Original Article

Cranial Computed Tomographic Findings in Transient Ischemic Attack Patients

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Background: Transient ischemic attack (TIA) is an important risk factor for ischemic stroke, with temporary disruption of cerebral blood flow. Despite TIA was only short-time neurological dysfunction, it might cause following permanent brain damage. Magnetic resonance imaging (MRI) is the modality of choice for assess cerebral imaging in TIA, but widely unavailable and limited by contraindication, hence cranial computed tomography (CT) scan has a role in diagnosis of TIA patients.

Objective: The purpose of the present study was to describe CT appearance, the prevalence of negative CT findings for acute pathology and review the clinical information of patients whom diagnosed TIA for characterize the associated factor.

Materials and Methods: Retrospectively reviewed medical records and CT findings with TIA were recruited. Correlation between clinical data and CT appearance was done.

Results: 231 patients were analyzed. CT scan discovered negative acute pathology in 97.40% of cases. 2.16% had intracranial hemorrhages and possibly acute infarct in 0.43%. The risk of acute ischemic stroke within 90 days after TIA is 3.46%. Significant differences of the risk factor in older TIA patients are hypertension (p = 0.042), diabetes mellitus (p = 0.008), alcoholic drinking (p<0.001), current smoking (p = 0.002), and symptom presentation of muscular weakness (p = 0.002) that associated with TIA event.

Conclusion: The prevalence of negative CT findings for acute pathology in TIA is quite high still remained uncertain etiology, which should be performed additional MRI for recognizing acute and/or small lesions.

Keywords: Cerebral tomography, Transient ischemic attack (TIA)

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Transient ischemic attack (TIA) is currently tissue-base defined as a transient episode of neurological dysfunction initiated by focal brain, spinal cord, or retinal ischemia, without evidence of acute cerebral infarction. In the US, affirmative a tissue-based definition of TIA wound abated appraisal of annual incidence of TIA and accrue appraisal of annual incidence of stroke. In the part, the TIA was traditionally determined as a concise episode of focal neurological deficit caused by ischemia, with instantaneous onset and speedy resolution within 24 hours, following the 2002s classified TIA as a brief episode of neurological dysfunction occurred by focal brain or retinal ischemia, with clinical symptoms naturally lasting less than an hour, and absent verification of acute infarction[1-4].

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Annually, approximately 200,000 to 500,000 patients are diagnosed as TIA[5]. The prevalence of TIA in US adults was 2.3% which translates into 5 million patients; the population incidence rate is quite high, up to 83 per 100,000 for all age groups. The mortality rate was 12.3% at one year after TIA[6,7]. Besides incidence rate of TIA for the Republic of Croatia is 100.55 per 100,000, higher than in the coastal part of Croatia 82%[8]. In Thailand, they are unavailable regarding the incidence and prevalence of TIA for the federal report; however statistics are accessible for individual hospitals[9]. TIA illustrates an actual medical emergency and frequently an admonishing of an impending stroke, the risk of stroke is highest in the first 48 hours after TIA. It is crucially important to be aware of this chance for primary stroke prevention arrangement[10]. 10 to 15% of acute ischemic stroke patients had previous TIA and the risk of consequent stroke after TIA is 10.5% within 90 days[11,12]. In the US, approximately 795,000 patients endure a new or recurrent stroke annually; stroke is a principal cause of mortality and morbidity worldwide[13] and is the first major cause of death and long-term disability in Thailand[14].

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Computed tomography (CT) scan has a role in assessing patients with transient urological symptoms. In patients with TIA, CT would be achieved to identify dense calcifications, subarachnoid hemorrhage, intracranial hemorrhage, subdural hematoma and conditions that mimic TIA such as tumor, abscess or other masses; nevertheless CT is not sensitive for evaluating abnormality in brainstem and cerebellum[15]. CT displays ischemic infarction in 4 to 13% of TIA patients[16], that low sensitivity in detecting acute and small infarcts. Diffusion-weighted imaging (DWI)-Magnetic resonance imaging (MRI) confirmed ischemic abnormality in almost half of clinically suggestive TIA patients, which a positive DWI lesion increases with symptom duration[17]. MRI is advantageous more than CT, particularly if executed early with using diffusion-weighted imaging/fluid-attenuated inversion recovery (DWI/FLAIR), and has high sensitivity for detection of minute ischemic lesions and hyperacute lesions, which may be invisible on CT[18].

