THE PREDICTIVE VALUE OF EARLY CT FINDINGS FOR SUBSEQUENT CEREBRAL INFARCTION IN HYPERACUTE ISCHEMIC STROKE

Parichad CHANPONG, M.D.[1] Patama NAKBUMRUNG, M.D.[2]

PURPOSE: To evaluate the relationship and the prediction of early CT signs of ischemic stroke and the location of subsequent infarction.

METHOD: We prospectively evaluated cranial CT signs of 75 consecutive patients with hyperacute ischemic stroke (within 6 hours of ictus) in the territory of the middle cerebral artery with evaluated at least 2 CTs (1 initial, within first 6 hours and 1 repeated CT within 48-72 hours of onset to confirm the infarct location) by one radiologist. On the first CT, early signs were hyperdenseMCA sign (HMCAS), early parenchymatous signs (attenuation of the lentiform nucleus [ALN], loss of the insular ribbon [LIR]), and early cortical edema are hemispheric sulcal effacement [HSE] and cortical hypodensity. Subsequent infarct locations were classified according to total, partial superficial, deep, or multiple MCA territories.

RESULT: Early CT abnormalities were found in 42 patients (56%). Isolated sign (HMCAS, isolated ALN and isolated LIR) 6 patients (14%), two signs (ALN/LIR) 7 patients (16%) and more than two signs 29 patients (70%). We found isolated HMCAS, two parenchymatous signs (ALN/LIR) and one or both parenchymatous signs (ALN or LIR) with cortical edema (HSE /cortical hypodensity) were strongly associated with subsequent large infarction and the positive predictive value of these signs for subsequent large infarctions were 100%, 85.7% and 86% respectively. The positive predictive value of isolated parenchymatous sign (isolated ALN or LIR) for deep infarction was 100% but the positive predictive value of negative early CT signs and isolated parenchymatous sign for subsequent large infarction were 0% and negative predictive value of these signs were 75% and 86.5% respectively.

CONCLUSION: Our findings suggested that positive early CT signs in first 6 hours allow the prediction of subsequent infarct locations. Early parencymatous signs associated with early cortical edema are strongly associated with subsequent large infarction but negative early CT signs and isolated parenchymatous sign are associated with subsequent deep infarction.

MCA = Middle Cerebral Artery
HMCAS = Hyperdense MCA sign.

[1] Division of neuroradiology Prasat Songkhla Neuropsychiatric Hospital Thailand
[2] Division of radiology Surathanee Hospital Thailand

INTRODUCTION

Cerebrovascular disease (CVD) or stroke is considered to be a national medical problem in Thailand.[1] In Thailand cerebral infarction was found about 70% of all stroke[2] lower than in America, ischemic stroke was found about 80-85%.

CT is widely used for early evaluation of acute strokes. Most importantly, CT excludes acute hemorrhage or other diseases mimicking ischemia (tumor, subdural hematoma). Therefore, CT is the main imaging examination in patients with brain ischemia and when antithrombotic agents are considered.[3,4] There are many early CT signs of ischemic stroke, hyperdense middle cerebral artery sign (HMCAS), early parenchymal abnormalites which are the attenuation of lentiform nucleus (ALN), loss of the insular ribbon (LIR), hemispheric sulcal effacement (HSE). Early parenchymal abnormalities might also predict subsequent infarct extension [5]and hemorrhagic transformation. [6]Finally, initial CT findings may help to predict response to therapy.[7] There are few previous studies about early CT signs and subsequent infarction. The purpose of this study is to evaluate the relationship and the prediction of the early CT signs in hyperacute period ischemic stroke in 6 hours of first ever stroke for each types of subsequent cerebral infarction.

METHOD

We prospectively evaluated cranial CT signs of 75 patients first ever episodic ischemic stroke in the MCA territories with clinically hemiparesis, from April 2001 to April 2002. All patients were evaluated neurological sign; Glasgow coma score and muscle power by the neurologist and taking CT scan before admitted in the stroke unit Prasat songkhla neuropsychiatric Hospital. The time from ictus to the time at the perfomed CT scan was not more than 6 hours. The all CT imagings were evaluated and analyzed by a radiologist, who did not know the clinical but aware the clinical stroke. The first CT scans were done in all 75 patients within 6 hours after onset. Repeated imaging studies (second CT scan) were performed in all cases within 48 –72 hours after stroke onset to confirm the size and location of infarction. Collected information included analysis cranial CT signs within 6 hours of onset (hyperacute period) of 1st CT scan and subsequent CT findings in second CT scan.

