Majalah Kedokteran Sriwijaya
Th. 53 Nomor 1, January 2021

CORRELATION OF COGNITIVE FUNCTIONS AND LOCATION OF FOCAL EPILEPSY IN RSUP DR. MOHAMMAD HOESIN PALEMBANG

Yusril Harun, Sri Handayani, Rini Nindela

Lecturer, Department of Neurology, Faculty of Medicine, Sriwijaya University / RSUP Dr. Mohammad Hoesin, Palembang

Email: rini_nindela@unsri.ac.id

ABSTRACT

Cognitive impairment is one of the most common consequences of epilepsy and has a major impact on the patient's life. Temporal lobe epilepsy is the most common type of focal epilepsy and is reported to be closely associated with cognitive impairment. Patients with other focal epilepsy syndromes such as frontal, parietal, and occipital lobe epilepsy are also known to experience cognitive impairment, although studies are not as numerous as temporal lobe epilepsy. The relationship between certain epileptic foci and complications of cognitive impairment in patients is not yet certain. This study was conducted to determine the correlation between cognitive function and the location of focal epilepsy in the neurology clinic of RSUP Dr. Mohammad Hoesin (RSMH) Palembang.

This research is an analytic observational study with a cross sectional approach. This research was conducted at the RSMH Palembang to collect secondary data from medical records of outpatients between 1 January 2019-31 December 2019 and at the Neurology Clinic to collect primary data on patients seeking treatment between 1 July-31 August 2020. Focal epilepsy location data based on clinical syndrome, age of epilepsy onset, gender, education, frequency and duration of seizures, duration of epilepsy, type of antiepileptic drugs (AED), and duration of consuming AED were obtained through medical record and cognitive function was examined using the validated Indonesian version of the Montreal Cognitive Assessment (MoCA-Ina). From 42 samples, 29 people (69%) had decreased cognitive function and only 13 people (31%) had normal cognitive function. The correlation analysis between cognitive function and focal epilepsy location resulted in correlation coefficient of 0.107 (weak correlation) with p value of 0.365 (not significant). Other factors such as age of onset, level of education, frequency of seizures, duration of seizures, type of treatment, duration of epilepsy and duration of taking AED were also not associated with cognitive function of patients in this study. Many factors have been reported to affect cognitive function in epileptic patients, so it is unlikely that one single factor can be sole factor causing cognitive impairment in epilepsy patients.

Keywords: focal epilepsy location, cognitive function, MoCA-Ina

1. INTRODUCTION

According to the Indonesian Association of Neurology Specialists, epilepsy is defined as a brain disorder characterized by a tendency to cause continuous epileptic seizures with various consequences, one of which is cognitive.[1] Cognitive abnormalities are among the most common and severe comorbidities of epilepsy. It is reported that 30% -40% of epilepsy patients experience impaired cognitive function.[2] Epilepsy patients, especially those resistant to pharmacotherapy or who have seizures from an early age, are at increased risk of mental retardation, learning disabilities and memory impairments. This cognitive comorbidity greatly affects the quality of life of patients with epilepsy because it has a major impact on the patient's educational process and life-long achievement.[3]

The association of cognitive function and epilepsy has been most studied in temporal lobe epilepsy because this syndrome is the most common cause of focal epilepsy. In addition, Bonn *et al* reported that 80% of pharmacoresistant epilepsy patients were those with temporal lobe epilepsy. Temporal lobe epilepsy also

generally manifests early (early onset) and the patient often has a history of febrile seizures, this long total duration of seizures increases the likelihood of cognitive impairment. Impaired cognitive function in temporal lobe epilepsy is reported to cover many domains including memory, executive function, language, and processing speed.[4,5]

