Original article

Correlation of clinical and imaging findings in difference stage of neurocysticercosis

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Background: The common clinical manifestations of neurocysticercosis (NCC) was determined by several important factors. There is no previous study showing a correlation between difference stage of NCC and clinical presentation. The propose of this study was to determine correlation between different stages of NCC and its clinical presentation.

Methods: A retrospective descriptive study was performed using medical records and picture archiving and communication system from October 14, 2004 to October 18, 2019 in patients who definite diagnosed NCC from computerized tomography (CT) or magnetic resonance imaging (MRI) of brain. Neuroimaging was reviewed by two observers. The correlation between stage of NCC and clinical presentation was analyzed independently using Chi-square test and regression analysis.

Results: Statistical analysis was performed on 28 patients; Vesicular stage was seen in 8 (28.6%) patients, colloidal vesicular stage was seen in 8 (28.6%) patients, granular nodular stage was seen in 10 (35.7%) patients, nodular calcified stage was seen in 11 (39.3%) patients. Most patients, the lesions were located in the cerebral hemisphere 25 (89.3%) patients. Perileisonal edema was seen in 16 (57.1%) patients. There was no correlation between different stage of NCC and clinical presentation. But the location of NCC on cerebral hemisphere, perilesional edema and onset of clinical presentation were significant on seizure group ($P = 0.026$, $P = 0.010$ and $P = 0.018$, respectively). Location in cerebral hemisphere, perileisonal edema and onset of clinical presentation were imaging and clinical variables that was associated with seizure group on univariate analysis regression. However, on multivariate binary logistic regression, location in cerebral hemisphere, perilesional edema and onset of clinical presentation were not the factor to be independently associated with seizure group.

Conclusion: There is no correlation between different stages of NCC and its clinical presentations.

Keywords: Neurocysticercosis, stage, seizure.

Neurocysticercosis (NCC) is a helminthic infection of the central nervous system cuased by the cystic larval stage of the pork tapeworm Taenia solium.[1] It is the most common helminthic central nervous system infection worldwide. NCC is endemic or presumed to be endemic in many low-income countries. However, industrialised countries are not free from neurocysticercosis, and it contributes, sometimes considerably, to the burden of disease in patients with seizures or intracranial hypertension attending emergency room or accessing neurological or neurosurgical survices.[2] Neurocysticercosis is endemic in most Latin America countries, Sub-Saharan Africa, and large regions of Asia including the Indian subcontinent, most of Southeast Asia and China. The prevalence of neurocysticercosis is 0.2 - 0.6 per 100,000 inhibitants in some western states of the USA.[2] The prevalence in Thailand has no previous study. In 2007, Anantaphruti MT, *et al*.[3] reviewed cysticercosis and taeniasis in Thailand, infection rates by stool examination varied, depending on the place and time of examination, and reported as 0.2 - 7.0%. From 1947 to 2004, approximately 500 cases of human cysticercosis were reported in Thailand.

The first diagnostic criteria in 1996 for cysticercosis was proposed, based on the objective evaluation of clinical, radiological, immunological and epidemiological data. However, the criteria is very complex, and the specificity has been questioned.[4]

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Now, there is a consensus meeting in Peru, the working group has revised and simplified the criteria. This criteria will standardize the diagnosis and may aid in future clinical studies.[5]

The common clinical manifestations of neurocysticercosis are seizures, headaches and focal neurodeficit.[4] NCC is associated with a wide variety of clinical manifests. These are determined by several important factors including the burden of organisms, the location of encystment, the stage of cystercerci and the host response to the infection.[4]

However, there is no study showing a correlation between different stages of neurocysticercosis and the clinical presentations. The propose of this study was to determine correlation between different stages of neurocysticercosis and its clinical presentations.

Materials and methods

The Institutional Review Board (IRB) of the Faculty of Medicine, Chulalongkorn University has approved this study and waived informed consent (IRB no. 426/62).

Subjects

The retrospective analytic data was obtained by using medical records from hospital information system (HIS)) and picture archiving and communication system (PACS) from October 14, 2004 to October 18, 2019 in patients who were diagnosed with neurocysticercosis from computed tomography (CT) or magnetic resonance imaging (MRI) of the brain.The diagnosis of the patient was definite diagnosis by the criteria of revised diagnostic criteria and degrees of diagnostic certainty for neurocysticercosis in 2012.[5] The definite diagnosis was defined as: one absolute criterion, or two major neuroimaging criteria plus any clinical/exposure criteria, or one major and one confirmative neuroimaging criteria plus any clinical/exposure criteria, or one major neuroimaging criteria plus two clinical/exposure criteria (including at least one major clinical/exposure criterion), together with the exclusion of other pathologies producing similar neuroimaging findings.