CT technique: All non-contrast CT brains were performed using a Toshiba spiral CT scanner - Auklet /FS with 512x512 metrix and 1 second scan time. The section thickness and increments were 5 mm., from the foramen magnum to the suprasellar region and 10 mm. contiguous slices above, the early CT signs were classified as:

1. HyperdenseMCA sign (HMCAS): spontaneous high contrast in the MCA. This definition required that the vessel appear not only brightly than adjacent brain tissue but also brighter than the other intracranial arteries, especially contralateral MCA and finally that it not be attributable to calcification.[8]
2. Attenuation of the lentiform nucleus (ALN): a decrease in density involving the lentiform nucleus area inducing the loss of the precise delineation of this area.[9]
3. Loss of insular ribbon sign (LIR): decreased precision in delineating the gray-white interface at the lateral margin of the insula.[10] The ALN and LIR signs are parenchymatous signs.
4. Hemispheric sulcal effacement (HSE)
5. Cortical hypodensity. HSE and cortical hypodensity are represent early cortical edema.

We analyzed CT findings in the first 6 hours after stroke

The results of CT finding were classified as:

1. No abnormality visible or negative findings.
2. Abnormality visible or positive findings, one or more than one signs in CT scan.

Second CT scans were performed in 48 hours after first CT scan in all cases. Using templates,[11] infarct locations were classified, as follow

1. Large MCA territory, if total superficial and deep MCA territories or the total superficial MCA territory were involved.
2. Multiple MCA infarct; if partial superficial MCA and deep territories were involved.
3. Deep MCA infarct, if the infarct was limited to the deep MCA territory.
4. Partial superficial MCA infarct, if the superficial (superior, insular, or inferior) MCA territory was Involved.

RESULT

The analyzed CT signs were

Table 1

First CT (within 6 hours of stroke)	Number of patients(%)
Negative finding	33 (44%)
Positive findings	42 (56%)

Table 2 Number and percentage of patients for each positive CT signs

ALN	LIR	HSE	Cortical hypodensity	HMCAS
34 (81%)	30 (71%)	29 (69%)	29 (69%)	2 (4.7%)

Table 3 Distribution of early CT signs (isolated and in association with each other)

	ALN (n= 34)	LIR (n=30)	HSE (n=29)	Cortical hypodensity (n=29)	HMCAS (n=2)
Isolated sign	2	2	-	-	2
2 signs					
ALN		7	-	-	-
LIR	7		-	-	-
3 signs	8	-	8	8	-
	-	4	4	4	-
4 signs					
(ALN/LIR/HSE/ cortical hypodensity)	17	17	17	17	-

ALN = Attenuation of lentiform nucleus, **LIR** = Loss of the insular ribbon
HSE = Hemispheric sulcal effacement

Table 4 Distribution between early CT signs and subsequent infarct lesions

2nd CT / 1st CT	Subsequent large Infarction	multiple Infarction	deep Infarction	partial superficial Infarct
Negative CT [n=33(44%)]	-	2	25	6
Positive CT [n = 42(56%)]				
-Isolated HMCAS (n = 2)	2 (2.6%)	-	-	-
-Isolated ALN (n = 2)	-	-	2	-
Isolated LIR (n = 2)	-	-	2	-
2 signs				
-ALN/LIR (n = 7)	6 (86%)	1 (25%)	-	-
3 signs				
-ALN/HSE/cortical Hypodensity (n = 8)	6 (75%)	2 (25%)	-	-

Table 4 (Continue) Distribution between early CT signs and subsequent infarct lesions

2nd CT / 1st CT	Subsequent large Infarction	multiple Infarction	deep Infarction	partial superficial Infarct
-LIR/HSE/cortical Hypodensity (n = 4)	3 (75%)	1	-	-
4 signs				
-ALN/LIR/HSE/ cortical Hypodensity (n = 17)	16 (94%)	1	-	-
Total	33 (44%)	7 (9%)	29 (39%)	6 (8%)

RESULT

There were 41 men and 34 women with a mean of 53 years old.