Frontal lobe epilepsy is the second most common type of focal epilepsy (20-30%). Frontal lobe epilepsy is also the second most common cause of pharmacoresistence seizures (15%).[6] Although there are fewer studies than those with temporal lobe epilepsy, the available data show that frontal lobe epilepsy patients may experience memory problems (especially working memory), social cognition, and attention, while intelligence is generally still normal.[6-8] . The features of cognitive function in other focal epilepsy syndromes, namely parietal and occipital lobe epilepsy, have not been widely reported because of their low prevalence. Parietal lobe epilepsy syndrome is present in 5-6% of patients with focal epilepsy. Occipital lobe epilepsy is not much different, being the cause of 5-8% of focal epilepsy.[9] The studies available so far report cognitive impairment in the spatial domain, orientation, touch perception, constructional apraxia and neglect in patients with parietal lobe epilepsy. Other studies have shown an association between seizures originating in the right parietal lobe and deficits in spatial and constructional function and anosgnosia, whereas seizures of the left parietal lobe cause impaired reading, writing, and calculations or generally affect the patient's verbal ability.[9] Piazzini *et al* reported that some patients with occipital lobe epilepsy had mild problems with naming. Bilo *et al* also reported the presence of complex visuospatial disorders, constructional abilities and executive function in patients with occipital lobe epilepsy. In addition, cortical blindness, simultanagnosia, and visual hallucinations have also been reported in patients with occipital lobe epilepsy.[10-11]

Many factors have been reported to increase the risk of epilepsy patients developing cognitive impairments. In addition to the pathogenesis of epilepsy itself, namely the epileptogenic focus as described in the previous paragraph, other factors such as the duration of epilepsy, the adverse effects of AED, and the educational level of patients have also been reported to have an effect on cognitive dysfunction.[3] This study aims to find out whether a particular focus of epilepsy is definitely associated with cognitive impairment in patients because the available studies are generally carried out separately according to the type of syndrome.

2. METHOD

This research is an analytic observational study with a cross sectional approach. This research is conducted at the Medical Record Installation of Dr. Moh. Hoesin (RSMH) Palembang to collect data on patients who came for medical treatment between 1 January 2019-31 December 2019 and also at the Neurology Clinic to collect data on patients undergoing treatment between 1 July-31 August 2020. The total sampling method was applied in this study. All subjects who meet the inclusion criteria, namely (a) age ≥18 years, (b) diagnosed with focal epilepsy syndrome through clinical symptoms, (c) have received epilepsy treatment, and (d) have undergone MoCA-Ina examination and the results are recorded in medical records (for secondary data) will all be included as study samples. Subjects with unreadable / damaged / missing medical records, illiterate, deaf / speech impaired or experiencing decreased consciousness were excluded from this study. Data on independent variables such as location of focal epilepsy based on clinical syndrome, age of epilepsy onset, sex, education, frequency and duration of seizures, type of treatment and duration of

epilepsy and AED were obtained through medical record, while data on the dependent variable, namely cognitive function, was examined using the validated Indonesian version of the Montreal Cognitive Assessment (MoCA-Ina). The data was processed using SPSS for windows. This research had been declared to be ethically appropriate by Health Research Ethics Committee of RSMH Palembang by decree number 95/kepkrsmh/2020.

3. RESULTS

During the months of July-August 2020, there were only 3 subjects who met the inclusion criteria of the study, while through the search for medical records, there were 39 subjects, so that overall there were 42 research samples. Of these 42 samples, 18 people (42.9%) had frontal lobe epilepsy syndrome, 17 people (40.5%) had temporal lobe epilepsy syndrome, 4 people (9.5%) had occipital lobe epilepsy syndrome and 3 people (7.1%) had parietal lobe epilepsy syndrome.

The table below describes the characteristics of the study sample based on the location of the focal epilepsy they experienced. It can be seen that the majority of focal epilepsy syndromes (64.3%) were experienced by patients since they were less than 18 years of age. Male patients (59.5%) were slightly more than female patients (40.5%).

