REFERRAL OF EPILEPTIC PATIENTS IN NORTH EAST COAST OF WEST MALAYSIA AN AREA WITH POOR MRI COVERAGE : AN ANALYSIS

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Abstract. Advances in neuroimaging techniques, particularly Magnetic Resonance Imaging (MRI), have proved invaluable in detecting structural brain lesions in patients with epilepsy in developed countries. In Malaysia, a few electroencephalography facilities available in rural district hospitals run by trained physician assistants have internet connections to a government neurological center in Kuala Lumpur. These facilities are more commonly available than MRI machines, which require radiological expertise and helium replacement, which may problematic in Southeast Asian countries where radiologists are found in mainly big cities or towns. We conducted a cross-sectional study over a two year period begining January 2001 on rural patients, correlating EEG reports and MRI images with a clinical diagnosis of epilepsy to set guidelines for which rural patients need to be referred to a hospital with MRI facilities. The patients referred by different hospitals without neurological services were classified as having generalized, partial or unclassified seizures based on the International Classification of Epileptic Seizures proposed by the International League Against Epilepsy (ILAE). The clinical parameters studied were seizure type, seizure frequency, status epilepticus and duration of seizure. EEG reports were reviewed for localized and generalized abnormalities and epileptiform changes. Statistical analysis was performed using logistic regression and area under the curve. The association between clinical and radiological abnormalities was evaluated for sensitivity and specificity. Twenty-six males and 18 females were evaluated. The mean age was 20.7 ±13.3 years. Nineteen (43.2%) had generalized seizures, 22 (50.0%) had partial seizures and 3 (6.8%) presented with unclassified seizures. The EEG was abnormal in 30 patients (20 with generalized abnormalities and 10 localized abnormalities). The MRI was abnormal in 17 patients (38.6%); the abnormalities observed were cerebral atrophy (5), hippocampal sclerosis (4), infarct/gliosis (3), cortical dysgenesis (2) and tumors (2). One patient had an arachnoid cyst in the right occipital region. Of the 17 patients with an abnormal MRI, 14 had an abnormal EEG, this difference was not statistically significant. There was no significant associaton between epileptographic changes and MRI findings (p=0.078). EEG findings were associated with MRI findings (p=0.004). The association between an abnormal EEG and an abnormal MRI had a specificity of 82.4%, while epileptogenic changes had a specificity of 64.7% in relation to abnormal MRI findings. This meants that those patients in rural hospitals with abnormal EEGs should be referred to a neurology center for further workup and an MRI to detect causes with an epileptic focus.

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INTRODUCTION

Epilepsy is a common neurological disorder affecting millions of people worldwide. It is estimated that the prevalence of epilepsy is about 5-10 cases per 1,000 population. Certain regions have a high reported prevalence, this is particularly so for countries in Latin America and Africa. Of Southeast Asian countries, only Singapore has estimations of the prevalence of this illness. Puvanendran (1993) reported a lifetime prevalence of epilepsy in Singapore of 3.8 per 1,000 population; it varies with age, sex and race (Puvanendran, 1993). Epilepsy occurs in all ages. In one study conducted at University Hospital Kuala Lumpur, the age of onset of epilepsy ranged from 3 months to 77 years with a mean age of 18.7 years (Manonmani and Tan, 1999).

For many years, electroclinical findings were used to help in diagnosing and managing patients with epilepsy. More often than not, the underlying pathology that was responsible for seizure generation was not known. The advent of modern neuroimaging techniques has reversed this situation and improved the ability to detect the structural basis of the seizure disorder. This is especially important in patients with medically intractable seizures. Magnetic resonance imaging (MRI) an important neuroimaging technique in the assessment of epilepsy but is not available in all developing Southeast Asian countries, such as the East Coast of Peninsular Malaysia where there is currently one MRI per 3.5 million people. The structural abnormalities detected on MRI can be categorized into tumors, cortical dysgenesis, vascular malformations, hippocampal sclerosis, infarct/gliosis and other miscellaneous findings. Even though the identification of a focal structural lesion on MRI may not always indicate the site of seizure origin, correlative studies have shown that certain neuroimaging abnormalities are frequently associated with epileptic process (Spencer, 1994; Cendes et al, 2000).