The researchers started the data collection after the research project has been approved by the IRB. The process began with manual search for imaging of the cases by the hospital number (HN) whose definite diagnosis of neurocysticercosis from the Inpatient Department (IPD) and Outpatient Department (OPD) records.

The imagings were searched from picture archiving and communication system (PACS). Disagreement of imaging features should be concluded by consensus of one radiology resident and one neuroradiologist.

The inclusion criteria required the following: a patient who was definitely diagnosed with neurocysticercosis from IPD and OPD records, and underwent CT or MRI of the brain with contrast material injection between October 14, 2004 to October 18, 2019.

The exclusion criteria were incomplete clinical history and/or uncertain diagnosis, no imaging available, incomplete imaging (non-contrast study), the imaging from outside of the hospital and lesions that had previously received treatment (surgery, antihelmint drug or corticosteroid).

MRI protocol

The magnetic resonance imaging (MRI) included routine multiplanar brain scans on 1.5 Tesla [Philips Ingenia (Netherland), Siemen Aera (German)] and 3T [Philips Ingenia (Netherland), GE Discovery MR750w (USA), Siemen Skyra (German)] MR scanners. T1WI, T2WI, FLAIR, DWI, ADC and SWI, as a routinely conventional MRI brain protocol, were performed before administration of contrast medium (gadolinium-based contrast agent). The parameters for T1WI were TR/TE of 12/563.44, a field of view of 18 – 23 x 18 - 23 cm, thickness of 5 - 6 mm. The parameters for T2WI were TR/TE of 100/3941.64, a field of view of 18 – 23 x 18 - 23 cm, thickness of 5 - 6 mm. The parameters for SWI were TR/TE of 42.25/31.25, a field of view of 18 – 23 x 18 – 23 cm, thickness of 5 - 6 mm. The SWI was acquired in the axial plane. The parameters for DWI were TR/TE of 93.32/4000.50, a field of view of 18 – 23 x 18 - 23 cm^2, thickness of 5 mm. b value of 0 and 1000 s/mm^2 were noted.

The CT imaging included routine multiplanar brain scans [Siemens (German), Toshiba (Japan) and Phillips (Netherland)] with contrast medium administration (Iodinated-based contrast agent) were note. The slice thickness was 4 - 5 mm.

All data were referred to the PACS system to be interpreted by two observers:one neuroradiologist (W.S.) and one radiology resident (S.S.).

Imaging analysis

Qualitative assessment was observed. The observers were blinded to clinical history,

laboratory tests, and histopathologic information and independently evaluated the findings of stage of neurocysticercosis, number, location involvement, perilesional edema and hydrocephalus. The differences in opinion were solved by consensus.

The diagnosis of staging of neurocysticercosiswas accorded to Osborn's diagnostic neuroradiology[6] and review imaging spectrum of neurocysticercosis in radiology ofinfectious diseases.[7] The vesicular stage was defined as small cerebrospinal fluid-like cyst with thin wall and an eccentrically located scolex, no contrast enhancement of the cyst's wall, no surrounding tissue edema. The colloidal vesicular stage was defined as the density and signal intensity of the cystic fluid change from that of cerebrospinal fluid (CSF). The cystic wall is thicker. The scolex becomes ill-defined and finally shrinks in its size. Ring-like enhancement is seen. The surrounding tissue edema is obvious. The granular nodular stage was defined as small enhancing cyst or nodule, with mild surrounding edema and little mass effect. The nodular calcified stage was defined as small calcified nodule, no surrounding edema, better seen on CT.

