged between 6-73y. 52 males & 54 females as shown in table below.

| Age | Number of the patients | Male | Female |
|-------|------------------------|------|--------|
| 6-15 | 23 | 10 | 13 |
| 16-25 | 22 I | 12 | 10 |
| 26-35 | 20 | 9 | 1 I |
| 36-45 | 8 | 3 | 5 |
| 46-55 | 10 | 6 | 4 |
| 56-65 | 15 | 7 | 8 |
| 66-75 | 8 | 5 | 3 |
| Total | 106 | 52 | 54 |

Table 1: Demographic data (age, sex) of the patients



Figure (1) : Demographic data (Sex) of the patients

5patients had abnormal neuro-imaging study & these abnormalities consisted with brain abscess all of them were proved by surgical exploration & histopathological examination. 3 of them at age of 26, 30, 35 age while1patient at 14y of age while other at 55y as showed by table (2) and figure (2).

| Age | No. of all patient | "brain abscess | Hemorrhaage | infractions. | Temporal Sclerosis | Encephalitis | Tumors | No. Of pt. with Lesion |
|--------|-----------------------|----------------|-------------|--------------|-----------------------|--------------|--------|------------------------------|
| 6-15 | 23 | 11 | | 11 | 11 | | | 33 |
| 16-25 | 222 | | | 22 | 33 | | 22 | 7 |
| 26-35 | 220 | 33 | | 55 | 22 | 11 | 66 | 117 |
| 36-45 | 88 | | 11 | 22 | | | 55 | 88 |
| 46-55 | 110 | | | 44 | | 11 | 22 | 77 |
| 56-65 | 115 | 11 | 11 | 99 | | | 44 | 115 |
| 66-75 | 88 | | 22 | 44 | | | 22 | 88 |
| TTotal | 1106 | 55 | 44 | 227 | 6 | 22 | 221 | 665 |

Table 2Types of lesion in relation to age:



Figure (2) The distribution of patients with lesion according to age

Twenty one patients had abnormalities on CT & MRI revealed neoplasm 3 of them diagnosed as maningioma, 16 patients glioma, 2 patients with cystic tumor mostly astrocytoma. Two of above patients are below age of 25y while the others are above this age. Neuro-imaging study revealed intracerebral hemorrhage in 4 of our patients, one patient at age of 40 other at age of 62) ear while 2 patient at 70y of age. We find that 6 patients presented with complex partial. They gave history of febrile convulsions in early childhood. All of them the MRI revealed mesialtemperal sclerosis. One patient at age below 5y 3 patients between 16-25y of age and 2 patients at age of 33 year and 35 year.

About 27 patients. their neuro-imaging revealed infraction, three of them had cortical in fraction with dotts of hemorrhage due to sinus venous thrombosis

as a complication of meningitis (TB meningitis confirmed by CSF examination) both of them between the age of 16-25y, while one patient had a history of neurobrucellosis at age of 15y. Nineteen patients with infarction at age above age 45 year, while 5 patients between 26-35y.Two patient, one at age of 25y, the other al age of 50 year complained of abnormal behavior with attack of loss of consciousness and fever CSF go with viral meningitis and MRI revealed area of hyper intense lesion in brain, so those patients were diagnosed as meningo-encephalitis. In 65(61%) patients the neuro-imaging study (MRI & CT) was abnormal, while 41(39%) patients no lesion can be defined as in table (3) and figure (3). From those patients without apperant brain lesion on neuroimaging and no focal signs in examination had a history of head injuries before the onset of seizure at variable times.

Table 3: Neuro-imaging study in 106 patients with partial seizure

| Neuroima gi ng study | Patient No. | % | |
|----------------------|-------------|------|--|
| Abnormal | 65 | 61 | |
| Normal | 41 | 39 | |
| Total | 106 | 100% | |



Figure (3): Neuro-imaging study in 106 patients with partial seizure

Sixty five patients with partial epilepsy had lesions that were revealed on MRI & CT 2 1(32%) patients with tumors: 16 patients with glioma, 3 patient, with maningioma, 2 patients with cystic tumor, both of them had astrocytoma. Twenty seven (42%) patients

complained of infraction, 4 (6%). patients had intracerebral hemorrhage. Other lesion included brain abscess is 5 (8%) patients. Encephalities occurred in 2 (3%) patients, while 6 patients (9%) had mesial temporal sclerosis see table (4) and figure (4).

| Lesion | Patient no. | % | % of the lesion from all patients (106) | |
|--------------------|-------------|------|---|--|
| Infraction | 27 | 42 | 25.5 | |
| Tumor | 21 | 32 | 19.8 | |
| Temporal sclerosis | 6 | 9 | 5.7 | |
| Brain abscess | 5 | 8 | 4.7 | |
| Hemorrhage | 4 | 6 | 3.8 | |
| Encephalitis | 2 | 3 | 1.9 | |
| Total | 65 | 100% | (60.9%) | |

Table 4: Frequency of lesions in PE.