TIA is a common problem in all hospitals in Thailand, but most hospitals including essentially provincial hospitals have limited access to MRI, which is the modality of choice for TIA or stroke patients. At our institution, Chumphae Hospital, Khon Kaen Province, is a regional hospital and limited access to MRI, however, CT is available and initial service as of October 1, 2011. We found that the number of patients diagnosed as TIA or stroke was relatively high, but MRI has not performed to determine the associated etiology. In Thailand, there is no study of CT findings in patients with TIA, which is a major risk factor for acute and/or recurrent stroke. The prevalence of negative CT findings in these patients has not been reported. Some patients may require further investigation such as MRI or other specialized diagnostic radiology for definite diagnose and the cause of TIA. Furthermore, we studied the clinical and imaging data of all patients who diagnosed TIA for determine the associated etiology and improve practical TIA guideline.

Objective

General objective

To describe the CT findings of TIA patients in Chumphae Hospital, Khon Kaen Province, Thailand for the period of October 2011 to April 2017.

Specific objective

1) To determine prevalence of negative CT findings for acute pathology in patients whom diagnosed as TIA.

2) To definite baseline characteristics data included clinical information, risk factor and medical treatment of patients with a diagnosis of TIA.

Materials and Methods

Subjects

This was a single-center retrospective data collection and described study of transient ischemic attack (TIA) patients. All information were derived and sustained in regard to protect the confidentiality of the patients and approved by the Khon Khaen University Ethics Committee for Human Research (KKUEC), based on the rule of the declaration of Helsinki. The medical records and CT images of consecutive TIA patients who diagnosed as having TIA at Chumphae Hospital, Khon Kaen Province from October 2011 to April 2017 were reviewed.

Inclusion criteria for the study as the following; 1) Age ≥18 years, 2) TIA verified by clinician at the emergency room and/or admission ward. Exclusion criteria for the study is unavailable CT. A total of 723 patients were diagnosed as having TIA. Finally, after CT scan there were 231 TIA patients in who enrolled into the study.

Baseline characteristic data was the following: 1) Age and gender; 2) Risk factors: hypertension (HT), diabetes mellitus (DM), atrial fibrillation (AF), valvular heart disease (VHD), coronary artery disease (CAD), previous cerebrovascular accident (CVA), previous TIA, history of ischemic heart disease (IHD) or myocardial infarction (MI), current smoking and alcoholic drinking; 3) Medical treatment: aspirin, cilostazol, clopidogrel, warfarin, unfractionated heparin (UFH), low-molecular-weight heparin (LMWH), statin and fibrate; 4) Clinical information: duration of symptoms, blood pressure, symptom presentation, clinical severity (calculated ABCD2 score: A = age, B = blood pressure, C = clinical features, D = duration of symptoms and diabetes mellitus; baseline NIH Stroke Scale: NIHSS and baseline modified Rankin Scale: mRS); functional outcome (NIHSS and mRs at discharge); hospital length, 90-days acute stroke after TIA were assembled.

Imaging techniques

CT studies available for 231 patients which performed in Chumphae Hospital. The CT was performed with a 16 detector device (Alexion™, Toshiba, Chennai Sea, China) for 5 mm axial slice thickness (120kV, 150 mA), and practiced in horizontal (or transaxial) planes parallel to the orbitomeatal line, from base of skull to vertex.

Imaging analysis

An experienced neuroradiologist and a general radiologist reviewed the CT images separately and achieved a determination on the definite interpretations by consensus. A neuroradiologist had fifteen years total experience and a general radiologist had six years total experience.

Firstly, readers were blinded to symptom presentation of TIA and to any other clinical information. Consequently, with learning of TIA radiographic features, the following lists were recorded: 1) Appearance of an acute pathology (such as acute infarct or intracranial hemorrhage) and negative acute pathology (normal study, isolated brain atrophy, only chronic infarct, or calcified granulomatous lesions), 2) Sites of infarct base on vascular territories (anterior or posterior circulation), 3) Locations of acute infarcts and chronic infarcts, 4) Size of infarct: lacunar infarct (diameter <1.5 cm), medium-sized infarct (diameter 1.5 to 3 cm) and large infarct (diameter >3 cm)[19]. The location of infarcts was proposed as the following: centrum semiovale, corona radiata, caudate nucleus, lentiform nucleus, internal capsule, external

capsule, insular cortex, thalamus, frontal lobe, parietal lobe, occipital lobe, temporal lobe, cerebellum and the others.