Table 1. Characteristics of Subjects by Location of Focal Epilepsy

Variable	n (%)				Total n (%)	p value
	Temporal epilepsy	Frontal epilepsy	Parietal epilepsy	Occipital epilepsy		
Age of Onset						
<18 years	10 (37)	12 (44.4)	2 (7,4)	3 (11.1)	27 (100)	0.925
≥18 years	7 (46.7)	6 (40)	1 (6,7)	1 (6,7)	15 (100)	
Gender						
Male	10 (40)	10 (40)	3 (12)	2 (8)	25 (100)	0.51
Women	7 (41.2)	8 (47.1)	0 (0,0)	2 (11.8)	17 (100)	
Basic education						
0-9 years	2 (20)	5 (50)	1 (10)	2 (20)	10 (100)	0.365
> 9 years	15 (46.9)	13 (40.6)	2 (6.3)	2 (6.3)	32 (100)	
Frequency of seizure						
≥1x / month	10 (38.5)	12 (46.2)	1 (3.8)	3 (11.5)	26 (100)	0.666
<1x / month	7 (43.8)	6 (37.5)	2 (12.5)	1 (6.3)	16 (100)	
Duration of seizure						
≥15 minutes	3 (42.9)	4 (57.1)	0 (0,0)	0 (0,0)	7 (100)	0.612
<15 minutes	14 (40)	14 (40)	3 (8,6)	4 (11.4)	35 (100)	
Type of epilepsy treatment						
polytherapy	11 (45.8)	8 (33.3)	2 (8,3)	3 (12.5)	24 (100)	0.529
monotherapy	6 (33.3)	10 (55.6)	1 (5,6)	1 (5,6)	18 (100)	
Duration of epilepsy						
≥5 years	12 (41.4)	12 (41.4)	2 (6.9)	3 (10.3)	29 (100)	
<5 years	5 (38.5)	6 (46.2)	1 (7,7)	1 (7,7)	13 (100)	0.987
Duration of OAE consumption						
≥2 years	16 (42.1)	15 (39.5)	3 (7,9)	4 (10.5)	38 (100)	0.559
<2 years	1 (25)	3 (75)	0 (0,0)	0 (0,0)	4 (100)	

**Chi Square test*

Most of the patients with focal epilepsy syndrome in this study had completed their basic education (76.2%). At the onset of focal epilepsy, most of the patients had seizures ≥1 times / month (62%) with a duration of less than 15 minutes (83.3%). More temporal, parietal and occipital epilepsy patients who consumed more than one type of AED (polytherapy) with a percentage of 64.7%, 66.7%, and 75%, respectively. Otherwise, the percentage of frontal lobe epilepsy patients who received only one drug (monotherapy) was slightly higher than those who received polytherapy, amounting to 55.6% and 44.4%, respectively. Most of the patients in this study had epilepsy for ≥5 years (69%) and consumed OAE ≥2 years (90.1%). When bivariate analysis was performed, all of these variables did not show a significant relationship with the type of focal epilepsy syndrome.

The characteristics of research subjects based on their cognitive function can be seen in the table below. Focal epilepsy patients with decreased cognitive function (69%) were more than patients whose cognitive function was still normal (31%). Patients who experienced a decline in cognitive function were mostly from the age group of onset <18 years (62.1%) and male (65.5%).

Table 2. Characteristics of Subjects based on Degree of Cognitive Function

Variable	n (%)		Total n (%)	OR (CI 95%)	p value
	Decreased cognitive function	Normal cognitive function			
Age of Onset					
<18 years	18 (66,7)	9 (33.3)	27 (100)	0.727	0.466b
≥18 years	11 (73.3)	4 (26.7)	15 (100)	(0.18-2,939)	
Gender					
Male	19 (76)	6 (24)	25 (100)	2,217	0.237a
Women	10 (58.8)	7 (41.2)	17 (100	(0.585-8,402)	
Basic education					
0-9 years	8 (80)	2 (20)	10 (100)	2,095	0.33b
> 9 years	21 (65,6)	11 (34.4)	32 (100)	(0,378-11,615)	
Frequency of seizure					
≥1x / month	19 (73.1)	7 (26.9)	26 (100)	1,629	0.35b
<1x / month	10 (62.5)	6 (37.5)	16 (100)	(0.430-6,173)	
Duration of seizure					
≥15 minutes	6 (85.7)	1 (14.3)	7 (100)	3.13	0.287b
<15 minutes	23 (65.7)	12 (34.3)	35 (100)	(0.337-29,086)	
Type of epilepsy treatment					
polytherapy	16 (66,7)	8 (33.3)	24 (100)	0.769	0.7a
monotherapy	13 (72.2)	5 (27.8)	18 (100)	(0.202-2,925)	
Duration of epilepsy					
≥5 years	18 (62.1)	11 (37.9)	29 (100)	0.298	0.135b
<5 years	11 (84.6)	2 (15.4)	13 (100)	(0.055-1,601)	
Duration of OAE consumption					
a. ≥2 years	25 (65.8)	13 (34.2)	38 (100)	0.658	0.212b
b. <2 years	4 (100)	0 (0,0)	4 (100)	(0.523-0,827)	

[a]Chi Square test, [b]Fischer exact test

The proportion of patients with a higher level of education and decreased cognitive function (65.6%) was greater than that of patients with the same education level but still normal cognitive function (34.4%). Most of the focal epilepsy patients with decreased cognitive function experienced seizures that were quite frequent, ≥1 times / month (65.5%) with a duration of <15 minutes (79.3%). The number of patients who received polytherapy was more than those who received monotherapy, both in the group of patients whose cognitive function was still normal (61.5%) and those who had decreased (55.2%). The majority of focal epilepsy patients with decreased cognitive function had epilepsy for ≥5 years (62%) and had consumed OAE for ≥2 years (86.2%). In the bivariate analysis, none of the variables had a significant relationship with cognitive function.