Figure (4) Show the frequency of lesions in PE

The patients that presented with simple partial seizure are 16(15.1%) while 19 (17.90/0) patients complained of complex- partial seizure. Secondary Generalization occurred in 71 patients (67%) as shown in table (5) and figure (5). About 56 (52.8%)

patients complained of headache especially in patients with space occupying lesion and in those with infection and intracerebral hemorrhage and in patients with temporal epilepsy at the same side of brain lesion.

| Types of seizure | Number of patient % | | |
|---|--|-----------------|--|
| Simple partial | 16 15.1 19 179 | | |
| Compl ex partial | | | |
| Secondary G.T.C | 71 | 67 | |
| Total | 106 | | |
| 80 70 60 50 40 30 20 10 0 simple partial complete | x partial secondary GTC | ■ % of patients | |

Table 5: Types of partial seizure



In our study we found that there are 19 patients who had a history of febrile convulsions in childhood, 5 (26%) of them had focal area at fronto-parietal area while 14 (74%) patients had focal area of epilepsy at temporal lobe. In those patients focal area of epilepsy at temporal lobe had a changes in MRI go with mesial temporal sclerosis in 6 (32%) patients see table (6) and figure (6).

Table6: febrile convulsion in association with area of partial seizure

| Focal lesion | No. of patient with fibrile convulsion | % |
|-------------------|--|------|
| Fron to-pariet al | 5 | 26.3 |
| Temporal | 14 | 73.6 |
| (Mesialsclerosis) | (6) | (32) |
| Total | 19 | |



Figure (6) reveal febrile convulsion in association with area of partial seizure

About 30 patients had a history of epilepsy in their family, 18(60%) of patients had family history of G.T.C epilepsy while12 patients (40%) had a history

of partial epilepsy in their family. From those patient with family history of epilepsy they had a history of febrile convulsion in 16(53%) patients.

| Type of seizure | | Brain area of foci | No. of patient | % |
|-----------------|-----|--------------------|----------------|------|
| Complex partial | 37 | Temporal | 31 | 8.7 |
| | 57 | Frontal | 6 | 16.2 |
| Simple partial | 69 | Frontal | 34 | 49 |
| | | Parietal | 13 | 19 |
| | | Pronto-parietal | 22 | 132 |
| Total | 106 | | | |

Table 7: Types of seizure and brain foci

The number of patients who have complex partial seizures were 37, in 31 (83.7%) patients the lesions or foci of epilepsy were in temporal lobe while 6(16%) patients had them in frontal lobe, while the number of patients with simple partial epilepsy was 69. In 34

(49%) patients the foci were in frontal lobe and 13 (19%) patients had foci in parietal area while in 22 (32%) patients the foci occur at fronto-parietal area , as shown in table (7) and figure (7 a, b).

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Figure (7a, b) Show types of seizu re and brain foci

Association of simple partial seizure and complex partial seizure with brain lesion : neuro-imaging in patients with SPS revealed lesion in about 52 (75.3%) patient from 69 patients, while there are lesions in 13 (35.1%) patients complained from complex partial epilepsy see table (8) and figure (8).

| Types of seizure | No. of patient | Associated brain lesion | % of brain lesion |
|------------------|----------------|-------------------------|-------------------|
| Simple | 69 | 52 | 75.4 |
| partial | | | |
| Complex | 37 | 13 | 35.1 |
| partial | | | |
| Total | 106 | 65 | |

Table 8 : partial seizure and brain structural lesions :



Figure (8) show the partial seizure and brain structural lesion

Relationship between partial seizures duration and abnormal neuro-imaging. We found that when there is a long duration of epilepsy the abnormalities on neuroimaging or structural brain abnormalities decrease as shown in table (9) and figure (9)

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| Duration of | No. of patients | Abnormal MRI | % |
|---------------|-----------------|--------------------|------|
| epilepsy | | and <i>I</i> or CT | |
| Less than 1yr | 43 | 35 | 81.4 |
| 1-2yr | 24 | 17 | 70.8 |
| 3-5yr | 16 | 7 | 43.8 |
| >5yr. | 23 | 6 | 26.1 |
| Total | 106 | 65 | |

Table 9: Duration of epilepsy and brain lesions



Figure (9) Show the duration of epilepsy and brain lesion

Discussion

In our study there are 65 (61%) patients with partial epilepsy had abnormal neuro-imaging (CT and MRI), while there are no abnormalities in about 41 (39%) patients. These results are slightly more than previous (23-55%) reported studies ^(40,41).

