Partial Seizures, Etiologies and Associated Comorbidity Factors

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Abstract

**Background:** Partial seizure is well-defined as sudden extreme, profligate, and limited electrical discharges by gray matter from some portions of the brain due to certain structural & metabolic abnormalities.

**Objective:** To distinguish the etiologies of partial seizures and to clarify its association with the age of affected patients.

**Patients and Methods:** A prospective study, done on all patients with neurological consultation in Al- Batool Teaching Hospital, Baqubah Teaching Hospital and Al Yarmouk teaching Hospital from Nov, 2016 to Dec, 2018. Patients with partial seizures and/with secondary generalization were merged. This was fortified through a full history, physical checkup, EEG, and MRI of the brain. The study’s sample comprising 106 patients with partial seizures, the age ranged from 6-75 years, with 52 males and 54 females.

**Results:** Atypical neuroimaging was found in (61%) of patients. Tumors occurred in (19.7%) of patients, the highest of them below 40 years of age while infarctions comprised 25.5% of patients outside this age. Complex partial seizures(CPS) patients with temporal lobe foci comprised 83.7 % and (16.2%) had frontal lobe problems, while (49%) of Simple partial seizures (SPS) patients had frontal lobe foci, 22% frontoparietal and 13% had parietal lobe foci and had brain lesions were spotted in 75.4% of patients with SPS and (35.1%) with complex partial seizures.

**Conclusion:** Infarction is a common reason for partial seizures in patients above 40 years while below this age the tumor is common etiology. A partial seizure is connected mostly with brain lesions.

**Keywords:** Partial seizures, comorbid factors, brain lesions

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**Introduction**

Partial (focal or localization linked) epilepsy the greatest seizure disorder met with patients with epilepsy [1-5]. Roughly 5-10% of people will have at least one seizure
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during their lifespan, with the highest frequency happening in early childhood and late adulthood [3-6]. Cells suffering repetitive epileptic releases suffer morphological and physiological alterations which brand them more likely to yield subsequent atypical discharges [6-9]. In partial seizure, there is paroxysmal depolarization of the membrane of a confined group of neurons, which match temporally to the results of a focal spike and wave complex on the EEG [10-14]. A focal seizure may blowout from one lobe to another or from one hemisphere to another through association and commissural fibers, or it may blowout via projection fibers to the thalamus and turn into generalized [15-19]. The focal symptoms are named an aura and afford an indication of the site of seizure origin [11,20-26]. Seizure threshold: The point at which a seizure will occur [12,27-29]. Certain structural and metabolic anomalies in the brain will unsurprisingly lower the epilepsy threshold [13,30-3]. It seems that rise in seizure is best explained by the inheritance of a greater vulnerability to epilepsy [14,37-42]. Seizures before the age of 20 years are rarely associated with tumors [16]. From 30 to 60 years, the incidence of primary brain tumors peaks [18], reaching approximately 15% in patients with PSs [19-23]. Approximately 30% of patients with PE have mass lesions like neoplasms or vascular malformations as the cause of their seizure disorders [23-27]. Aura occurs in more than 50% of patients with CPS, which may be useful in localizing the onset of seizure [27-34]. EEG is used in the assessment of patients with PE [33-37]. A positive yield between 60 and 93% has been claimed [36,38,43-49].

Patients and Methods

All patients with partial seizure and partial with secondary generalization were enrolled in this study who referred for neurological consultation to Al- Batool Teaching Hospital, Baqubah Teaching Hospital & Al-Yarmouk Teaching Hospital from (Nov.2016-Dec. 2018). This was fortified through a detailed history, elaborate examination, plus nervous system. All patients were evaluated by an interview questionnaire from patient themself or their relatives. The patients were examined, investigated, and treated by a senior (neurologist) in the above-stated centers. All patients had 16 channel EEG tracing (by use of Nihon Kohden Corporation: 432 l F), sometimes more than one record is needed with highlighting on initiating process like hyperventilation, or photostimulation if needed. Each tracing is for 20 minutes. Some of the EEGs were done in a private clinic; quality control over the tracing was exercised heavily. All patients had neuroimaging like brain spiral CT (Somato tom Plus 4- Siemens, Version C l OB) plus/minus contrast when required. MRI (Gyro scan NT 1.5 tesla power. Philips Medical System) was carried out in patients with no anomalies shown by CT and also for the patients with doubt of tumors or arteriovenous deformity. All these neuroimaging were done and reported by radiologists of the centers stated above. Other investigations, like blood sugar, blood urea, electrolytes, and complete blood count were also done.
Ethical consent