On the East Coast of West Malaysia, an MRI is requested if there is a strong suspicion of a structural brain abnormality based on the clinical and EEG findings, since nearly all general hospitals have an EEG machine. It is more cost effective to do an MRI than a CT scan of the brain. This study intended to associate the demographic, clinical and EEG findings with MRI results. We also determined the sensitivity and specificity of radiological parameters in detecting MRI abnormalities amongst patients with epilepsy.

METERIALS AND METHODS

A cross-sectional study was conducted on 44 patients with epilepsy who had undergone MRI in the Radiology Department, Hospital Universiti Sains Malaysia (HUSM) between January 2001 and the end of 2003 with ethical approval and written consent.

All epileptic patients referred to the Department of Radiology, HUSM during the study period for MRI of the brain were included.

Inclusion criteria were patients clinically diagnosed with epilepsy who had undergone EEG examination and patients who agreed to undergo an MRI examination of the brain based on the standard epilepsy protocol of the Radiology Department, HUSM.

Exclusion criteria were patients who were known to have underlying brain pathology, infants who were given sedation during EEG examination and patients with incomplete clinical or EEG data.

Definitions of operational terms

Definitions for seizure, epilepsy, and epileptic seizure were based on the classification of seizures and epileptic syndromes by the International League Against Epilepsy (Doherty and Cole, 2001) and were adapted from papers by Puvanendran (1993) and Sirven (2002).

Patients who fulfilled the selection criteria were identified and their clinical information was reviewed. Clinical diagnosis, seizure type, frequency of seizures, status epilepticus and duration of seizures were recorded. Based on this clinical information, the seizure was classified into generalized seizure, partial seizure or unclassified seizure. These seizure types were further sub-classified into abscence seizures, myoclonic seizures, clonic seizures, tonic seizures, tonic-clonic seizures, and atonic seizures for generalized seizures and into simple partial seizures, complex partial seizures, and partial seizures with secondary generalization for partial seizures. The EEG reports of the patients were recorded via a 21 channels EEG, employing scalp electrodes placed according to the international 10-20 system. An anterictal EEG was performed for 20-30 minutes while awake with inclusion of activation by hyperventilation and photic stimulation. A sleep EEG was done in selected cases when the routine EEG was equivocal and further confirmation of the diagnosis was required. The EEG was only reported after the completion of the MRI where the findings were blinded to two neurologists. The background frequencies and paroxysmal activities (epileptiform or non-epileptiform) were recorded. The EEG findings were classified into normal and abnormal. An abnormal EEG was further classified into generalized abnormalities or localized abnormalities. Furthermore; if it was abnormal, then the site of abnormality was recorded according to the cerebral lobe affected. Background frequencies were divided into delta (0.1-3.5 Hz), theta (4-7 Hz), alpha (8-13 Hz/s) and beta (>13Hz/s) and further assessed for any background abnormality, such as focal slowing or diffuse slowing (Gibbs et al, 1993). By incorporating the additional information from the interictal EEG the seizure types were classified according to the International Classification of Epileptic Seizures (Doherty and Cole, 2001).

MRI protocol

All MRI examinations of the brain were done using a 1.0T Signa Horizon LX (GE Medical System) at the Department of Radiology, HUSM. Scanning was performed using a routine clinical epilepsy protocol with 5 mm thickness slices and 2.0 mm interslice gaps in the sagittal plane with T_1 -weighting (TR/TE/NEX 400/10/4) and in the axial plane with T_1 -weighting (520/11/1), T_2 -weighting (4260/79/1) and a Fluid Attenuation Inversion Recovery (FLAIR) sequence (9002/157/1). In addition, an oblique coronal T_1 -inversion recovery sequence (TR/TE/TI/NEX 2000/15/700/1) of the temporal lobe perpendicular to the long axis of the hippocampus was performed with 4.0 mm thickness slices and a 1.0 mm interslice gap. Intravenous contrast media was not routinely given unless there was a strong indication for Gadolinium-DTPA enhancement for cases such as tumors or vascular abnormalities.