This may be attributed to that our study was hospital based and more stringent inclusion of patients in this study. Cerebral vascular accidents or diseases as a cause of partial epilepsy was found in about 27 (25.5%) patients and mostly at age above 40 yr. which is more than previous studies ^(25, 37, 42) which revealed that the percentage of vascular disease as a cause of partial seizure is 18%. While the incidence of tumour as a cause of partial epilepsy was found in about 21 (19.7%) patients and most of them are under the age at 40 years in comparison with 20% in previous reported studies ^(29, 30, 43) and more than percentage reported in other studies (4-12%) ^(29, 30, 32, 44).

Which can be explained by the nature of our study which 1s a prospective nature with particular attention to find the cause of the partial epilepsy in our patient and we collected the patient that visited our hospitals complained of partial epilepsy only. We found that the patients complained of simple partial seizure were 69 (65%) while 37 (35%) patients complained of complex partial seizure these results comparable previous reported study ⁽³²⁾

56 (52.8%) patients complained of headache most of them associated brain lesions, and temporal lobe epilepsy in whom occur at the same side of focal area. These results are in keeping with other reported study ⁽⁴⁹⁾ in which about 47% of PE had (per ictal). headache 2/3 with temporal lobe usually epsilateral and Y2 with extra temporal lobe epilepsy. There is highly association of structural abnormality in brain&SPS occurred in about 52 (75.3%» as opposed to 13(35.1%) pt. CPS , our figures are comparable (48-71%) of previous reports ^(44, 47, 48).

About febrile convulsion .we found there is increasing m its incidence with temporal epilepsy especially mesial temporal sclerosis (32%) which was less than 48% of previous reports. Which may be attributed to many patient may be misdiagnosed by radiology & there is no relations between a degree of sclerosis & atrophy in MR1 & history of seizure⁽⁵⁶⁾. These changes may occur in individual that never had seizure ⁽⁵⁰⁾. In other study, the identification of mesial sclerosis was incidental by MRI & was but significant needs investigation ⁽⁵¹⁾.

From our result there is (60%) of patients with SPE & (40%) patients with Complex PE had family history of epilepsy this result also was reported by other several studies ⁽¹⁴⁾, that suggest an increase in the incidence of epilepsy in family members of patient with PE. There are 16 (53%) patient had febrile convulsion also had a family history of epilepsy. This result also corresponding to previous reports⁽¹⁵⁾that genetic propensity for seizure may expressed early by occurrence of febrile convulsion. Most foci in patients with complex partial epilepsy occurred at temporal lobe (92%) & (18%) at Frontal lobe while in patient with SPE most foci are in Frontal (34%), then fronto-parietal (22%) and last parietal lobe (13%). These result arc comparable with previous reported studies⁽²⁾ revealed 80% of CPE are of temporal lobe while most extra- temporal epilepsy emenate from the frontal lobe. We noticed that when there is prolonged duration of epilepsy the incidence of structural abnormalities of brain lesions decreased while short duration, neuro-imaging when there is abnormalities will increased these result were corresponding to previous reported study (31, 45, 47)

Conclusion

Most patients with partial seizure are associating with abnormal neuro-imaging study, most causes of partial seizure above age of 40 are cerebral vascular associated while below this age are Tumors.In high percentage or patients with SPE the frontal lobe was responsible for epilepsy while in CPE was temporal lobe.There is association between febrile convulsions & temporal lobe epilepsy especially temporal sclerosis and epilepsy.When there is long duration of epilepsy incidence or brain lesion (structural) was decrease . There is high association between febrile convulsion and family history of epilepsy.