Statistical analysis

The data analyzed were performed using the STATA program for windows, version 7.0. For descriptive analysis of patient's information including baseline characteristic data and CT findings were analyzed using descriptive statistics for frequency and percentages (%), with mean (standard deviation) for continuous data. The prevalence of negative CT findings for acute pathology of TIA was also defined. Proportion test was used for compare the proportion of two groups. If expected value more than 5 or 20%, using Pearson's Chi-square test (χ^2-test) or not, we analyze by Fisher's exact test as appropriate. The t-test was used for compare average statistics.

Results

723 patients were diagnosed at the emergency room and/or admission ward as having TIA from 1 October 2011 to 30 April 2017. Of these, 492 cases were excluded because they had not received a CT scan or had incomplete data. There are 231 patients, enrolled into the study. 135 of these were males (58.44%) and 96 (41.56%) were females, with the male-to-female ratio of 1.4: 1. The mean age was 63.86±13.14 years (range 24 to 95 years). Most are above 60 years old (61.04%). A total of 18 (7.79%) of 231 patients were 45 years of age or younger, and defined as the younger group. Through the younger group, 12 (5.19%) were males, 6 (2.6%) were females, with a mean age of 37.83±6.42 years, and the male: female ratio was 2: 1. The older group comprised of 79 (34.20%) males, 62 (26.84%) females, with the male: female ratio was 4: 3. The mean age of this group was 66.06±11.04 years.

The majority of patients (86.58%) had at least one risk factor. The five most common risk factors are hypertension (56.14%), diabetes mellitus (34.35%), dyslipidemia (32.9%), current smoking (12.61%) and alcoholic drinking (12.12%), although patients without risk factors were recognized in 13.42% cases.

The mean time from symptom onset to hospital for all patients (with available data) was 807 minutes (range 5 minutes to 14 days). 43 (18.62%) of all 231 patients reached at hospital within 60 minutes. 61.3% of patients presented with unilateral hemiparesis, following by speech impairment (47.83%), dizziness (13.42%), facial palsy (12.55%) and paresthesia (11.74%). Among 231 patients whom diagnosed with TIA, 8 (3.46%) acute ischemic strokes happened within 90 days after TIA. In all, 71.43% treated with lipid-lowing medication (statin), 70.43% treated with an antiplatelet agent (aspirin). Baseline characteristics are displayed in Table 1.

Appraisement of CT images disclosed negative acute pathology in 225 (97.40%) cases. Normal study and isolated brain atrophy is found in 145 (62.77%) of all 231 patients. 5 (2.16%) patients had subacute infarcts. There are 5 (3.31%) with intracerebral hemorrhage and 1 acute infarct in results. 14 (20%) of 70 patients had only single infarcts.