Interpretation of MRI

All the images were assessed by two radiologists who was blinded to the patient's clinical background. The scans were examined for any asymmetry of hippocampal size or signal intensity, developmental abnormalities, atrophic abnormalities, grey matter thickness and cortical abnormalities or other lesions. Asymmetry of the hippocampal size and signal intensity was assessed to diagnose hippocampal sclerosis. Head rotation was carefully evaluated by assessing the symmetry of the internal auditory canals and the atria of the lateral ventricles to prevent false positive findings. Developmental abnormalities were assessed by evaluating periventricular regions for subependymal heterotopias. In addition, abnormal cortical or sulcal morphology as well as the corticomedullary junction were scrutinized for indistinctness. Atrophic abnormalities were detected by assessing tissue loss, especially subtle focal cortical atrophy. Other obvious lesions, such as the presence of vascular lesions, infarcts, gliosis or mass lesions indicative of tumors were thoroughly evaluated. The findings were classified into tumors, cortical dysgenesis, vascular malformations, hippocampal sclerosis, infarct/gliosis, atrophy or miscellaneous findings. For a systematic assessment of the MRI images, the method proposed by Bronen et al (1997) was employed.

Statistical analysis

Statistical analysis was done using SPSS for Windows version 12.0. Descriptive analysis was used for socio-demographic data, such as age, race and gender. Clinical and EEG findings were correlated with the MRI findings using cross tabulation. The clinical parameters tested against the MRI findings included seizure type, frequency of the seizure, status epilepticus and duration of seizure. For the EEG parameters, the MRI findings was tested against generalized or localized abnormalities, frequency of EEG waves and epileptiform changes. For univariate analysis, simple logistic regression was applied. The level of significance was set at 0.05. The sensitivity and specificity of the radiological findings were determined by using a receiver operating characteristics (ROC) curve. The cut-off value was set at 0.05, and area under curve was calculated.

RESULTS

A total of 44 patients were analyzed and the age ranges from 5 to 62 years. Most of these patients were young adults with the mean age of 20.7 ± 13.3 years. There were 26 males and 18 females. Forty-two patients (95.5%) were Malays and the other two were Chinese and Thai.

Initial data collection revealed that 26 patients had generalized seizures, 15 partial seizures and three unclassified seizures. With additional information from the interictal EEG, seven patients were subsequently diagnosed as having partial seizures with secondary generalization. These seven patients had interictal EEGs that showed localized abnormalities on either the right or left sides of the cerebral hemisphere except for one patient who had generalized epileptiform changes with diffuse slow background activity. Overall, 19 patients (43.2%) had generalized seizures, 22 patients (50%) had partial seizures and 3 patients (6.8%) had unclassified seizures.

Out of the 19 patients who had generalized seizures, tonic clonic seizure was the most common type (63.1%). This was followed by tonic seizures (26.3%), myoclonic seizures (5.3%) and absence seizures (5.3%). For partial seizures, complex partial seizure was twice as common as simple partial seizure. The 7 patients categorized as having partial seizures with secondary generalization made up 31.8% of the 22 total patients (Table 1). Five patients were diagnosed with temporal lobe epilepsy (TLE) based on clinical assessment. Of these 5 patients, 4 had complex partial seizures. The fifth patient had partial seizures with secondary generalization. Only two patients had a history of status epilepticus.