Epilepsy is not an uncommon disease, so the clinician must take care of it. The disease can be treated effectively if a precise and accurate diagnosis is done. MRI and CT scan of the brain should be done to exclude secondary cause of epilepsy especially in partial seizures with aura.

Statistical analysis

The data were processed and analyzed using the Statistical Package for Social Sciences version 23 (SPSS Inc., Chicago, IL, USA. The results were expressed using percentages and frequencies. A p-value <0.05 was considered significant.

Results

The age of the patients extended from 6-75 years; 52 males & 54 females as shown in table 1. Sixty-five patients with partial epilepsy had lesions that were revealed on MRI &CT were shown in table 2. The patients who presented with simple partial seizure are 16 (15.1%), while 19 (17.9%) patients complained of complex-partial seizure, however secondary generalization occurred in 71 patients (67%) as shown in table 3. Nineteen patients who had a history of febrile convulsions in childhood, 5 (26%) of them had a focal area of discharge at frontoparietal region, while 14 (74%) patients had a focal area of epilepsy at the temporal lobe. In those patients, focal areas of epilepsy at the temporal lobe had changes in MRI go with mesial temporal sclerosis in 6 (32%) patients. Family history was documented in 30 patients, 18 of them (60%) of patients had a family history of G.T.C epilepsy while 12 patients 40% had a history of partial epilepsy in their family. From those patients with a family history of epilepsy, they had a history of febrile convolution in 16 (53%) patients. The association of simple partial seizures and complex partial seizures with brain lesions: were shown in table 4. The association of partial seizures with brain lesions as detected by neuro-imaging it was abnormal in 52 out of 69 (75.3%) of patients with SPS, while in CPS it was 13 out of 37 (35.1%). The relationship between partial seizures duration and abnormal neuro-imaging were shown in table 5., when there is a long duration of epilepsy the abnormalities on neuroimaging or structural brain abnormalities decrease in frequency.

| Table (1): Demographic data (Age and Gender) of 106 affected patients |
|-----------------|-----------------|--------------|--------------|
| Age (years)     | Number of patients | Male | Female |
| 6-15            | 23              | 10  | 13  |
| 16-25           | 22              | 12  | 10  |
| 26-35           | 20              | 9   | 11  |
| 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 (2): Frequency of lesions in 106 patients with PE

| 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 (3): Types of partial seizures seen in 106 patients

| Types of seizures | Number of patient | % |
|-------------------|-------------------|---|
| Simple partial    | 16                | 15.1 |
| Complex partial   | 19                | 17.9 |
| Secondary G.T.C   | 71                | 67  |
| Total             | 106               | 100 |

Table (4): Association of types of seizures and brain foci in 106 affected patients

| Type of seizure         | Brain area of foci | No. of patient | %   |
|-------------------------|--------------------|----------------|-----|
| Complex partial         | Temporal           | 31             | 83.7|
|                         | Frontal            | 6              | 16.2|
| Simple partial          | Frontal            | 34             | 49.0|
|                         | Parietal           | 13             | 19.0|
|                         | Pronto-parietal    | 22             | 32.0|
| Total                   | 106                |                |     |

Table (5): Relationship between partial seizures duration and abnormal neuro-imaging