Table 1. Baseline characteristic parameters

Variables	n (%)
Male	135 (58.44)
Age group (years)	
18 to 45	18 (7.79)
46 to 60	72 (31.17)
>60	141 (61.04)
Risk factors	
Hypertension	128 (56.14)
Diabetes mellitus	79 (34.35)
Dyslipidemia	76 (32.9)
Current smoking	29 (12.61)
Alcoholic drinking	28 (12.12)
Atrial fibrillation	25 (10.82)
Previous CVA	16 (6.93)
History of IHD or MI	15 (6.49)
Coronary artery disease	12 (5.19)
Previous TIA	11 (4.76)
Valvular heart disease	5 (2.16)
None	31 (13.42)
Medications	
Aspirin	165 (71.43)
Cilostazol	162 (70.43)
Clopidogrel	10 (4.33)
Warfarin	9 (3.90)
Statin	8 (3.46)
UFH/LMWH/fibrate	0 (0)
None	46 (19.91)
Clinical information	
Duration of symptom (onset to door time)	
<10 mins	2 (0.87)
10 to 59 mins	41 (17.75)
≥60 mins	188 (81.39)
Baseline SBP ≥140 or baseline DBP ≥90	156 (67.53)
Symptom presentation	
Muscular weakness	141 (61.3)
Right unilateral weakness	67 (29.13)
Left unilateral weakness	62 (26.96)
Bilateral weakness	12 (5.22)
Speech impairment	110 (47.83)
Dizziness	31 (13.42)
Facial palsy	29 (12.55)
Numbness/paresthesia	27 (11.74)
Headache	6 (2.6)
Seizure	1 (0.43)
Nausea/vomiting	1 (0.43)
Complication	1 (0.43)
Death	0 (0)
90 days acute stroke after TIA	8 (3.46)
Clinical severity (mean ± SD, range)	
ABCD2 score	4.89±1.25, 1 to 7
Baseline NIHSS	2.39±4.16, 0 to 23
Baseline mRs	1.51±1.17, 0 to 5
Functional outcome (mean ± SD, range)	
NIHSS at discharge	0.61±1.26, 0 to 7
mRs at discharge	0.86±1.08, 0 to 41.4
Hospital length, days (mean ± SD, range)	7±7.53, 1 to 9

CVA = cerebrovascular accident; IHD = ischemic heart disease; MI = myocardial infarction; TIA = transient ischemic attack; UFH = unfractionated heparin; LMWH = low-molecular-weight heparin; SBP = systolic blood pressure; DBP = diastolic blood pressure; NIHSS = NIH Stroke Scale; mRS = modified Rankin Scale

Based on vascular territories, infarcts were classified as localized in the anterior circulation 63 (85.33%) cases, 3 (4%) posterior circulation and only 8 (10.97%) cases confined in both anterior and posterior circulations. Total of subacute infarct patients had large infarcts. Temporal lobe (48.46%) is the most common area affected with subacute large cerebral infarcts. 34 (49.18%) of 65 chronic infarct patients had only lacunar infarct, 16 (21.33%) medium-sized infarct only and 7 (10.14%) had only large infarct. The most common site of chronic medium infarct is corona radiata (30.96%). Lentiform nucleus (25%), corona radiata (20%) and internal capsule (16.25%) are the three most common locations of acute lacunar infarct. CT appearance is demonstrated on Table 2 to 4.

The distribution of baseline characteristics were similar between acute pathology and negative acute pathology subgroups, except statin treatment (p = 0.011) and no mediation treatment (p = 0.012) which is exhibited in Table 5. The distribution of baseline characteristics and CT findings resemble between the younger and the older subgroups, except mean age (p<0.001), risk factors of hypertension (p = 0.042), diabetes mellitus (p = 0.008), alcoholic drinking (p<0.001), current smoking (p = 0.002) and symptom presentation by muscular weakness (p = 0.002), which being more common in the older group; revealed in Table 6.

Table 2. Computed tomography (CT) appearance

Computed tomography appearance	n (%)
Acute pathology	6 (2.60)
Intracranial hemorrhage	5 (2.16)
Acute infarct	1 (0.43)
Negative acute pathology	225 (97.40)
Normal	78 (33.77)
Isolated brain atrophy	67 (29.00)
Only chronic infarction	65 (28.14)
Calcified granulomatous lesions	6 (2.60)
White matter disease	4 (1.73)
Subacute infarct	5 (2.16)

Discussion

TIA is an often occurrence and a significant proportion of patients have a high risk of renewal; furthermore TIA is a robust predictor of subsequent stroke and death[20]. Recurrent TIA possibly frivolous and cease spontaneously, however it showed significant increase risk of stroke up to 9.3-fold[21]. Precise and quick specification of high-risk TIA patient is important to decide treatment for secondary stroke prevention[9].

Cranial computed tomography (CT) scanning without intravenous contrast administration is a widely available imaging modality for indicated hemorrhage, tumor, or other disease that causes stroke-like symptoms and may exhibits acute or chronic infarcts, moreover additional CT findings could improve prediction of stroke risk after TIA[22,23].