Newly three quarters of the cases (32 patients/72.7%) had a history of seizures for five years or less. The duration ranged from 1 year to 30 years with a mean of 5 ± 5.9 years. The longest history of seizures occurred in a 45 year-old women with complex partial seizures. The MRI for this patient was normal. Seven patients had a history of seizures for 6 to 10 years, and the other five patients had a history of seizures for more than 10 years.

A total of 30 patients had abnormal EEGs (68.2%); 2/3 of them had generalized abnormalities and 1/3 had localized abnormalities. Of the 20 patients with generalized EEG abnormalities, 18 had generalized epileptiform changes and 2 had generalized (diffuse) slow background activity. Of the 10 patients with localized EEG abnormalities, seven had focal epileptiform changes and three had focal slow background activity. These findings gave rise to a total of 25 patients with epileptiform changes and five patients with slow background abnormalities.

A total of 12 patients had abnormal slow wave activity, but focal slow activity was found in 4 of them. The other 8 patients had diffuse slow background activity, which could have been due to post-ictal changes. Only one pa-

NR	NR Age Seizure type		EEG abnormality	MRI abnormalities	Site	
1	6	Simple	None	Glioma	Left parietal	
2	15	Simple	None	HS	Right temporal	
3	9	Simple	Generalized epileptiform with diffuse slowing	CD	Left temporal	
4	21	Simple	Left temporal focal delta way	ve Infarct	Left temporal	
5	14	Complex	Generalized epileptiform with diffuse slowing	HS	Right temporal	
6	5	Complex	Right focal slowing	Atrophy	Bilateral hemisphere	
7	18	Complex	Generalized epileptiform	HS	Left temporal	
8	12	Secondary generalization	Diffuse slowing	HS	Left temporal	
9	16	Secondary generalization	Left temporal epileptiform	DNET	Left temporal	
10	15	Secondary generalization	Central focal epileptiform	Atrophy	Frontal region	
11	62	Secondary generalization	Left hemisphere epileptiform changes	Infarct	Left parietal	
12	53	GTC	Normal	Atrophy	Bilateral hemisphere	
13	38	GTC	Generalized epileptiform with diffuse slowing	Infarct/Gliosis	Right temporal	
14	18	Generalized tonic	Generalized epileptiform with focal slowing	Arachnoid cyst	Right occipital	
15	15	GTC	Generalized epileptiform	CD	Right temporal	
16	22	GTC	Generalized epileptiform	Atrophy	Bilateral hemisphere	
17	32	GTC	Excessive beta wave	Atrophy	Bilateral hemisphere	

Table 1 Summary of 17 patients with type of seizure and abnormal MRI.

HS = hippocampal sclerosis, CD = cortical dysgenesis, DNET = dysembryonic neuroepitelial tumor GTC = generalized tonic clonic seizure, CD = cortical dysgenesis

tient had abnormal beta waves (diffuse excessive slowing without epileptiform changes). This was observed in a patient with generalized tonic-clonic seizures. This observation could represent EEG changes due to anti-epileptic drug. Delta waves were observed in six patients, but only one had focal delta waves localized to the left temporal region. This patient was a 21 year-old man and presented with simple partial seizures for the last 12 years.

MRI findings were abnormal in 17 out of 44 patients (38.6%); 14 of them had abnormal EEGs. The details of these 17 patients with

abnormal MRIs are as follows: 11 patients had partial seizures and six had generalized seizures (Table 1). Out of 19 patients with generalized seizures, an MRI was abnormal in six patients (35%) and all but one of these 6 patients had an abnormal EEG. Half the patients with partial seizures (11 out of 22) had an abnormal MRI. Of these 11 patients, four had simple partial seizures (SPS), three had complex partial seizures (CPS) and four had partial seizures with secondary generalization (PSSG) (Table 1).

Two of the patients with simple partial seizure had normal EEG findings. However, the

MRIs showed left hippocampal sclerosis and a left parietal glioma. The abnormalities observed were cerebral atrophy (5), hippocampal sclerosis (4), infarct/gliosis (3), cortical dysgenesis (2) and tumor (2). One patient had an arachnoid cyst at the right occipital region and was classified as having "other" findings. Overall, there were four patients with partial seizures where the seizure focus on the EEG correlated with the site of the abnormality detected on MRI. All three patients with unclassified epilepsy had normal MRIs.