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

Discussion

In this study, there are 65 (61%) patients with partial epilepsy had abnormal neuroimaging (CT and MRI), while there are no abnormalities in 41 (39%) patients. These results are slightly more than in previously reported studies 23-55% [40,41]. This may be attributed to that our study was hospital-based and more stringent inclusion of patients. Cerebral vascular accidents or diseases as a cause of partial epilepsy was
found in 27 (25.5%) patients and mostly at age above 40. which is more than in previous studies [25,37,42] which revealed that the percentage of vascular disease as a cause of the partial seizure is 18% that may be due to different sociodemographic characteristics. The frequency of tumors as a cause of partial epilepsy was found in 21 (19.7%) patients and most of them are under the age of 40, in comparison with 20% in previously reported studies [29,30,43] and more than that reported in other studies 4-12% [29,30,32,44]. This can be explained by the nature of this study which is a prospective one with particular attention to finding out the causes of partial epilepsy in our patients and we collected the patients that visited our hospitals complained of partial epilepsy only. In this study, patients complained of simple partial seizure constituted 69 (65%), while 37 (35%) of patients complained of complex partial seizures, these results were consistent with previously reported study [32]. Fifty-six (52.8%) patients complained of headaches most of them associated with brain lesions and temporal lobe epilepsy in which occur at the same side of the focal area. These results are in agreement with previously reported studies [4,6,9]. Two-third with temporal lobe usually had an ipsilateral headache. There is a high association of structural anomaly in brain & SPS occurred in 52 75.3% as opposed to 13 35.1% patients. In CPS, our findings are comparable to previous reports (48-71%) [44,47,48]. Regarding febrile convulsion, we found an increasing incidence with temporal epilepsy especially mesial temporal sclerosis 32% which is less than in previous reports. This finding may be attributed to misdiagnosis by radiologists& there are no relations between a degree of sclerosis & atrophy in MRI technique & history of seizure [4’6]. These alterations may happen in people that never had seizures [40]. In another study, the identification of mesial sclerosis was incidental by MRI & but when significant needs investigation [51]. We found that 60% of patients with SPE & 40% of patients with complex PE had a family history of epilepsy which is also reported by several studies [14,18,20,28] that suggest an increase in the incidence of epilepsy in family members of a patient with PE. There are 16 (53%) patients had febrile convulsion had a family history of epilepsy. This result also consistent with previous reports [15] that genetic predisposition for seizures may be articulated early by happening of febrile convulsion. Most foci in patients with CPS occurred at the temporal lobe 92% and 18% at the frontal lobe while the patients with SPE, the most foci are in frontal (34%), then frontoparietal (22%), and the least is parietal lobe (13%). These results are comparable with previously reported studies [2,15,44,49] which revealed that 80% of CPE is of temporal lobe while most extra temporal epilepsy originates from the frontal lobe. We noticed that when there is prolonged duration of epilepsy the frequency of structural anomalies of brain lesions reduced while when there is short duration, neuroimaging abnormalities will augment these results were consistent with previous reported study [31,45,49], which may be a clinical fact.

**Conclusions**

Most patients with partial seizures are associated with the unusual neuroimaging
study, most etiologies of partial seizure above the age of 40 are cerebral vascular abnormalities while below this age is tumors in high frequency. In patients with SPE the frontal lobe were responsible for epilepsy while in CPE temporal lobe was prevalent. There is a link between febrile convulsions and temporal lobe epilepsy especially temporal sclerosis and epilepsy.

**Recommendations**

Epilepsy is not uncommon disease, so the clinician must take care of it. The disease can be treated effectively if precise and accurate diagnosis is done, MRI and CT scan of the brain should be done to exclude secondary cause of epilepsy especially in partial seizures with aura.

**References**

[1] Allan H. Ropper, Martin A. Samuels, Joshua P. Klein, Sashank Prasad Epilepsy and other seizures disorders; in Adams and Victor's Principles of neurology 11th ed. New York, McGrew Hill, 2019. ch.15; pp331-363.

[2] Cascino GD. Intractable partial epilepsy evaluation and treatment. Mayoclin Proc.1990 65:15758-1586.

[3] Lowenstein DH: Seizure and epilepsy. In: Braunwald E, Hauser SL, Tameson JL: Harrison• principle of internal medicine, 15th ed. New York, MC Graw Hill, 2001.pp2354-2369.

[4] Roger P. Simon, David Greenberg, Michael J. Aminoff: Investigative Studies; Evaluation Of Suspected Epilepsy in Clinical neurology, 10thed, tam ford Connecticut, Appleton and Lange, 2019.