CT CHARACTERISTICS

The first CT was performed at a mean time of 5.4 hours .The first CT was normal in 33 cases. (44%), and it showed at least one abnormalities in 42 pts. (56%) (table 1). ALN was observed in 34 patients and the most common sign(table2), isolated in 2 of 34 pts. (6%), and was frequently found with LIR 32 patients (94%) and with HSE/

cortical hypodensity 25of 32 pts. (78%) (table 3). LIR was found in 30 patients, isolated sign 2 patients (6%), associated with HSE and cortical hypodensity was 21 of 30 pts. (70%)which was nearly ALN. HMCAS was observed in 2 patients (2.6%) and seen isolated sign, no associated with other signs. HSE and cortical hypodensity were not found in isolated sign but they are found associated with one or both parenchymatous signs, associated with one parenchymatous sign (ALN or LIR) 12 of 29 pts. (41%)(ALN 28%, LIR 14%)and both ALN/LIR 17 of 29 pts (59%).The second CT was performed on mean 57 hours after the first CT performed. Subsequent infarcts involved the large MCA territory in 33 pts (44%)of the patients, the multiple MCA territories in 7 pts (9%), the deep MCA territory in 29pts (39%) and partial superficial MCA territory in 6 cases (8%)(table 4)

NUMBER OF EARLY CT SIGNS AND SUBSEQUENT INFARCT TOPOGRAPHY (TABLE4)

In isolated sign, only 2 HMCAS patients developed subsequent large infarction, 2 in 6 patients (33%) but all isolated parenchymatous signs (ALN or LIR) patients developed subsequent deep infarction, no patients developed subsequent large infarction, subsequent multiple infarctions and partial superficial infarction.

In the patients with two positive signs (all were parenchymatous signs), subsequently developed large infarction , 6 in 7 cases (86%) and 1 case with multiple infarctions. In the patients with 3 CT signs positive are also associated with subsequent large infarction 9 in 12 cases or 75% with 3 patients developed multiple infarctions. In patients with 4 signs positive 16 of 17 patients developed large infarction (94%). No association between two or more than two signs with deep partial superficial infarcts (0%). Deep infarction and superficial infarction were found in 25 of 33 patients (75%), 6 of 33 patients (18%) respectively in cases of no abnormalities of early signs. We found hemorhagic transformation 12 of 33 patients (36%) in subsequent large infarction and 10 of these 12 hemorrhagic transformation had midline shift and mass effect affecting lateral ventricle.

EARLY CT FINDINGS AND SUBSEQUENT INFARCT TOPOGRAPHY.

In cases with negative early signs (33 pts), developed subsequent infarcts involving the deep MCA territory in 25 patients, partial superficial territory in 6 patients, multiple MCA territories in 2 patients but never develope infarction in large MCA territory. (table 4)

In case positive early CT signs (42 cases), all 2 isolated HMCAS patients developed large MCA territories infarct (100%) equally to isolated parenchymatous signs (isolated ALN or isolated LIR) which all were associated with deep infarction (100%). Two parenchymatous signs were found association, ALN/LIR in 7 patients, 6 of 7 patients developed subsequent large MCA territory (86%). One parenchymatous sign with early cortical edema (ALN/ HSE/ and cortical hypodensity or LIR/ HSE/ and cortical hypodensity) developed subsequent large infarction 6 in 8 cases and 3 in 4 cases respectively or equal to 75%. Two parenchymatous signs with early cortical edema (ALN/LIR/ HSE/ and cortical hypodensity) was seen in 17 patients and 16 of 17 developed subsequent large MCA territory infarction (94%) and multiple infarctions only 1 case.