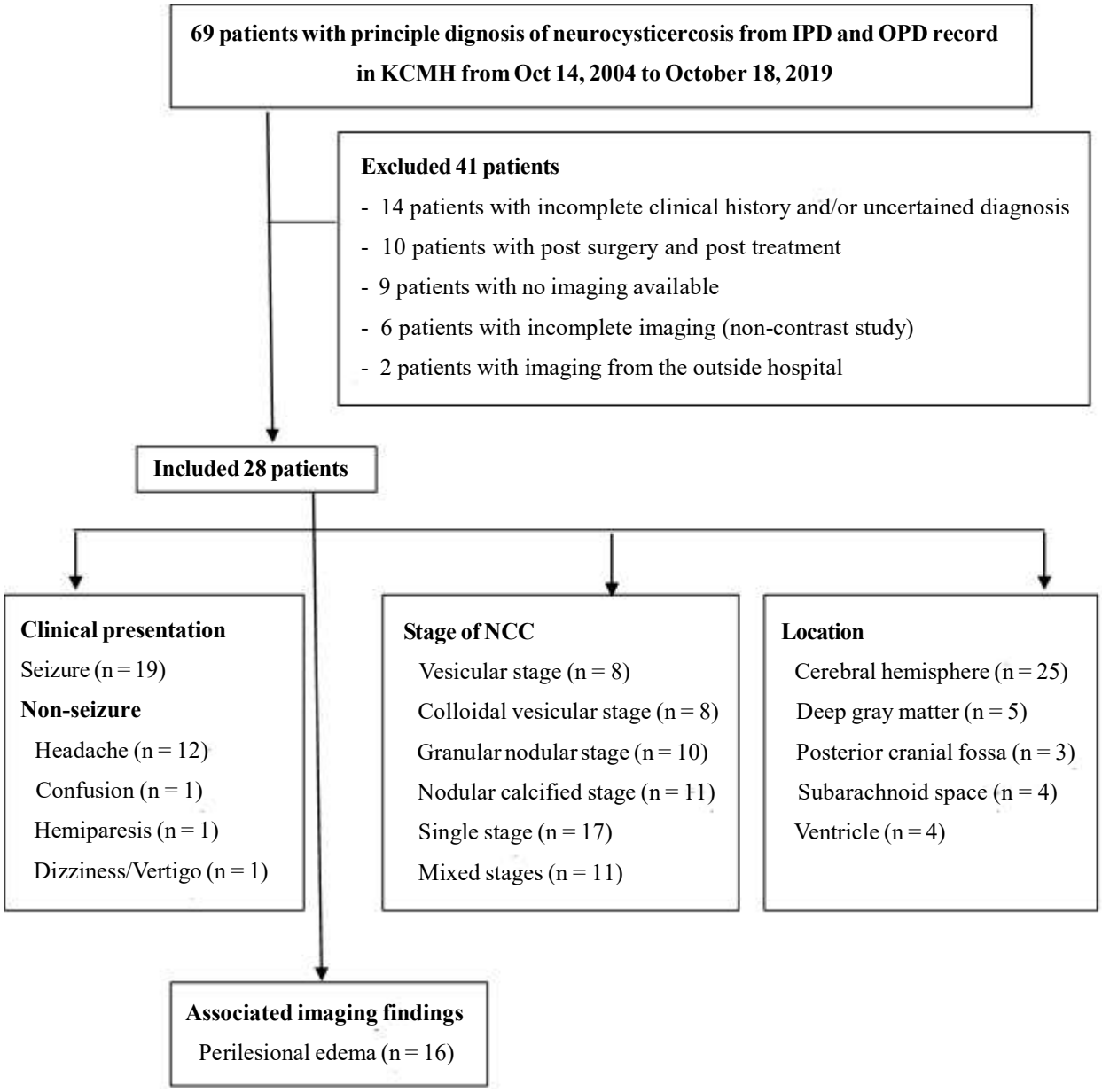

Figure 1. Flow diagram of the study.

Statistical analysis

Continuous variables are presented as mean ± standard deviation (SD) if normally distributed and otherwise as medians with interquartile range (IQR). Categorical variables showed as count and the percentage of the sample. The correlation between the stages of neurocysticercosis imaging and clinical presentation was analyzed independently using Chi-square test, univariate regression analysis and multivariate regression analysis. A P - value < 0.05 was considered statistically significant for all used tests. All statistical evaluations were performed with software (STATA Statistics, version 16).

Results

We initially included 69 consecutive patients with principle diagnosis of neurocysticercosis from IPD and OPD records at King Chulalongkorn Memorial Hospital (KCMH) from October 14, 2004 to October 18, 2019. Forty-one patients were excluded from the study due to the following conditions: 14 patients with incomplete clinical history and/or uncertain diagnosis, 10 patients with post prior surgery and post prior treatment, 9 patients with no imaging available, 6 patients with incomplete imaging (non-contrast study) and 2 patients with imaging from the outside hospital (Figure 1). Hence, 28 patients were finally evaluated.

Baseline characteristics

The baseline demographic, clinical and neuroimaging characteristics of 28 neurocysticercosis patients are shown in Table 1 and Figure 2. The mean age of the patients was 47.9 ± 17.2 years (range 17 - 82 years). The maximum age of the patient was 82-years-old and minimum age of the patient was 17-years-old. Twenty-three (82.4%) patients were males. Seven (25.0%) patients were residents in non-Bangkok location. Twelve (42.9%) patients were raw eaters. Nineteen (61.9%) patients were presentation with seizure. The median (IQR) time onsets of clinical before imaging investigation are 2 (1, 10.5). The mean time onset of clinical before imaging investigation was 11.1 ± 19.6 days (range 0 - 90 days).

Table 1. Baseline demographics and clinical manifestations in 28 patients with neurocysticercosis.