Patients with temporal, frontal and occipital lobe epilepsy mostly experienced a decline in cognitive function with the proportion of 76.5%, 66.7%, and 75% respectively, whereas in parietal lobe epilepsy the opposite occurred where the number of patients whose cognitive function is still normal (66.7%) greater than the declined one (33.3%) as shown in the following table.

Table 3. Relationship between Focal Epilepsy Location and Cognitive Function

Locations of Focal Epilepsy	Cognitive function		Total
	Decreased (MoCA-Ina score <26)	Normal (MoCA-Ina score ≥26)	
Temporal	13 (76.5%)	4 (23.5%)	17 (100%)
Frontal	12 (66.7%)	6 (33.3%)	18 (100%)
Parietal	1 (33.3%)	2 (66.7%)	3 (100%)
Occipital	3 (75%)	1 (25%)	4 (100%)

When analyzed to determine the correlation between the location of focal epilepsy and cognitive function using Somer's test, a correlation coefficient of 0.107 was obtained, which indicates a weak correlation with a significance value of 0.365 which means this correlation is not statistically significant. In addition, a multiple stepwise logistic regression test was also carried out to determine the relationship between the location of focal epilepsy and cognitive function by considering the influence of other factors but there was not any variable that was related to the cognitive function of focal epilepsy patients in this study. However, the mean MoCA-Ina score of all patients (22.10 ± 5.378) was lower than the normal range (≥26).

4. DISCUSSION

Seizures are not the only problem faced by epilepsy patients, but there are various other common and quite severe comorbidities; cognitive disorders such as decreased memory and attention function, mental health disorders such as depression and anxiety, and somatic disorders such as migraines and sleep disorders. These comorbidities are often seen as more troublesome than the seizure itself. Among the various comorbidities experienced by these epilepsy patients, cognitive abnormalities were the most common.[12] There were 69% of patients who experienced a decline in cognitive function in this study, which is higher from the Kumar and Vatsala report who found cognitive impairment in only 36% of their

study subjects. This difference can be caused by differences in the sample. This study only included subjects with focal epilepsy syndrome while the Kumar and Vatsala study included subjects with various types of seizures.[13]

In this study, although there was no statistical association, the number of patients who experience cognitive decline is more in the group who have had epilepsy since the age of <18 years compared with those whose epilepsy onset occurs at age ≥18 years. Past studies, including one reported by Farwell *et al* have found a higher incidence of low IQ in children with epilepsy than in healthy children. In addition, children with pharmacoresistant seizures tended to have lower IQs than children who responded well to epilepsy treatment. However, Thompson and Duncan in 2005 and Bell *et al* in 2011 reported that adults with chronic epilepsy are also prone to experiencing cognitive regression.[2]

Most of the focal epilepsy patients with cognitive impairment in this study were male. Overall, the sex proportion of subjects in this study was more male than female (25 vs 17), similar to that reported by Moorthy *et al* (63 vs 37).[14] This study did not find a significant relationship between the education level of patients with cognitive function, in contrast to the report by Wang *et al* that higher education level is a protective factor for the cognitive function of patients with epilepsy. Study of Wang *et al* used more than one neurocognitive examination methods including mini mental state examination / MMSE, verbal fluency test, digit span test and digital symbol test in addition to the MoCA-Ina, whereas cognitive function in this study was only based on the MoCA-Ina score. This difference in method may be the cause of the discrepancy in the results obtained.[3]