[5] Schomer DL. Partial epilepsy. N. Eng J med 1983 309:536-539.

[6] Allen CMC, Lue CKCT: Disease of nervous system. In: Edward sCRE, Bouchier DA: Davidson's principles and practice of medicine, 18thed, UK, Churchill livingstone, 1999.

[7] Wilkin Son S. Essential neurology, 3rd ed. London, Blackwell science, pp192.

[8] Laidlaw T, Richens A. Oxley J. Textbook of epilepsy, 3rd ed Edinburg Churchill Livingstone. 1988. pp153.

[9] Golden Sohn ES, Purpura DP. Intracellular potentials of cortical neurons during focal epileptogenic discharges. Science 1963:39:840-842.

[10] Prince DA, Futamachi KJ. Intracellular recordings in chronic focal epilepsy. Brain Res 1968;II:681-684.

[11] Daube JR, Reagan TJ., Sandos BA.: Medical neurosciences on approach to anatomy, Pathology, physiology by system level, 2nd ed, Boston, little brown, 1986. pp397.

[12] Suleiman J, Dale RC. The recognition and treatment of autoimmune epilepsy in children. Dev Med Child Neurol. 2015;57(5):431-440. doi:10.1111/dmcn.12647

[13] Liu Y, Guo XM, Wu X, Li P, Wang WW. Clinical Analysis of Partial Epilepsy with Auras. Chin Med J (Engl). 2017;130(3):318-322. doi:10.4103/0366-6999.198918

[14] Mannagetta GB. Genetic of the epilepsy. Berlin, Springer-Verlag, 1989, pp79.

[15] Spencer D. Auras are frequent in patients with generalized epilepsy. Epilepsy Curr. 2015;15:75–7. doi: 10.5698/1535-7597-15.2.75.
Partial Seizures, Etiologies and Associated Comorbidity Factors

[16] Camfield PR, Camfield CS. Intractable seizures after a lengthy remission in childhood-onset epilepsy. Epilepsia. 2017;58(12): 2048-2052. doi:10.1111/epi.13916.
[17] Rayn or R, Daine R. Carmichele E. Epilepsy at late onset. urology. 1959; 9: 1-7.
[18] Woodcock Cosgrove JBR. Epilepsy after the age of 50: a five years follow up-study. Neurology. 1984; 47:225-230.
[19] Reisman D, Fitz-Hugh T Jr. Epilepsiatarda. Anu.Interu Med.1927;1:273-282.
[20] Shorven SD, Gilliatt RW., Cox TC; 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. Clinicalneurology2nd ed, London . Sydney. Auckland, 1998, pp 249.
[22] HiYoshi T, Yagi K. Epilepsy in late onset epilepsy. Journal of Neurology, Neurosurgery and Psychiatry. 1984; 47:225-230.
[23] Ramirez-Izcoa A, Varela-Gonzalez D, Fonseca MI. Caracteristicas de las lesiones estructurales en pacientes pediátricos con epilepsia focal en un hospital de Honduras [Characteristics of structural injuries in pediatric patients with focal epilepsy in a Honduran hospital]. Rev Neurol. 2017;65(3):105-111.
[24] Jabbor B Prokhorcnko O, Khajavi K. 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] Shorborough FW : Complex partial seizure. In: H Luders, RP Lesser . Epilepsy: Electro clinical syndromes. London, Springer-Verlog. 1 987,pp279-302.
[27] Heck CN, King-Stephens D, Massey AD, et al. Two-year seizure reduction in adults with medically intractable partial onset epilepsy treated with responsive neurostimulation: final results of the RNS System Pivotal trial. Epilepsia. 2014;55(3):432-441. doi:10.1111/epi.12534.
[28] Electro clinical syndromes . London, Springer- Verlog , 1987, pp 223-278.
[29] Walker EB .Shorborough FW. The significance of lateralized ictal paretic accruing during complex partial seizure. Epilepsia 1988: 29 :665.
[30] Wyllie Ej., Luders H., Morris HH : The lateralizing significance of versive and eye movements during epileptic seizures. Neurology 1988; 36 : 606-611.
[31] Bonelli B: Banmgertner C. Frontal lobe epilepsy clinical seizure semiology. Weinklinwachenciner2002 ; 114 (8-9) :334-340.
[32] Yaqub B, Danayiotopoulos CP., Al-Nazha M.: Cause of late onset epilepsy in Saudi Arabian: the rate of cerebral granuloma. J Neural, Neurosurgery, Psychiatry 1987; 50 :90-92.
[33] Manford M . Hart Ym., Sander JE:. National general practice study of epilepsy (NGPSE) : Partial seizure pattern in general population Neurology 1992 ; 42 : 1911-1917.  [34] Amit V ., Rodney R.: EEG of Partial Seizures ; J Clin Neurophysiol 2006;23: 333–339.
Partial Seizures, Etiologies and Associated Comorbidity Factors