Baseline demographic data	**No. of patient** (%)
Age (years)	
Mean ± SD	47.9 (± 17.2)
Median (IQR)	48.5 (36.5, 59.5)
Age group	
< 20 years	1 (3.6)
20 - 40 years	9 (32.1)
41 - 60 years	11 (39.3)
61 - 80 years	6 (21.4)
> 80 years	1 (3.6)
Gender	
Male	23 (82.1)
Female	5 (17.9)
Residence	
Bangkok	7 (25.0)
Non-Bangkok	21 (75.0)
Diet	
Raw eating	12 (42.9)
Non raw eating	16 (57.1)
Smoking	10 (35.7)
Alcohol drinking	11 (39.3)
Clinicalmanifestation	
Seizure	19 (67.9)
Non-seizure	
Headache	12 (42.9)
Confusion	1 (3.6)
Hemiparesis	1 (3.6)
Dizziness/vertigo	1 (3.6)

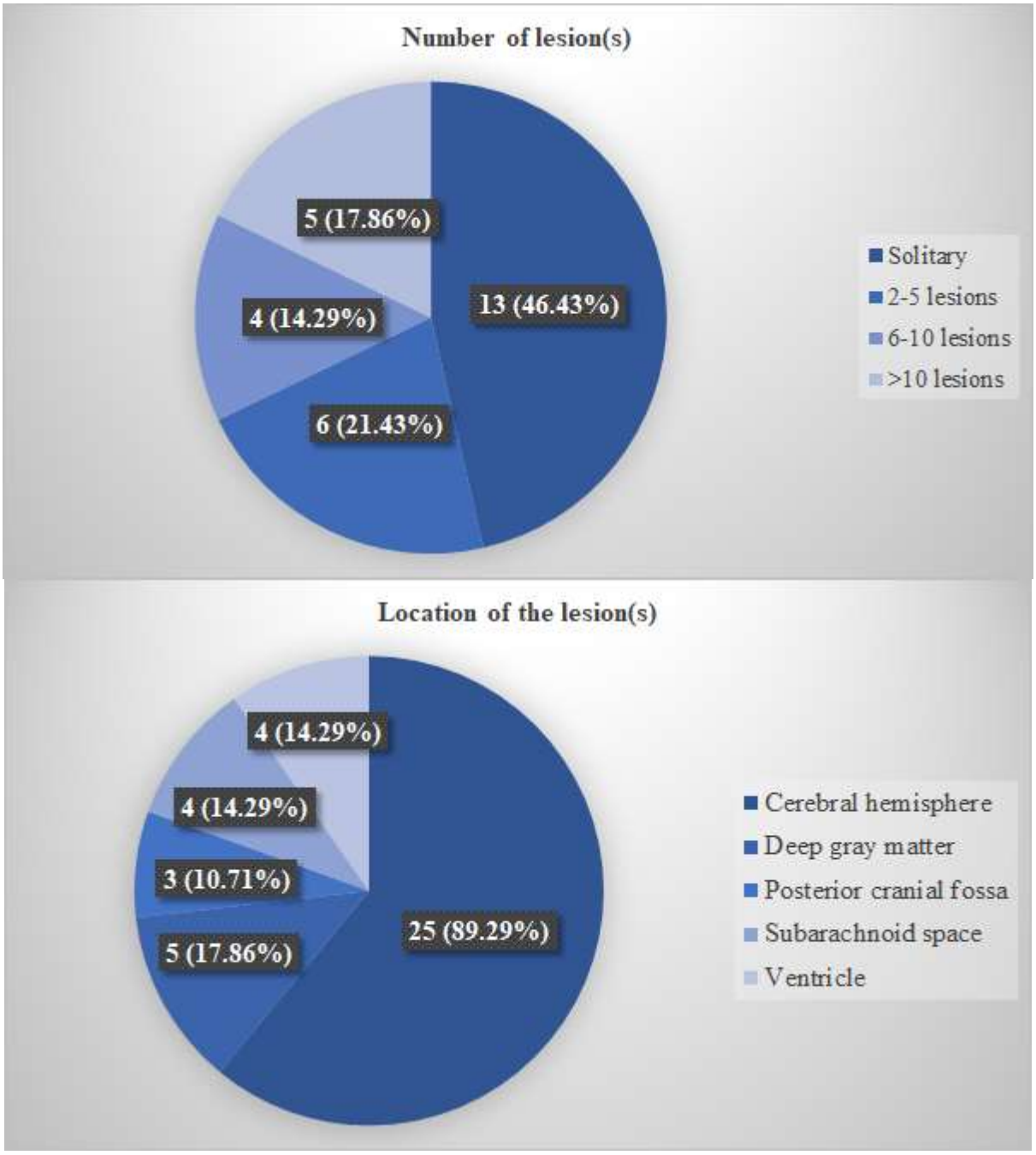

Figure 2. The imaging findings of number and locations of NCC in 28 patients with neurocysticercosis.