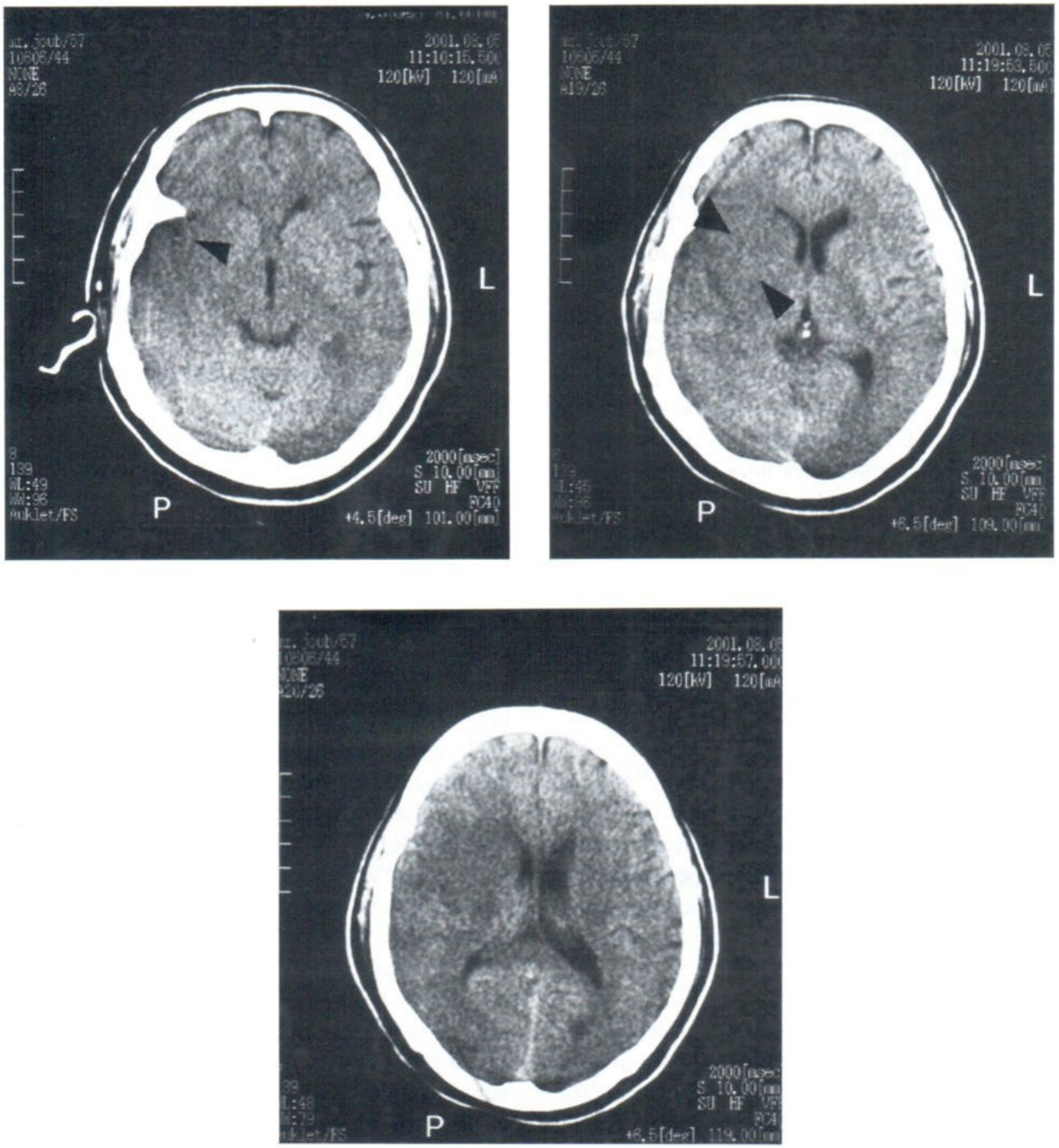

Fig. 1-3 Patient presented with acute stroke about 6 hours of onset. CT scan shows loss of the insular ribbon sign (LIR) (arrow head in figure 1), Attenuation of the lentiform nucleus (ALN) sign; (between arrows heads in figure 2) and Rt. hemispheric cortical effacement (HCE) compare to normal Lt. side in figure 3.