Perfusion computed tomography (PCT) has been less meticulously carried out for TIA, however one-third of hemispheric TIA patients had focal abnormal PCT and 29% of abnormal PCT noticed in completely resolved symptomatic TIA patients. PCT is widely available in most institutions and has more several preferable than magnetic resonance perfusion (MRP) by using helical and spiral CT scan technique, however positive DWI-MRI were found in 25% of patients with normal PCT[24].

Current guidelines recommended MRI with DWI for the best evaluation of patients with TIA, in virtue of most sensitive and specific tests for depicted acute cerebral ischemia, included cerebral microhemorrhage which is too small to demonstrated on CT[22,25,26]. Comparative CT and MRI in previous studies by Foster et al, acute infarcts were ascertained in 8% of TIA patients via CT and 39% via MRI (p<0.0001), CT had a sensitivity of 20% and a specificity of 98% for detected acute ischemic lesions. 306 patients had negative CT findings, presented positive acute ischemic lesion on DWI-MRI in 51%; likewise many studies (32 to 60%),

Table 3. Vascular territories & size of infarcts

Variables	n (%)	Stage	
		Subacute (%)	Chronic (%)
Site of circulation (n = 74)		n = 5	n = 69
Anterior	63 (85.14)	3 (60.00)	60 (86.96)
Posterior	3 (4.05)	1 (20.00)	2 (2.89)
Both	8 (10.81)	1 (20.00)	7 (10.14)
Size of infarction		n = 8	n = 69
Lacunar infarction only	34 (45.33)	-	34 (49.28)
Medium infarction only	16 (21.33)	-	16 (23.19)
Large infarction only	15 (20.00)	8 (100)	7 (10.14)
Lacunar& medium infarction	10 (13.33)	-	10 (14.49)
Medium & large infarction	-	-	-
Lacunar& large infarction	1 (1.33)	-	1 (1.45)
All size infarction	1 (1.33)	-	1 (1.45)

Table 4. Locations of chronic infarcts

Location	n (%), n = 135	Size		
		Lacunar (%), n = 80	Medium (%), n = 42	Large (%), n = 13
Corona radiata	29 (21.48)	16 (20.00)	13 (30.96)	-
Lentiform nucleus	21 (15.55)	20 (25.00)	1 (2.38)	-
Centrum semiovale	14 (10.37)	7 (8.75)	7 (16.66)	-
Internal capsule	13 (9.63)	13 (16.25)	-	-
External capsule	7 (5.18)	7 (8.75)	-	-
Caudate nucleus	6 (4.44)	6 (7.50)	-	-
Insular cortex	3 (2.22)	2 (2.50)	1 (2.38)	-
Parietal lobe	12 (8.89)	-	8 (19.04)	4 (30.76)
Frontal lobe	10 (7.41)	3 (3.75)	3 (7.14)	4 (30.76)
Temporal lobe	8 (5.93)	-	3 (7.14)	5 (38.46)
Thalamus	4 (2.96)	4 (5.00)	-	-
Occipital lobe	4 (2.96)	1 (1.25)	3 (7.14)	-
Cerebellum	4 (2.96)	1 (1.25)	3 (7.14)	-
Total	135 (100)	80 (100)	42 (100)	13 (100)

sustainable DWI-MRI preferred imaging modality to perform with TIA patients[26-31]. In prior studies, 35 to 67% suggestive of cerebral ischemia by restricted diffusion in DWI, and the likelihood of a positive DWI finding increased with symptom duration, so DWI is imaging modality to individually identification at highest risk of early acute ischemic stroke[32,33]. Suspected TIA patients with low risk for intracranial hemorrhage should acquire MRI rather than dual CT and MRI, purposely unnecessary radiation dose prevention from CT with increase CT accessibility for other patients in emergency indications[34].

Regarding previous studies, estimation of CT images disclosed acute infarcts in 3 to 28% and negative acute pathology in 71.4 to 95.7%[27,28,35,36], which related to the authors' results. Some series have informed CT abnormality of every infarcts for TIA patients in 17 to 34%[37-40], through the authors' analysis (30.7%). Furthermore, the most common location of infarcts in TIA patient is at the basal ganglia[41] in accordance with the authors' presentation.

In prior studies, CT images presented normal study in 46% and 21% old infarcts, while cerebral atrophy is detected in TIA patients approximate 20 to 30%[36,38], which correlated with the authors' results. Two prior studies have reported evidence of white matter disease in 23 to 29% cases[36,42], even higher than the authors' CT review.