Neuroimaging

On MRI and CT imaging:vesicular stage was seen in 8 (28.6%) patients, colloidal vesicular stage was seen in 8 (28.6%) patients (Figure 3), granular nodular stage was seen in 10 (35.7%) patients, nodular calcified stage was seen in 11 (39.3%) patients. Parenchymal neurocysticercosis was seen in 20 (71.4%) patients. Non-parenchymal neurocysticercosis was seen in 8 (28.6%) patient, which 4 patients located in fourth ventricle and 4 patients located in subarachnoid Space (Figure 4). And also, single stage was seen in 17 (60.7%) patients, consisting of: 1 (5.9%) patient was vesicular stage, 3 (17.7%) patients were colloidal vesicular stage, 7 (41.2%) patients were granular nodular stage, 4 (23.5%) patients were nodular calcified stage, and 2 (11.8%) patients were located in the ventricle. Mixed stages were seen in 11 (39.3%).

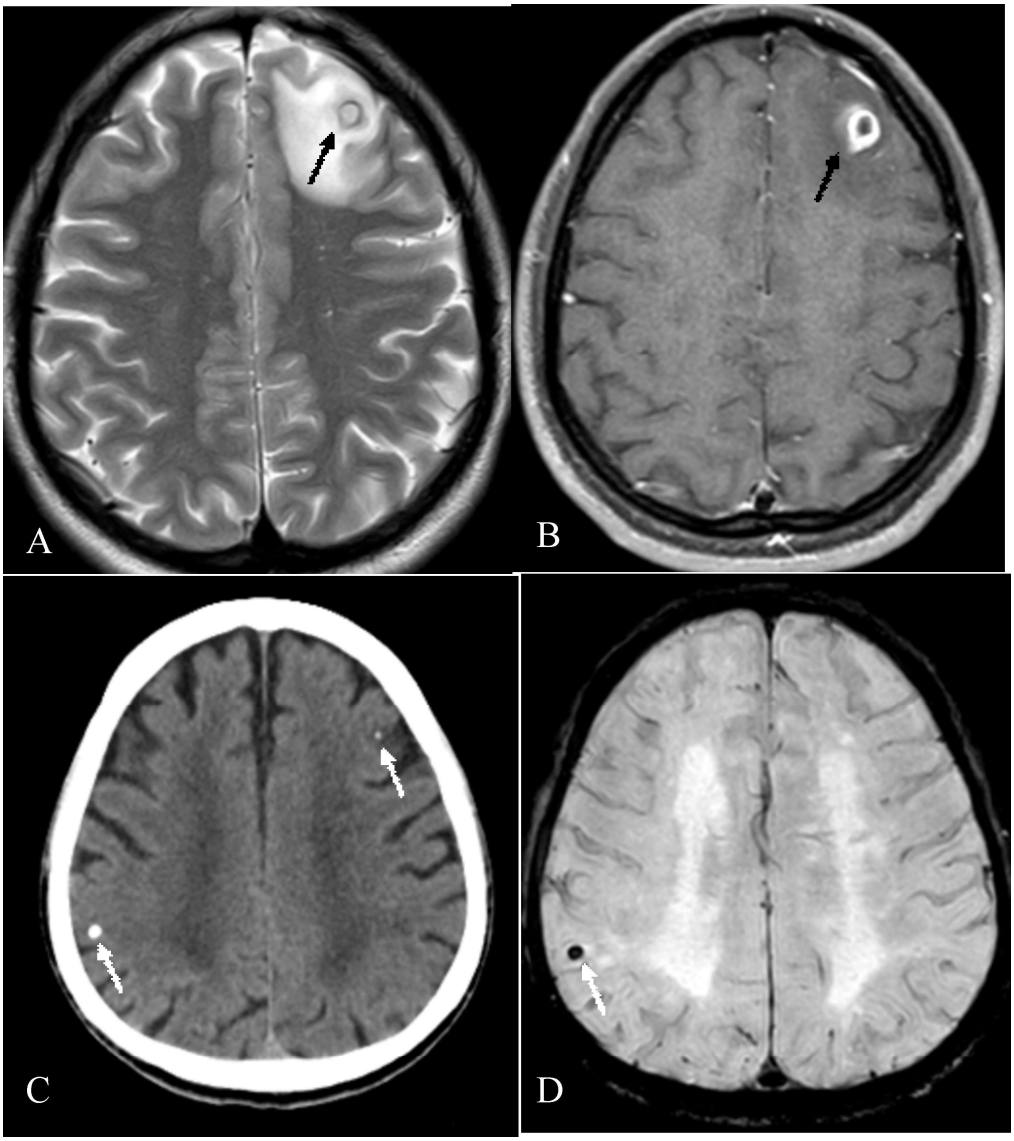

Figure 3. **(A)** and **(B)** images from a 27-year-old man presented with seizure, showing colloidal vesicular stage of neurocysticercosis in left frontal lobe (black arrow). The cyst wall was thickened, perilesional edema and ring-like enhancement, **(C)** and **(D)** Images from an 81-year-old man