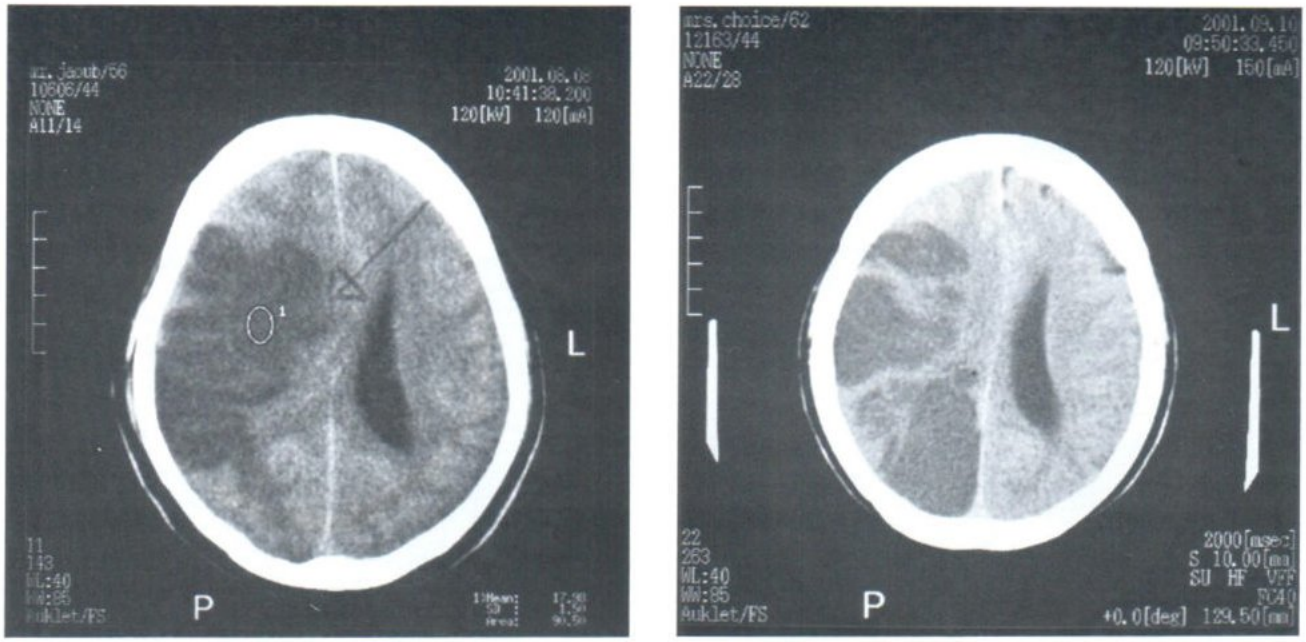

Fig. 4-5 The same patient followed up CT at 48 hours developed subsequent large infarction

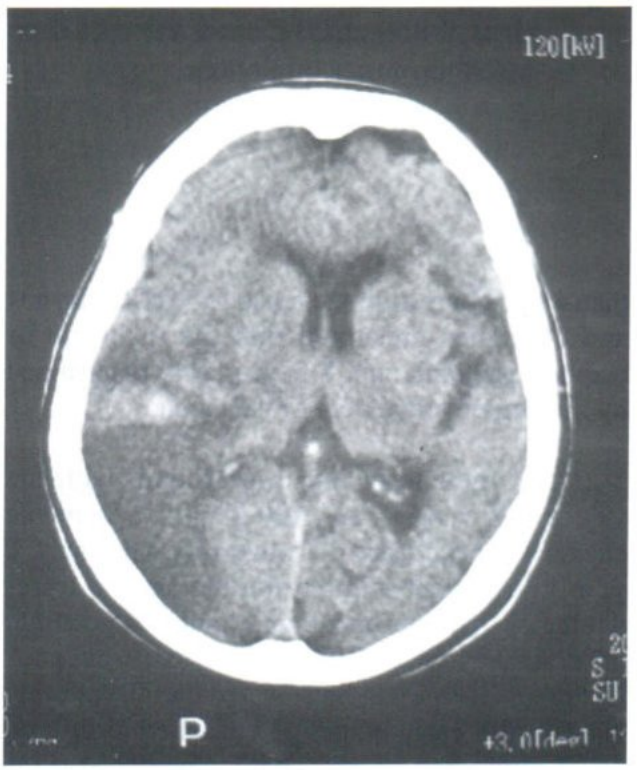

Fig. 6 Another patient with positive early CT signs showed subsequent large infarction with hemorrhagic transformation.