The proportion of focal epilepsy patients who experienced a decline in cognitive function in this study was greater in the group whose seizure frequency was higher (≥1 times / month) and had longer duration of epilepsy (≥5 years), although the duration of seizures was shorter (<15 minutes). The study by Kumar and Vatsala also found no significant association between seizure frequency and cognitive function, similar to this study.[13] However, that study also found a lower mean cognitive score in the group of patients whose higher seizure frequency than in the group with a lower frequency of seizures. Studies show that recurrent and prolonged seizures (*status epilepticus*) produce morphological and functional changes in neural circuits that directly affect the brain's ability to process information normally. In experimental animals that are induced to experience status epilepticus, there is neuronal loss of both main cells and interneurons throughout the hippocampus, parahipocampus and entorhinal cortex. After the death of these cells, synaptic reorganization is formed with abnormal growth in the form of sprouting of axon granule cells (*mossy fibers*). Sprouting and formation of new synapses also occur in pyramidal neurons. These newly formed synapses cause an increase in glutamatergic synaptic currents. These abnormally located ectopic granule cells in the hilum may contribute to the increased excitability of the hippocampal tissue. Repeated seizures can trigger a persistent decrease in GABA flow in the hippocampus and neocortex, resulting in increased excitation in the neocortex. Nevertheless, when tested whether neuronal loss was directly related to cognitive impairment in experimental animals, only a weak correlation was found.[2] Therefore, it cannot be concluded that one single factor can cause cognitive impairment in epilepsy patients.

Majority of patients with cognitive dysfunction in this study had consumed AED for ≥2 years although statistical analysis did not find a significant relationship between these two variables. Park and Kwon's study examined cognitive impairment in epileptic patients who had taken AED for at least 1 year and did not

find conclusive results even though the majority of previous studies indicated that long-term use of OAE (without a clear time limit) caused cognitive decline. Antiepiletic regimens may affect cognition by suppressing neuronal excitability or increasing inhibition of neurotransmission. The effect of AED on cognitive function appears to be dosage-dependent and is mainly exacerbated by polytherapy. On monotherapy and when serum AED levels are within a safe therapeutic range, this cognitive dysfunction tends to be mild.[12] The study by Miller *et al* also found that the amount of AED consumed was associated with cognitive impairment, especially in the language and visuospatial domains.[15] In this study, the comparison of epilepsy patients with cognitive impairment who received polytherapy and monotherapy was not much different, although more people received polytherapy.

Among the early generations of AED carbamazepine, phenytoin and valproic acid exhibits a relatively similar effect on cognitive function, whereas phenobarbital clearly has a greater effect on cognitive impairment. The effect of the new generation of AED on cognitive function appears to be better than that of the earlier generation. Studies show that gabapentin, lamotrigine, and levetiracetam have fewer side effects on cognitive function when compared to carbamazepine.[14] Among the newer AED generations, topiramate was associated with a greater risk of developing cognitive impairment although this risk was decreased with the administration of the drug via slow titration and low target dose. The current study did not describe the therapeutic regimens consumed by the subjects so that it could not analyze the relationship between certain AED and cognitive function in patients with focal epilepsy. Cognitive abnormalities due to AED generally involve attention, psychomotor speed and memory domains. Another study showed that IQ, learning performance and mood status were generally the same in epilepsy patients and the control group while working memory, executive function and verbal fluency were impaired in the patient group. In Park and Kwon study, it appears that cognitive deficits also do not correlate with patient characteristics, epilepsy variables or the type of OAE, similar to this study.[12]

Although available data suggest that cognitive impairment in epileptic patients is dependent on the pathophysiology of the seizure itself, for example, patients with temporal lobe epilepsy are likely to experience memory impairment or frontal lobe epilepsy patients tend to have problems with their executive function,[5] the current study was unable to find a direct correlation between locations of focal epilepsy syndrome and cognitive function. The differences in the neuropsychiatric tools used may have contributed to this difference in results, in addition to the analysis in this study did not go deep into the comparison of function in each cognitive domain. In structural epilepsy, such as those caused by head trauma, stroke, and encephalitis, this underlying cause plays a role in the decline in cognitive function especially when the deficit is in line with the location of the lesion. Pathogenesis of focal epilepsy of unknown cause is much more complex. This study does not describe the etiology of epilepsy because not all subjects have undergone a complete examination such as magnetic resonance imaging / MRI to find out the exact cause of their focal epilepsy syndrome. In addition, focal epilepsy syndrome does not always represent the ictal and interictal electrical manifestations. The locations of abnormal electrical bursts during seizures and between seizures are often difficult to localize because they are simultaneously connected to various areas of the brain, allowing a rapid and wide-spread epileptic activity.[6] This research was conducted in the midst of the COVID-19 pandemic where the number of hospital visits greatly decreased. The sample obtained in this study did not reach the calculated minimum sample size (68

samples) and had to combine primary data and secondary data from medical records. Cognitive function is based on the MoCA-Ina score only because this test is routinely performed in all epilepsy patients as a screening tools so that data is generally available, while other tests are selective according to the results of initial screening.