presented with seizure, showing nodular calcified stage of neurocysticercosis in right parietal lobe and left frontal lobe (white arrow) without perilesional edema. The lesions also show hyposignal intensity on GRE T2*WI, representing calcified nodule (white arrow).

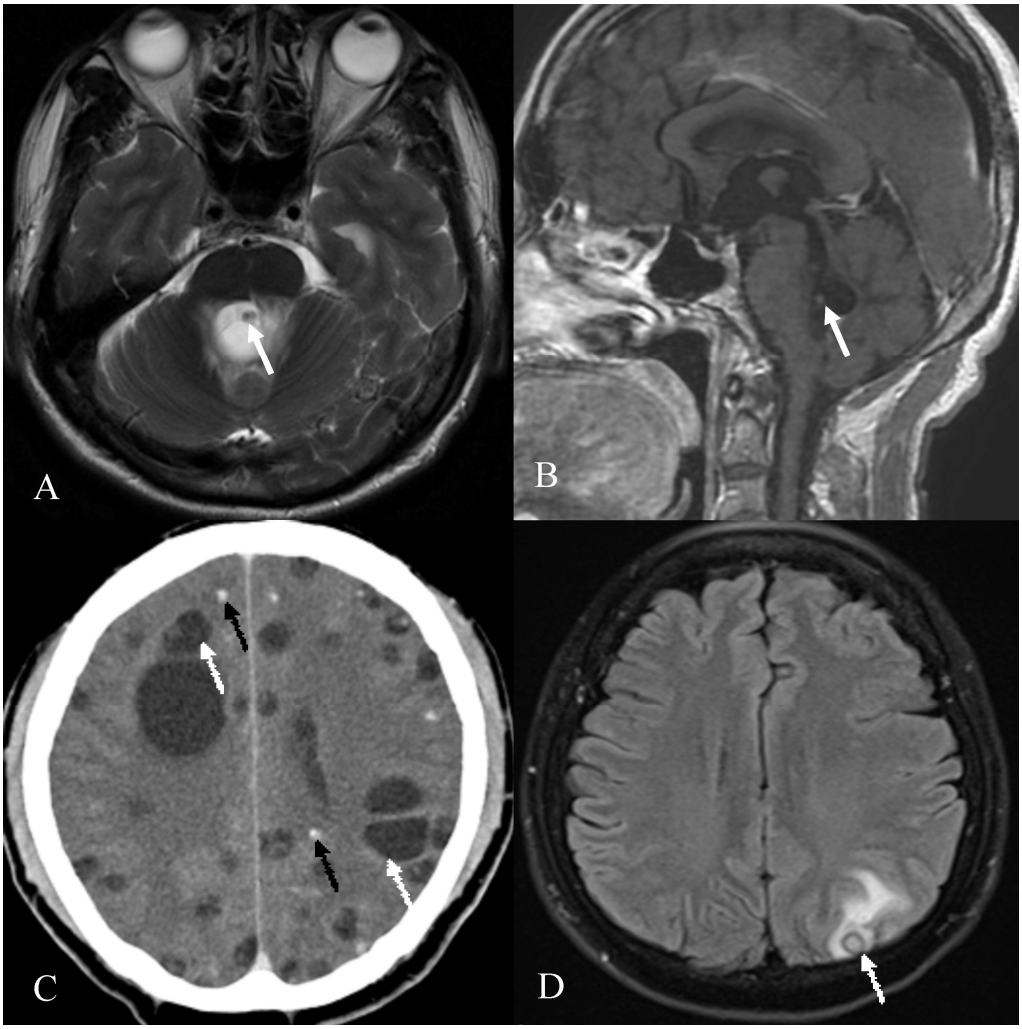

Figure 4. (A) and **(B)** images from a 53-year-old man presented with headache, showing of neurocysticercosis in the fourth ventricle. The cyst content appears similar to CSF. There is mild perilesional edema. No contrast enhancement after intravenous administration of contrast material is observed. The round structure within the cyst represents the scolex (white arrows). This patient was suboccipital craniotomy with cyst removal, pathology was cestode parasite with presence of scolex, compatible with cysticercosis. **(C)** images from a 40-year-old man presented with headache showing multistage of neurocysticecosis (vesicular stage (white arrow) and nodular calcified tage (black arrow)). **(D)** image from a 58-year-old man presented with dizziness showing colloidal vesicular stage of neurocysticercosis in left parieto-occipital lobe (white arrow) with perilesional edema.