In preceding clinical case series, 34 (1.59%) of 2,137 spontaneous intracranial hemorrhage by CT scan had TIA episodes[43]. The authors encountered 5 (3.31%) of all patients who had intracerebral hemorrhage, that slightly higher than older report[35]. Otherwise, prior research found 7 focal subarachnoid hemorrhages of TIA patients detected by primary CT image[44], which differed from the authors' inspection.

The most common age of TIA patients is >60 years old; there were 18 (8%) young TIA patients, which resemble previous studies[41,45,46]. According to prior deliberation in South Africa, there were significant different risk factors in older TIA patients which were hypertension, previous smoking, ischemic heart disease and peripheral vascular disease; some of these were related to the authors' results (hypertension and smoking). The younger groups had frequently significant etiologic differences consisting of fibromuscular dysplasia, mitral valve prolapse, and oral contraceptive use more than the older groups[47] which were unavailable data for the authors' record. The present study unveiled significant differences of the risk factors in older TIA patients which were hypertension ($p = 0.042$), diabetes mellitus ($p = 0.008$), alcoholic drinking ($p<0.001$), current smoking ($p = 0.002$), and symptom presentation of muscular weakness ($p = 0.002$) associated with TIA events.

Several meta-analysis recounted a subsequent stroke within 90 days following a TIA event, ranging from 3.9% to 17.3% with slightly lower rates in what the authors' collected (3.46%); the early risk of stroke reported varied from 1.4% to 9.9% at 2 days and from 3.2% to 17.7% at 30 days[48]. At 90-day follow-up, outcome in events for TIA patients cohort series, 4.2% had an acute- or subacute infarcts detected by CT, 17.9% through MRI, 17% stenosis >50% on carotid artery imagings, 12% recognized potential cardioembolic cause via echocardiography[49]. Furthermore, the risk of stroke after TIA with evidence of acute infarction is more than TIA without acute infarction detected by CT or DWI-MRI[40,50] which contrasts with the authors' report, possibly because of lost follow-up medical records.

Even though the favored modality of choice for TIA patient is MRI, MRI is still unavailable in many hospitals in Thailand, which means that CT scan is then commonly applied in TIA patients more than DWI-MRI, because relatively inexpensive, facile availability, and speedy and simple evaluation of CT[51]. In our cerebrovascular service,

Table 5. Consequence of baseline characteristic parameters on CT appearance

Variables	Negative acute pathology n = 225	Acute pathology n = 6	*p*-value
Mean ± SD	71.65±7.95	68.00±5.7	
Age ≥60	146	5	0.732
Male	129	6	0.137
Risk factors:			
Hypertension	125	3	0.472
Diabetes Mellitus	77	2	0.744
Dyslipidemia	75	1	0.284
Atrial fibrillation	25	0	0.348
Coronary artery disease	11	1	0.273
Valvular heart disease	5	0	0.689
History of IHD or MI	14	1	0.398
Previous CVA	15	1	0.439
Previous TIA	11	0	0.547
Alcoholic drinking	28	0	0.315
Current smoking	29	0	0.303
Medications:			
Aspirin	159	3	0.104
Cilostazol	8	0	0.611
Clopidogrel	9	0	0.589
Warfarin	10	0	0.568
Statin	163	2	0.011*
None	182	3	0.012*
Clinical information:			
Duration of symptom ≥60 mins	183	5	0.492
Baseline SBP ≥140 or baseline DBP ≥90	142	4	0.736
ABCD2 score >3	197	6	0.859
Baseline NIHSS ≤5	67	2	0.209
Baseline mRs <2	41	1	0.851
NIHSS at discharge ≤5	60	1	0.745
mRs at discharge <2	29	1	0.891
90 days acute stroke after TIA	8	0	0.491
Symptom presentation			
Muscular weakness	137	4	0.955
Facial palsy	27	2	0.426
Speech impairment	104	6	0.125
Dizziness	31	0	0.560
Numbness/paresthesia	27	0	0.608
Seizure	1	0	0.969
Headache	6	0	0.893
Nausea/vomiting	0	1	0.969

CVA = cerebrovascular accident; IHD = ischemic heart disease; MI = myocardial infarction; TIA = transient ischemic attack; SBP = systolic blood pressure; DBP = diastolic blood pressure; NIHSS = NIH stroke scale; mRS = modified rankin scale

CT has been the primary imaging modality for symptomatic stroke patients, however, many TIA patients should have a further DWI-MRI to diagnose definite etiology and prevent secondary acute ischemic stroke or other disabling events.