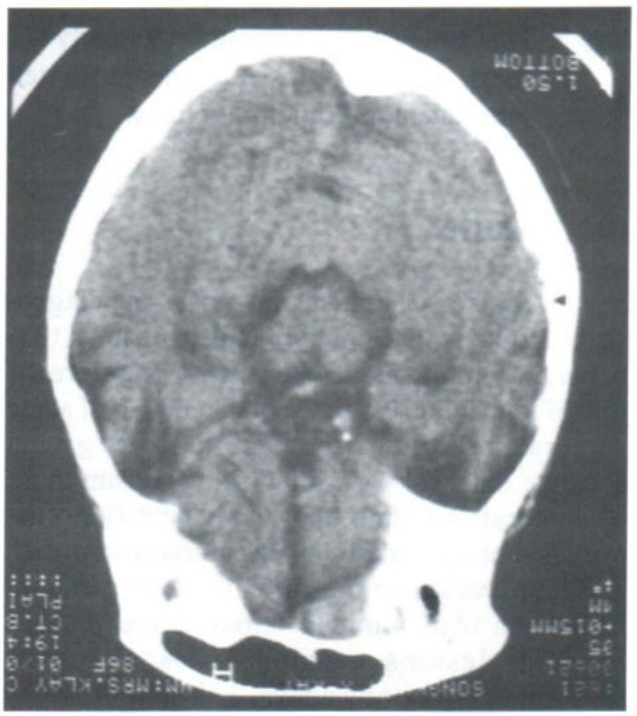

Fig. 7 Hyperdense Middle Cerebral Artery Sign (HMCAS). The patient unenhanced CT showing left hyperdense sign (HMCAS), this patient developed subsequent large infarction.

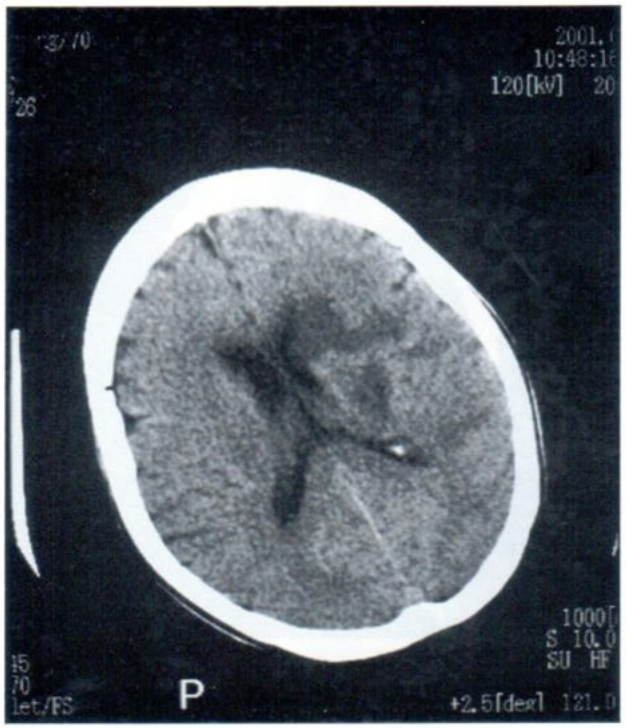

Fig. 8 This patient showed positive for early CT signs, followed up CT demonstrated subsequent multiple infarctions.