Clinical and imaging findings correlation in difference stage of neurocysticercosis on univariate analysis

On Chi-square test showing comparison of baseline demographic, staging and imaging findings amongst the "seizure" and "non-seizure" groups was shown on Table 2. The location of neurocysticercosis located in cerebral hemisphere and present of perilesional edema were significant on seizure group ($P = 0.026$ and $P = 0.010$, respectively). The stage of nuerocysticercosis was not statistically significant as compared with seizure and non-seizure group. The onset of clinical presentation also showed significance between seizure and non-seizure group ($P = 0.018$). The clinical presentation with seizure showed shorter onset of time than the non-seizure group.

Table 2. Comparison of baseline demographic, staging and imaging findings amongst the seizure and non-seizure groups.

Variables	Seizure group (n = 19)	Non-seizure group (n = 9)	*P* - value
Age (mean years)	44.7	54.6	0.305
Gender			
Male	15	8	1.000
Female	4	1	1.000
Residence			
Bangkok	5	2	1.000
Non-Bangkok	14	7	1.000
Raw eating	8	4	1.000
Onset of presentation (mean days)	2.7	28.9	0.018
Stage			
Vescular stage	5	3	1.000
Colloidal vesicular stage	7	1	0.214
Granular nodular stage	9	1	0.098
Nodular calcified stage	7	4	1.000
Single stage	10	7	0.214
Mixed stages	9	2	0.214
Imaging findings			
Located in cerebralhemisphere	19	6	0.026
Located in deep gray matter	4	1	1.000
Located in posterior cranial fossa	1	2	0.234
Located in subarachnoid space	3	1	1.000
Located in ventricle	1	3	0.084
Perileisonal edema	14	2	0.01
Number of the lesion (s)			
Solitary lesion	7	6	0.64
2 - 5 lesions	6	0	0.64
6 - 10 lesions	3	1	0.64
< 10 lesions	3	2	0.64

On univariate analysis regression showing variables found to be independently associated with seizure group. Location in cerebral hemisphere, perilesional edema and onset of clinical presentation were imaging and clinical variables that was associated with seizure group (P = 0.014, odds ratio = 3.17, 95% confidence interval 1.26 - 7.93, P = 0.017, odds ratio = 9.80, 95% confidence interval 1.50 - 63.85 and P = 0.019, odd ratio = 0.84, 95% confidence interval 0.73 - 0.97, respectively).

On multivariate binary logistic regression, location of neurocysticercosis in cerebral hemisphere, perilesional edema and onset of clinical presentation were not the factor to be independently associated with seizure group (P = 0.657, odds ratio = 0.7, 95% confidence interval 0.11 - 3.98, P = 0.128, odds ratio = 7.9, 95% confidence interval 0.55 - 113.18 and P = 0.094, odd ratio = 0.85, 95% confidence interval 0.71 - 1.02, respectively).

Discussion

Cysticercosis is a parasitic disease caused by larva of *Taenia solium*, which parasitizes in human body as the intermediate host after ingesting of pork tapeworm eggs. Cysticercosis of the CNS (neurocysticercosis) is the most common parasitic infection in the CNS with predilection sites of parenchyma, meninges or ventricles.[8] Neurocysticercosis was commonly seen in developing countries. Its incidence in Thailand is uncertain, but trends to be increasing due to the raw pork eating habits and the immigration of the population.

Clinical symptoms of neurocysticercosis are various, in this study we found that the clinical presentation including seizure, headache, confusion, hemiparesis and vertigo. The most frequency of the clinical presentation from this study was seizure, 19 patients (67.9%). The literature consistently supports that seizures are the most common symptom of

NC, occurring in 70.0% to 90.0% of symptomatic patients.[9] However, there is no uniformity in the reported distribution of the types of seizure in patients with NCC.

Imaging examination was an important diagnostic means of neurocysticercosis. In our study, 28 patients corresponding to definitive diagnosis were included; vesicular stagewas seen in 8 patients (28.6%); colloidal vesicular stage was seen in 8 patients (28.6%); granular nodular stage was seen in 10 patients (35.7%); and, nodular calcified stage was seen in 11 patients (39.3%). According to the location in the CNS, neurocysticercosis can be divided into parenchymal cysticerci, subarachnoidal cysticerci and ventricular cysticerci. In some publications, the subarachnoid space is the most common site, followed by the parenchyma.[8] However, in our series, cysticerci located in the cerebral parenchyma were the most frequent with a percentage of 89.3%, followed by deep gray matter 17.9%, followed by subarachnoid and ventricle of 14.3%, and followed by posterior cranial fossa 10.7%.