Since our study was retrospective, it consequently had some limitations. First, particulars of information depended on the accuracy and consistency in recording patient's information by attending clinicians. Second, regarding the method of patient selection, the present study may underestimate causes by lots of patients who were excluded due to incomplete data information such as CT images. Third, although a number of patients diagnosed with TIA was up to 723 cases (ICD-10 = G45.9), two-thirds of them were excluded, because of unavailable CT scans. Our patients were essentially from the Northeastern district of Thailand; furthermore, our results were useful data in planning the management of the patients around this region.

Conclusion

The present study revealed the prevalence of negative CT findings for acute pathology in TIA which was quite high for normal findings, isolated brain atrophy, only chronic infarction and calcified granulomatous lesions still remained of uncertain etiology, which should have additional

Table 6. Consequence of baseline characteristic & CT appearance classified by age

Variables	Younger age ≤45 n = 18	Older age >45 n = 213	*p*-value
Age (mean ± SD, range)	37.83±6.42 (24 to 45)	66.06±11.04 (46 to 95)	0.000*
Male	12	123	0.461
Male: female ratio	2:1	4:3	
Risk factors:			
Hypertension	6	122	0.042*
Diabetes mellitus	1	78	0.008*
Dyslipidemia	5	71	0.621
Atrial fibrillation	0	25	0.123
Coronary artery disease	1	12	0.300
Valvular heart disease	0	5	0.510
History of IHD or MI	0	15	0.243
Previous CVA	1	15	0.808
Previous TIA	0	11	0.322
Alcoholic drinking	7	21	0.000*
Current smoking	6	23	0.002*
Clinical information:			
Duration of symptom ≥60 mins	14	174	0.752
Baseline SBP ≥140 or baseline DBP ≥90	11	135	0.848
ABCD2 score >3	17	172	0.480
Baseline NIHSS ≤5	8	61	1.000
Baseline mRs <2	3	24	1.000
NIHSS at discharge ≤5	7	54	0.322
mRs at discharge <2	3	27	0.233
90 days acute stroke after TIA	2	6	0.112
Symptom presentation:			
Muscular weakness	17	126	0.002*
Facial palsy	4	26	0.265
Speech impairment	6	104	0.193
Dizziness	3	28	0.717
Numbness/paresthesia	3	25	0.465
Seizure	0	1	1.000
Headache	0	6	1.000
Nausea/vomiting	0	1	1.000
Computed tomography appearance:			
Acute pathology			
Possibly acute infarct	0	1	0.771
Intracranial hemorrhage	0	5	0.771
Negative acute pathology	18	207	0.435

CVA = cerebrovascular accident; IHD = ischemic heart disease; MI = myocardial infarction; TIA = transient ischemic attack; SBP = systolic blood pressure; DBP = diastolic blood pressure; NIHSS = NIH Stroke Scale; mRS = modified Rankin Scale

MRI's performed to recognize acute and/or small lesions. Hypertension, diabetes mellitus, history of alcoholic drinking, current smoking and symptom presentation by muscular weakness are significantly associated factors for TIA in older patients.

What is already known on this topic?

TIA is a robust predictor of subsequent stroke and death. Magnetic resonance imaging (MRI) is the modality of choice for assessing cerebral imaging in TIA, but widely unavailable and limited by contraindication; hence cranial computed tomography (CT) scan play a role in diagnosing TIA patients.

What this study adds?

This study presents CT appearance, the prevalence of negative CT finding for acute pathology, clinical information and associated factors of patients who were diagnosed with TIA. Further investigations such as MRI, carotid or cardiac imaging will assist in defining the most likely cause of the TIA event and to guide management strategies in the patients with history of repeated TIA.

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Potential conflicts of interest

The authors declare no conflicts of interest.

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