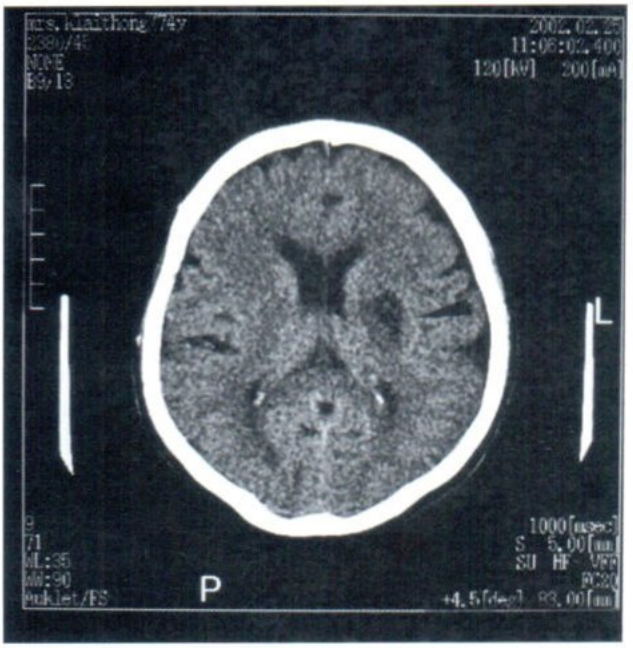

Fig. 9 This patient showed negative early CT signs, follow up CT demonstrated Lt. deep MCA territories infarctions.

DISCUSSION

Stroke is a clinical diagnosis, the diagnosis of stroke is inaccurate in approximately 13% of patient admitted to stroke units.[12] Therefore the role of immediate CT in the management of acute cerebral infarction is two fold: 1. In giving accurate diagnosis or excluding ICH from those of cerebral ischemia and 2. In identifying the presence of an underlying structural lesion such as tumor, vascular malformation, or subdural hematoma that mimick stroke clinically, non vascular lesions causing 1-2% of stroke syndromes[13] and nowadays, it is important to select patients for given thrombolytic therapy. The CT findings in acute cerebral infarction evolve with time. Although almost 60% of CT scans obtained within the first few hours after cerebral infarction are normal,[14] but several early signs of acute stroke can often be identified in stroke less than 4 to 6 hours after the episode. Von Kummer R and collagues presumed that the visibility of cerebral ischemia in CT is primarily determined by the degree and extent of ischemia.

If hypoperfusion is profound, with the values below 10-15 mL. 100 g^{-1}. min^{-1}, ischemic edema will occur early.[15] Conversely, a normal CT scan in a patient with stroke indicates a lesser degree of hypoperfusion and indicates potential reversibility of the functional disturbance.[16] Cytotoxic edema can be detected by CT scanning even in the early stages after ischemic infarct, which presented as early parenchymatous signs (ALN and LIR).

The x-ray attenuation seen with CT is directly proportional to tissue water content. A 1%

increase in tissue water content results in a 3 % to 5% decrease in x-ray attenuation, which translates into a decrease of approximately 2.5 Housfield units (HU).[17 - 18] In the past decade, a number of authors have identified CT findings which are based on the detection of early cytotoxic edema.[19-21] In our series, early CT abnormalities in the first 6 hours were usually found in all patients(56%). Our result were similar to those previously reported which vary from 31 to 92 %, depending on the time on which the CT was performed (4 to 8 hours).[9- 10, 22 - 26]

Previous studies evaluated early prenchymatous signs including ALN and LIR.[27] Many previous studies demonstrated that ALN was the earliest and the most common sign. More than 70% of cases (range from 60-92%) which CT were performed within 6 hours of onset. Our study shows ALN is the most common sign (81%) in agreement with the previous studies.[28 - 31]

HSE is the reflection of extracellular vasogenic edema, which is subsequent to intracellular cytotoxic edema. This is the superficial MCA territories sign and usually seen in early 3 hours or more after the onset of ischemia and rarely found isolation.[32 - 33] Our study found that HSE always associated with cortical hypodensity (100%) and frequently seen with one or both parenchymatous signs. Both HSE and cortical hypodensity represent cortical edema.[31]