Overall, only one out of six patients was found to have focal delta waves. The abnormalities detected in five out of six patients with delta waves (both focal and diffuse) were hippocampal sclerosis (2), temporal lobe infarct/ gliosis (2) and cortical dysgenesis of the temporal lobe (1). Of the five patients with cerebral atrophy, 4 (80%) had diffuse atrophy and one had focal symmetrical atrophy of the frontal lobe. Three out of these 5 patients had a history of seizures for 10 years or more.

Four patients had hippocampal sclerosis and only one of them had a normal EEG. All of them had partial seizures. One of the patients with hippocampal sclerosis presented clinically with complex partial seizures and had generalized epileptiform changes as well as diffuse background slowing (delta waves) on EEG.

The 2 tumor cases observed were glioma and a Dysembryonic Neuroectodermal Tumor (DNET). Both these patients were treated conservatively with follow-up MRI scan showing a similar appearance after two years. No histopathological diagnosis was ever made for these patients. The MRI findings of the first patient showed a small, well circumscribed enhancing white matter lesion at the left centrum semiovale with associated perilesional edema. This patient was a six-year old boy with simple partial seizures and a normal EEG. The second patient had a heterogeneously non-enhancing lobulated mass of the left temporal lobe, which was hypointense on T₁- weighted image, hyperintense on T₂-weighted image and not suppressed on FLAIR in keeping with Dysembryonic Neuroectodermal Tumor (DNET). This patient had partial seizures with secondary generalization and the EEG showed focal epileptiform changes at the left temporal region which coincided with the lesion detected on MRI. Overall, only 3 patients had localized interictal EEG abnormalities that corresponded to the lesions detected on MRI (concordance). The other two patients had focal left temporal delta waves and focal left hemisphere epileptiform changes with concordant lesions of the left temporal lobe, and left parietal lobe, respectively. Three of the 5 patients with a clinical diagnosis of temporal lobe epilepsy (TLE) had abnormal MRIs (1 had a left parietal infarct/gliosis and 2 had hippocampal sclerosis, one on the right and the other on the left side of the temporal lobe).

The parameters that were tested against the MRI findings included sex, type of seizures, frequency of seizures, duration of a history of seizures, general and local abnormalities, generalized seizures (tonic and tonic clonic), partial seizures (simple, complex and secondary generalized). Regarding association between clinical parameters and MRI findings, Univariate analysis (simple logistic regression) showed that there were no significant clinical factors associated with MRI findings (Table 2).

DISCUSSION

The focus of this study was to evaluate consenting patients who had abnormal EEGs who were referred for MRI. This was done because there are more centers able to do EEG on the East Coast of Malaysia then MRI facilities. The patients studied represented younger and older age groups. The mean age of 20.7 years and median of 18.5 years are in keeping with the fact that epilepsy is more common in the younger age group. Manonmani and Tan (1999) demonstrated a wide spectrum in terms

Clinical parameter	MRI fin	dings n (%)	Crude Odds ratio	Wald	p-value
·	Normal Abnormal		(95% CI)	statistic	
Sex					
Male	16(61.5)	10(38.5)	1	-	-
Female	11(61.1)	7(38.9)	1.02(0.30,3.50)	0.00	0.977
Seizure					
Generalized	13(65.0)	7(35.0)	1	-	-
Partial + unclassified	14(58.3)	10(41.7)	1.33 (0.39,4.52)	0.20	0.651
Seizure frequency					
1-6/week	11(64.7)	6(35.3)	1	-	-
2-4/week	10(71.4)	4(28.6)	0.73(0.16,3.38)	0.16	0.691
≤ 1/week	5(71.4)	2(28.6)	0.73(0.11,4.99)	0.10	0.751
Daily	1(16.7)	5(83.3)	9.17(0.86,97.69)	3.37	0.066
Seizure duration					
0-5 years	22(68.8)	10(31.3)	1	-	-
6-10 years	3(42.9)	4(57.1)	2.93(0.55,15.63)	1.59	0.207
> 10 years	2(40.0)	3(60.0)	3.30(0.48,22.94)	1.46	0.228

 Table 2

 Simple logistic regression analysis showing association between clinical parameters and MRI findings amongst patients with epilepsy.