5. CONCLUSION

There was no significant direct correlation between cognitive function and the location of focal epilepsy. Cognitive function in epilepsy patients is not influenced by one single factor but is multifactorial starting from the patient's own characteristics, the type of epilepsy, and the AED used. For further reasearch, a similar study can be carried out using an adequate sample size, a variety of neurocognitive examinations and a more in-depth analysis in order to find out what factors mainly affect cognitive function in patients with focal epilepsy for better treatment planning.

ACKNOWLEDGEMENT

This study was supported by research grant from the Medical Faculty of the University of Sriwijaya.

REFERENCES

[1]. Kusumastuti K, Gunadharma S, Kustiowati E (eds). Pedoman Tatalaksana Epilepsi (PERDOSSI). Edisi Kelima. Airlangga University Press. 2014.

[2]. Holmes GL. EEG abnormalities as a biomarker for cognitive comorbidities in pharmacoresistant epilepsy. Epilepsia. 2013; 54(0 2): 60–62. doi:10.1111/epi.12186.

[3]. Wang L, Chen S, Liu C, Lin W, Huang H. Factors for cognitive impairment in adult epileptic patients. Brain Behav. 2020;10(1):1–11.

[4]. Nouha F, Sawsan D, Salma S, Olfa H, Hanen HK, Mariem D, et al. Cognitive Impairment in Patients with Temporal Lobe Epilepsy. J Neuropsychiatr. 2018;02(02):1–7.

[5]. Zhao F, Kang H, You Li, Rastogi P, Venkatesh D, Chandra M. Neuropsychological deficits in temporal lobe epilepsy: A comprehensive review. Ann Indian Acad Neurol. 2014;17(4):374–82.

[6]. Patrikelis P, Angelakis E, Gatzonis S. Neurocognitive and behavioral functioning in frontal lobe epilepsy: A review. Epilepsy Behav [Internet]. 2009;14(1):19–26. Available from: http://dx.doi.org/10.1016/j.yebeh.2008.09.013

[7]. Risse GL. Cognitive outcomes in patients with frontal lobe epilepsy. Epilepsia. 2006;47(SUPPL. 2):87–9.

[8]. Helmstaedter C. Behavioral Aspects of Frontal Lobe Epilepsy. Epilepsy Behav. 2001;2(5):384–95.

[9]. Traianou A, Patrikelis P, Kosmidis MH, Kimiskidis V, Gatzonis S. The neuropsychological profile of parietal and occipital lobe epilepsy. Epilepsy Behav [Internet]. 2019;94:137–43. Available from: https://doi.org/10.1016/j.yebeh.2019.02.021

[10]. Santangelo G, Trojano L, Vitale C, Improta I, Alineri I, Meo R, et al. Cognitive dysfunctions in occipital lobe epilepsy compared to temporal lobe epilepsy. J Neuropsychol. 2017;11(2):277–90.

[11]. Knopman AA, Wong CH, Stevenson RJ, Homewood J, Mohamed A, Somerville E, et al. The cognitive profile of occipital lobe epilepsy and the selective association of left temporal lobe hypometabolism with verbal memory impairment. Epilepsia. 2014;55(8):80–4.

[12]. Park SP, Kwon SH. Cognitive effects of antiepileptic drugs. J Clin Neurol. 2008;4(3):99–106.

[13]. Kumar VJ, Matsala V. Cross sectional study to determine the cognitive impairment among epilepsy patients. Int J Res Med Sci. 2019;7(5):1465.

[14]. Mula M. Cognitive dysfunction in patients with epilepsy: Focus on clinical variables. Future Neurol. 2015;10(1):41–8.

[15]. Miller LA, Galioto R, Tremont G, Davis J, Bryant K, Roth J, et al. Cognitive impairment in older adults with epilepsy: Characterization and risk factor analysis. Epilepsy Behav [Internet]. 2016;56:113–7. Available from: http://dx.doi.org/10.1016/j.yebeh.2016.01.011