References

- 1-.AdamsD.R., 1ctors1, Rapper H.A:Epilepsy and disorder of consciousness. Principal of neurology 1thed. New York.McGrew.Hill, 2001.pp331-363.
- 2. Cascino GD. Intractable pa11ial epilepsy evaluation and treatment.1yoclin Proc1990 65:15758-1586.
- 3-LowenSteinDH: Seizure and epilepsy. In: Braunwald E, HauserSL, Tameson JL..etal (eds.). Harrison· principle of internal medicine,15thed. New York, MC Graw.Hill, 2001.pp2354-2369.
- 4-Simon RP. Aminoff JM; Greenberg AD. Clinical neurology, 4th ed, tamford Connecticut, Appleton and Lange,1999.
- 5- Schomer DL. Partial epilepsy. N. Eng J med1983 309:536-539
- 6-AllenCMC,LueCKCT:Disease of nervous system. In: Edward sCRE, Bouchier DA: Davidson's principles and practice of medicine,18thed,UK.Churchilllivingstone,1999.
- 7- WilkinSonS. Essential neurology, 3rded. London,Blackwellscience,ppl92.
- Laidlaw T,Richens A. OxleyJ. Textbook of epilepsy, 3rd ed Edinburg Churchilllivingstone .1988.ppl53.
- 9.GoldenSohn ES, PurpuraDP. Intracellular potentials of cortical neurons during focal epileptogenic discharges. Science1963;39:840-842.
- 10-Prince DA, FutamachiKJ. Intracellular recordings in chronicfocalepilepsy.BrainRes1968;II:681-684.
- 11—Daube JR.Reagan TJ.,Sandos BA;et al. Medical neurosciences on approach to anatomy, Pathology, physiology by system evel, 2nd ed, Boston, little brown,1986,pp397.
- 12-Lechtcnberg R. Seizure: recognition and treatment. Edinburg,Churchilllivingstone,1990,pp6.
- 13- Latack JT. Abu-Khalil BW., Siegel GJ.; et al . Patients with partial seizure : evaluation by MRJ , CT and PET imaging. Radiology 1980 ; 159 : 1 59.
- 14- MannagettaGB . Genetic of the epi lepsy .Berlin , Springer-Verlag .1989, pp79.
- 15- Bridge EM. Epilepsy and convulsive disorders in children. New York, MC Graw-Hill , 1949.
- 16- Vignaendra V, Ngkk, Lim CL ; et al . Clinical and electroencephalic data indicative of brain tumors in a seizure population. Post grad Med J 197 ; 54 : 1 -5.
- 17- Rayn or R, Daine R. Carmicheel E. Epilepsy at late onset. urology 1959; 9: 11-7.
- 18- Wood cock Cosgrove JBR. Epilepsy after the age of 50 : a five years follow up-study . Neurology 1 964; 14 : 34-40 .

- 19- Reisman D, Fitz-Hugh TJr. Epilepsiatorda. Anu.Interu Med. 1927;1:273-282.
- 20- Shorven SD, Gilliatt RW., Cox TC ; et al . Evidence of vascular disease from CT scanning in late onset epilepsy. Journal of Neurology, Neurosurgery and Psychiatry 1984; 47: 225-230.
- 21- Marsden CD Sowler TJ. Clinical neurology .2nded, London . Sydney. Aucklond, 1998, pp 249.
- 22- HiYoshi T, Yagi K. Epilepsy in late onset. Epilepsia 2000; I supple: 931-935
- 23-Cocito. Loeb C. Focal epilepsy as a possible sign of transiet subclinical ischemia. EurNeural 1989: 29(6) 339-344.
- 24- Jabbor B Prokhorcnko O, Khajavi K; et al . Intractable epilepsy and mild brain injury: incidence, pathology and surgical operation. Brain inj. 2002; 16 (6): 463-467.
- 25- Babb TL , Brown WJ : Pathological finding in epilepsy. In: J Engel Jr. Surgical treatment of the epilepsies. New York, Raven Press , 1987. pp 55 1-540.
- 26- Shorbrough FW : Complex partial seizure . In: HLuders, RP Lesser . Epilepsy: Electroclinical syndromes. London, Springer-Verlog. 1 987, pp 279-302.
- 27- Lesser PR ,Luders H. Diuner DS ; et al : Simple partial seizure . In HLuders, RP Lesser .Epilepsy :Electroclinical syndromes . London, Springer-Verlog , 1987, pp 223-278.
- 28- Walker EB ,Shorbrough FW. The significance of lateralized ictal paretic accruing during complex partial seizure. Epilepsia 1988: 29 : 665.
- 29- Wyllie Ej., Luders H., Morris HH.; et al : The lateralizing significance of versive and eye movements during epileptic seizures. Neurology 1988; 36 : 606-61 l.
- 30- Bonelli B: Banmgertner C. Frontal lobe epilepsy clinical seizure semiology. Weinklinwachencinr2002 ; 114 (8-9) : 334-340.
- 31- Yaqub B, Danayiotopoulos CP., Al- Nazha M.: et al. Cause of late onset epilepsy in Saudi Arabian: the rate of cerebral granuloma. J Neural, Neurosurgery, Psychiatry 1987; 50: 90-92.
- 31- ManfordM . Hart Ym., Sander JE., et al .National general practice study of epilepsy (NGPSE) : Partial seizure pattern in general population Neurology 1992; 42: 1911-1917.
- 32-Klass DW. Electroencephalographic manifestation of complex partial seizure . Adu Neural 1975; 1 1 : 113-140.
- 33-EngelJJr : Seizure and epilepsy . Philadelphia , F.A Davin company,1989 .pp 303 -339.