Level of significance set at 0.05

of age of onset, with a minimum age of 3 months and maximum age of 77 years and a mean age of 18.7 years (Spencer, 1994). The difference between our study and that of Manonmani and Tan (1999) is that our study sample was slightly skewed towards the older age group. There were no young child below the age of 5 in our study because we excluded all children who were given sedation for EEG examination.

We found that partial seizures were more common than generalized seizures (50% vs 43.2%). These figures are in keeping with the findings of authors in Malaysia who did their studies in urban situations and found that generalized epilepsy accounted for 42.4% of the cases while 57.6% were due to localized epilepsy. Another study done in the West observed a higher percentage of partial seizures compared with generalized seizures (King *et al*,1998). Compared to a similar study of 593 patients in Kelantan done in 1993, 53.1% of patients were found to have generalized epilepsy and 16.7% had partial epilepsy (Win, 1993). This was a survey of EEGs done in a neurology laboratory without any radiological input. Both studies done in the state of Kelantan were conducted on epileptic patients in the same geographical location but with different findings over a 10 year period. The possible explanation is that in our study the number of patients was relatively small compared to that of Win's and the objectives of the two studies were different. The importance of neurological data collection and seizure classification have been emphasised by Sander et al (1990) who reclassified several generalized seizures into partial seizures in the light of additional information. They attributed the higher proportion of partial seizures in their study as due to thorough history and complete investigations (Sander et al, 1990).

In patients who presented with a first seizure, almost half the lesions detected on MRI were tumors (King *et al*, 1998). In another study that focused on adult patients with par-

Type of seizure	MRI fin	dings n (%)	Odds ratio Wa	ald statistic	p-value
	Normal	Abnormal	(95% CI)		·
Generalized seizure					
Tonic					
No	23(59.0)	16(41.0)	1	-	-
Yes	4(80.0)	1(20.0)	0.36(0.04,3.52)	0.77	0.379
Tonic clonic					
No	20(64.5)	11(35.5)	1	-	-
Yes	7(53.8)	6(46.2)	1.56(0.42,5.81)	0.44	0.509
Partial seizure					
Simple					
No	23(59.0)	16(41.0)	1	-	0.075
Yes	4(80.0)	1(20.0)	8.00(0.81,79.02)	3.17	
Complex					
No	20(58.8)	14(41.2)	1	-	
Yes	7(53.8)	3(30.0)	1.63(0.36,7.43)	0.40	0.526
Secondary generalized					
No	24(63.2)	14(36.8)	1	-	-
Yes	3(50.0)	3(50.0)	1.71(0.30,9.63)	0.37	0.542

Table 3 Simple logistic regression analysis showing association between type of seizure and MRI findings amongst patients with epilepsy

Level of significance was set at 0.05.

Factor	MRI findings n (%)		p-value	p-value		Area under
	Normal	Abnormal	·	Sensitivity (%)	Specificity (%)	the curve and 95% CI
EEG findings						
Normal	11	3	0.004	40.7	82.4	61.5
Abnormal	16	14				(44.7,78.4)
Epileptographic changes						
No	12	6	0.078	44.4	64.7	54.6
Yes	15	11				(37.0,72.1)

Table 4

^aMc Nemar's test applied; ^bCut-off p-value = 0.05

tial seizures (with or without secondary generalization), 109 out of 240 lesions detected on MRI were hippocampus asymmetry, while tumors only represented 16.7% of the total lesions (Li et al, 1994). Similarly, Lehericy et al (1997) found that 55% of 222 patients with temporal lobe epilepsy had hippocampal sclerosis (Lehericy et al, 1997). Hippocampal sclerosis is one of the most common lesions found in the brain of patients with intractable epilepsy. In our study, hippocampal sclerosis was observed in 23.5%.