There are four patients that the neurocysticercosis located in the ventricle. Two of four patients had single lesion and single stage; both patients presented with headache and the imaging finding was shown mild obstructive hydrocephalus that could be described by the common pathophysiology of obstructive hydrocephalus. Also, 2 of 4 patients had multiple lesions of multiple stages, which combined with cerebral hemisphere location. All two of four patients presented with seizure.

Seizures may occur at any evolutionary stage of the parasite, but neurocysticercosis-related seizures may be more common among children than adults.[10] But in this study the age was not the predisposing factor to seizure occurrence. And we also found the stage of neurocysticercosis was not associated with the seizure symptom.

Some studies show that clinical inconsistencies in the causal link between seizure and neurocysticercosis lesions.[11] For example, patients with seizures may have only one calcified lesion and patients with multiple cysts or calcifications may have no seizures. We believed that there may be another factor that can cause seizure, such as location of the lesion or other associated findings.

In this retrospective study, we found the location of neurocysticercosis in the cerebral hemisphere and perilesional edema were significant in the seizure group in univariate analysis regression. However, it is not clear whether this edema is the cause or the consequence of seizure.[12] Degree of blood brain barrier permeability may also play a role in epileptogenesis of calcified lesions. A study designed to evaluate epileptogenesis of calcified lesions using dynamic contrast-enhanced MRI reported that median values of the rate transfer constant and leakage volume were higher in symptomatic patients than in asymptomatic patients, indicating a higher degree of blood brain barrier permeability in symptomatic individuals.[13] So, we may conclude that the perilesional of the lesion may associated with the seizure.

Epilepsy is more frequently seen in patients with cysticerci located in the brain parenchyma, including deep cortical sulci,[14] which is the same direction of this study; the neurocysticercosis was located in the cerebral hemisphere of 25 patients (89.3%), deep gray matter 5 patients (17.9%) and posterior cranial fossa 3 patients (10.7%).

All the stages of involution may cause seizures; apparently the mechanisms involved in the generation of epileptogenicity are different. During the first stage, the seizures are probably related to compression of the surrounding brain parenchyma and inflammatory reaction; colloid cysts are associated with acute inflammatory changes. In contrast, granular and calcified cysticerci cause seizures most likely owing to astrocytic gliosis surrounding the lesions.[15]

This study is the first to determine the correlation between different stages of neurocysticercosis and clinical presentations. Although Chi-square test showed no correlation between the stage of neurocysticercosis and the seizure group. Although the colloidal vesicular stage and granular nodular stage showed no significance in the seizure group, from the raw data, it showed that 7 from 8 patients in colloidal vesicular stage and 9 from 10 patients were presented with seizure. Further study with a greater population is, therefore, suggested.

On the other hands, on univariate analysis regression showed the location in the cerebral hemisphere and perilesional edema were imaging variables associated with seizure group. Previous studies have also found that perilesional edema occurs frequently and it is associated with episodic seizure activity in calcified neurocysticercosis [16] and presence of perilesional edema and scolex can similarly predict seizure recurrence.[17] In a previous study,

neuroimaging findings like presence of perilesional edema, demonstration of scolex, and larger size of the lesion were found associated with seizure.[16] The perilesional edema most likely represents hosts response to newly released parasitic antigens.[18] Therefore, the finding as perilesional edema can be a potential target of treatment in these patients using anti-inflammatory agents like corticosteroids.[19]

On univariate analysis regression showed the onset of clinical presentation which was also clinical variable associated with seizure group. Patients who presented with seizure trend to have early investigation with CT or MRI imaging than those patients with non-seizure group.

Our study had some limitations, however. First, it was conducted with a limited study population which may account for a type II error. Single-center population was represented an acceptable bias. Due to this study was the retrospective study, it was the selection bias and limitation of the quality of the data. A multicenter study designed in the large group of population was recommended. For further research, a study in larger populations are necessary for more information.

Conclusion

Thus, we concluded that there was no correlation between difference stage of neurocysticercosis and clinical presentation. The location in cerebral hemisphere and perilesional edema were imaging variables that was associated with seizure group on univariate analysis regression but not on multivariate binary logistic regression. The onset of clinical presentation was a clinical variable associated with seizure group on univariate analysis regression but not on multivariate binary logistic regression.

Conflict of interest

The authors, hereby, declare no conflict of interest.

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