- 34- Kooi KA, Tucker PD, Marked RE Fundamentals of electroencephalography . 2nded. New York , Harper and Row , 1978.
- 35- Gibbs EC Gibb FA. Diagnostic and localizing value of electroencephalographic studies in sleep. Res Nervment Dis Proc 1997;26: 366-376.
- 36- Rovit RL , Gloor P, Rasmussen T. Sphenoidal electrodes in the electrografic study of patients with temporal lobe epilepsy :an evaluation . T Neurosurgery1981; 18: 151-158.
- 37- Sutula TP., Sackellores JC., Miller JQ ; et al . Intensive monitoring in refractory epilepsy. Neurology 1981 ; 31 : 243-247.
- 38- Porter RJ. Perny JK, Locy JR. Diagnostic and therapeutic reevaluation of patients with intractable epilepsy. Neurology 1977; 27 : 1006-101 1.
- 39- Bergen D., Bleck T., Ramsey R; et al. Magnatic resonance imaging as a sensitive and specific predicter of neoplasm removal for intractable epilepsy. Eplipsia 1989; 30: 318-321.
- 40- Jack CR..Sharbough FW, Twomay CK.; et al. Temporal lobe seizures: Lateralization with MRI value measurement of hippocampal formation .Radiology 1990 ; 175: 42,, -429.
- 41- Engel JJr; seizure and epilepsy. Philadelphia FA Davis Company 1989: 443-474.
- 42- AndermamF : Identification of candidates for surgical treatment of epilepsy . In : J Engel Jr . Surgical treatment of the epilepsies. New York , Raven Press 1987, pp 51-70.
- 43- Young A C., Borg CJ, Moher PD: et al Is routine computerized axial. tomography in epilepsy worthwhile ? Lancet 1982: 2 : 1446-1447.
- 44- Zimmerman RA., Gonzalez C., Bilaniuk LT.; et al. Computed tomography in focal epilepsy. Comput Tomogr1977; 1:83-91.
- 45- Fuertein J., Weber M., Kurtz D.; et al . Etude statistique des crises epileptic quesapparais santapresd'age de 60 ans. Sem Hop Paris 1970 ; 46 : 3115-3128.
- 46-MC Gahan JP, Dubin AB, Hill RP . The evaluation of seizure disorders by computerized tomography . J Neurosurg I 979; 50 : 328-332.
- 47- Gastaut H , Gastant JL .Computerised axial tomography in epilepsy Epilepsia 1976; 1 7 : 325-336.
- 48- Bcrhascon : A lateralizing value of peri-ictal headache ; study of 100 patients with partial epilepsy Neurology 2000 ; 56 (1) : 130-132.
- 49- Bower SP, Kilpatrick CJ ,Vagrin SJ . Degree of hippocampal atrophy is not related to history of febrile seizure in patients with proved

hippocampel sclerosis . J Neurolneurosurg psychiatry 2000; 69: 733-738.

- 50- Kobeyashi E., Li Lm., Lopes. Cendes I.; et al. Magnetic resources imaging evidence of hippocampal sclerosis in asymptomatic first degree relatives of patient with familial medical temporal lobe epilepsy. Acrch Neural 2002; 59 (12): 1891-1894.
- 51- Moore KR, Swallow CE, Tsuruder JS. Incidental detection of hippocarnpel sclerosis on MRI : it is significant. AJNR-Am-J- Neuro- radial 1999 ; 20 (4) : 1609-1612.



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