Epilepsy is a functional disorder, although in some patients there may be a structural abnormality in the brain. Epileptic seizures can exist in patients with no structural lesions but many patients with structural lesions never have epileptic seizures. Epilepsy results from intermittent functional changes in neuronal activity that may or may not be related to disturbances in neuronal structural. Therefore, it is very difficult to correlate the electrical changes detected on EEG with structural abnormalities detected on MRI. Our findings in this study showed there was no significant association between EEG changes and MRI findings (p = 0.109). Localized EEG abnormalities did not have a significant correlation with MRI findings. These findings conform to the view that the identification of focal, structural, hippocampal, or neocortical lesions on brain MRI are not always indicative of the site of seizure origin. Holmes et al (1999) reported a series of 20 adults with medically intractable partial epilepsy, where high resolution MRI disclosed unilateral, focal, structural, hippocampal, or neocortical lesions as the only abnormality in each case (Holmes et al, 1999).

The parameters that are significantly associated with abnormal MRI are seizure frequency (daily seizure, p < 0.05) and seizure type (simple partial seizure, p < 0.05). In 2001, Salmenpera et al, studied the factors associated with hippocampal and amygdaloid damage in 241 patients with partial epilepsy using volumetric MRI. They found the significant risk factors for reduced hippocampal volume were high lifetime seizure number, history of complex febrile convulsions and age equal to or less than 5 years at the time of the first spontaneous seizure. In addition, there was a 16fold risk of hippocampal damage in patients with a lifetime seizure number of >1,461 compared to the patients with ≤13 seizures (Salmenpera et al, 2001). We had expected that the longer the history of seizures, the more likely the patient would have structural brain abnormalities. However, this was not demonstrated in our study. From our data, almost three guarters of the patients had a history of seizures for less than 5 years. Chronic epilepsy of more than ten years only made up 11.4% of the total cases. There was no significant correlation between epilepsy duration and abnormal MRI (p>0.05). This could be explained by the fact that even though patients may have chronic epilepsy, the lifetime seizure number might not be that high to have a significant correlation with an abnormal MRI. Furthermore, the number of patients in our study was small and this could contribute to these findings. A prospective study with prolonged follow-up of the patients may give us more information regarding an association between duration of epilepsy and abnormal MRI.

In terms of seizure type, even though complex partial seizures were twice as common as simple partial seizures, they were associated with a lower percentage of abnormal MRI (30%). MRI abnormalities were significantly more common in patients with simple partial seizures (80%). Chee et al (1993) found that simple partial seizures with motor manifestations were most frequently associated with focal CT scan abnormalities. Three guarters of patients with chronic and partial epilepsy had lesions detected on high resolution MRI and were thought to be of etiological significance (Berg et al, 2000; Cendes et al, 2000). They found that partial seizures and focal EEG abnormalities were the significant indicators for imaging abnormalities. They also found that abnormal motor findings were the strongest predictor of abnormal imaging.

In conclusion, EEG is a good modality to investigate epilepsy. Detection of structural brain abnormalities is a main aim of physicians. Our findings suggest that a history of daily seizures and simple partial seizures is sensitive enough for patients in general hospitals without MRI facilities to be referred for MRI. The difference in cost between a CT scan and an MRI of the brain at a government hospital of approximately 150 to 200 Ringgit Malaysia makes MRI a cost effective investigation to investigate epilepsy. Facilities without an MRI or neurologist may use online EEG transmission and centralized reading. Patients may then be referred by general physicians on the advice of neurologists to radiological centers with MRI facilities. This translates into more MRI investigations than CT scan imaging in these patients regardless of socioeconomic background.