[35] Engel JJr: Seizure and epilepsy. Philadelphia F.A. Davis Company, 1989, pp 303-339.
[36] Kooi KA, Tucker PD, Marked RE: Fundamentals of electroencephalography. 2nd ed. New York, Harper and Row, 1978.
[37] Zhang Y, Wang B, Jing J, Zhang J, Zou J, Nakamura M: A Comparison Study on Multidomain EEG Features for Sleep Stage Classification. Comput Intell Neurosci. 2017;2017:4574079. doi:10.1155/2017/4574079.
[38] Rovit RL, Gloor P, Rasmussen T: Sphenoidal electrodes in the electrographic study of patients with temporal lobe epilepsy: an evaluation. T Neurosurgery 1981; 18:151-158.
[39] Beleza P: Refractory epilepsy: a clinically oriented review. Eur Neurol (2009) 62:65–71.10.1159/00022775.
[40] Ułamek-Kozioł M, Czuczwar SJ, Januszewski S, Pluta R: Ketogenic Diet and Epilepsy. Nutrients. 2019;11(10):2510. doi:10.3390/nu11102510.
[41] Bergen D, Bleck T, Ramsey R: Magnetic resonance imaging as a sensitive and specific predictor of neoplasm removal for intractable epilepsy. Epilepsia 1989; 30:318-321.
[42] Von Oertzen TJ: PET and ictal SPECT can be helpful for localizing epileptic foci. Curr Opin Neurol. 2018;31(2):184-191. doi:10.1097/WCO.0000000000000527.
[43] Engel JJr: seizure and epilepsy. Philadelphia FA Davis Company 1989:443-474.
[44] Andermann F: 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.
[45] Bernasconi A, Bernasconi N, Bernhardt BC, et al.: Advances in MRI for ‘cryptogenic’ epilepsies. Nat Rev Neurol. 2011;7:99–108.
[46] Cendes F, Theodore WH, Brinkmann BH, Sulc V, Cascino GD: Neuroimaging of epilepsy. Handb Clin Neurol. 2016;136:985-1014. doi:10.1016/B978-0-444-53486-6.00051-X.
[47] Cendes F., Theodore, W. H., Brinkmann, B. H., Sulc, V., & Cascino, G. D. (2016). Neuroimaging of epilepsy. Handbook of clinical neurology, 136, 985–1014. https://doi.org/10.1016/B978-0-444-53486-6.00051-X.
[48] Roy T, Pandit A: Neuroimaging in epilepsy. Ann Indian Acad Neurol. 2011;14(2):78-80. doi:10.4103/0972-2327.82787.
[49] Vattipally VR, Bronen RA: MR imaging of epilepsy: strategies for successful interpretation. Neuroimag Clin N Am 2004;14:373–400.
[50] A. Bernasconi, F. Andermann, N. Bernasconi, D.C. Reutens, F. Dubeur: Lateralizing value of peri-ictal headache: A study of 100 patients with partial epilepsy, January (1 of 2) 2001 Neurology 56;DOI: https://doi.org/10.1212/WNL.56.1.130.
[51] Finegersh A, Avedissian C, Shamim S, Dustin I, Thompson PM, Theodore WH: Bilateral hippocampal atrophy in temporal lobe epilepsy: effect of depressive symptoms and febrile seizures. Epilepsia. 2011;52(4):689-697.