REFERENCES

- Berg AT, Testa FM, Levy SR, Shinnar S. Neuroimaging in children with newly diagnosed epilepsy: A community-based study. *Pediatrics* 2000; 106: 527-32.
- Bronen RA, Fulbright RK, Kim JH, Spencer SS, Spencer DD. A systematic approach for interpreting MR images of the seizure patient. *Am J Roentgenol* 1997; 169: 241-7.
- Cendes F, Li LM, Watson C, Andermann F, Dubeau F, Arnold DL. Is ictal recording mandatory in temporal lobe epilepsy? Not when the interictal electroencephalogram and hippocampal atrophy coincide. *Arch Neurol* 2000; 57: 497-500.
- Chee MW, Lim SH, Tjia TL. Computed tomography in patients with recurrent seizures. *Ann Acad Med Singapore* 1993; 22 (3 suppl): 431-4.
- Commission on Classification and Terminology of the International League Against Epilepsy. Proposal for revised classification of epilepsies and epileptic syndromes. *Epilepsia* 1989; 30: 389-99.
- Commission on Neuroimaging of the International League Against Epilepsy. Recommendations for neuroimaging of patients with epilepsy. *Epilepsia* 1997; 38: 1255-6.
- Doherty CP, Cole AJ. The requirement for ictal EEG recordings prior to temporal lobe epilepsy surgery. *Arch Neurol* 2001; 58: 678-80.
- Gibbs J, Appleton RE, Carty H, Beirne M, Acomb BA. Focal electroencephalographic abnormalities and computerised tomography findings in

children with seizures. *J Neurol Neurosurg Psychiatry*1993; 56: 369-71.

- Holmes MD, Wilensky AJ, Ojemann GA, Ojemann LM. Hippocampal or neocortical lesions on magnetic resonance imaging do not necessarily indicate site of ictal onsets in partial epilepsy. *Ann Neurol* 1999; 45: 461-5.
- King MA, Newton MR, Jackson GD, et al. Epileptology of the first-seizure presentation: a clinical, electroencephalographic, and magnetic resonance imaging study of 300 consecutive patients. *Lancet* 1998; 352: 1007-11.
- Lehericy S, Semah F, Hasboun D, *et al.* Temporal lobe epilepsy with varying severity: MRI study of 222 patients. *Neuroradiology* 1997; 39: 788-96.
- Li LM, Fish DR, Sisodiya SM, Shorvon SD, Alsanjari N, Stevens JM. High resolution magnetic resonance imaging in adults with partial or secondary generalized epilepsy attending a tertiary referral unit. *J Neurol Neurosurg Psychiatry*.1994; 59: 384-7.
- Manonmani V, Tan CT. A study of newly diagnosed epilepsy in Malaysia. *Singapore Med J* 1999; 40: 32-5.
- Puvanendran K. Epidemiology of epilepsy in Singapore. *Ann Acad Med Singapore* 1993; 22 (3 suppl): 489-92.
- Salmenpera T, Kalviainen R, Partanen K, Pitkanen A. Hippocampal and amygdaloid damage in partial epilepsy: a cross-sectional MRI study of 241 patients. *Epilepsy Res* 2001; 46: 69-82.
- Sander JW, Hart YM, Johnson AL, Shorvon SD. National General Practice Study of Epilepsy: newly diagnosed epileptic seizures in a general population. *Lancet* 1990; 336: 1267-71.
- Sirven JI. Classifying seizures and epilepsies: A synopsis. *Semin Neurol* 2002; 22: 237-46.
- Spencer SS. The relative contributions of MRI, SPECT, and PET imaging in epilepsy. *Epilepsia* 1994; 35 (suppl 6): S72-89.
- Win MN. The EEG and epilepsy in Kelantan-a hospital/laboratory-based study. *Med J Malaysia* 1993; 48: 153-9.