HSE = Hemispheric sulcal effacement

The positive predictive value of isolated parenchymatous sign for subsequent large infarction and for deep infarction were 0% and 67% respectively and the negative predictive value of these signs were 87% and 0 % respectively. The positive predictive value of two parenchymatous signs (ALN/LIR) for subsequent large infarction was 85.7%, higher than one parenchymatous sign associated with early cortical edma (one parenchymatous sign with HSE/cortical hypodensity) which is 75% but less than the combination of two parenchymatous signs with cortical edema (both parenchymatous signs with HSE/cortical hypodensity) which is 94%. The negative predictive value for subsequent large infarction of two parenchymatous signs, three signs and four signs were 75%, 80% and 63% respectively. Our results confirmed an association between the number of early CT signs and subsequent large cerebral infarction corresponding to previous study[31] but bilateral parenchymatous signs should be more reliable signs than only one parenchymatous sign with sign of early cortical edema which has not been in the previous study. Therefore, we believe that both parenchymatous signs associated with signs of early cortical edema may be the most reliable predictive factor the for subsequent large cerebral infarction.

On the whole, hemorrhagic transformation (HT) seems to be correlated to both the intensity of ischemia and the extension of delayed infarcted rather than to the presence of early CT signs.[25,34] However, previous studies demonstrated a close relationship between hemorrhagic transformation and early CT signs[6,35] and another study found that hypodensity of the lentiform nuclei on early CT studied was strongly associated with later hemorrhagic transformation of the initially ischemic infarction.[36] Our study demonstrated that hemorrhagic transformation in 12 of 33 patients (36%) with subsequent large MCA infarct and 10 of these 12 patients had midline shift which is lower than in the previous study[31] which HT (Haemorhagic transformation) were found in about 50 % of the patients.

Hyperdense MCA sign is caused by acute intraluminal thrombus. The hyperdense MCA sign has been reported in 25% of all unselected acute infarcts and is seen in 35-50% of patients who have stroke symptoms in the MCA territory.[37 - 39] The hyperdense MCA sign typically occurs with cortical and large, deep MCA infarcts.[38] HMCAS in our results was found in 2 patients or 2.6% and had never been seen associated with other signs which was different from the previous studies (26-50%).[5, 25 - 26, 40] The specificity of HMCAS is very good, ranging from 85 to 100% in most series.[22,25–27,40]

However, some patients with clinical MCA ischemia do not exhibit HMCAS despite of an angiographic proven MCA occlusion, illustrating a lower sensitivity that varies from 29 to 69 %.[24-26,41] Our standard CT procedure did not include specific scan (less than 5mm.in thickness) on the perisylvian fissure, which may provide a higher positive rate. On the other hand. Although HMCAS usually occurs very early in time course of MCA occlusion, the MCA occlusion is often a transient phenomenon, as suggested by serial CT studies [24] and the value of isolated HMCAS as a predictor of outcome remains controversial.[27,40,42] Our study found the HMCAS only in 2 patients due to only a few patients had been studying.

In case of negative CT or no abnormalities visible with subsequent infarction, no studies have previously been reported, we found the positive predictive value of large infarction was 0% and negative predictive value was 78% but the positive predictive value for deep infarction and partial superficial infarction were 75%and 19% respectively. So our study confirm the clinician to avoid repeating CT in cases with negative early signs in hyperacute ischemic stroke patients because the chance of developing subsequent large infarction is low but it may associated with subsequent deep infarction and partial superficial infarction. The number of cases we studied is rather small. It needs further study in future. The association between each early CT signs, times related and subsequent infarct extension could be substantiated in further studies.

In summary, CT is still the first choice in the differential diagnosis of acute stroke and when elected thrombolytic therapy, has to be given within the first 3-6 hours after the onset of symptoms. Early CT signs of ischemia are frequently present, even during the first few hours of ischemic stroke. Early CT signs may allow the prediction of further infarct size and location. We mentioned on large infarction because there are many complications of large infarction such as hemorrhagic transformation, brain edema and most severely, brain herniation. Our study confirmed that the number and types of early CT signs may also indicate and predict the types of subsequent infarction, especially large infarction. The cases with negative early CT signs may develope deep infarction with few partial superficial infarction without developing large or multiple infarction. So repeated CT in cases with negative early sign in clinically ischemic stroke may be unnecessary. Finally, the presence or absence of early CT sign have implications in the diagnosis, prognosis, choice of supplementary examination and the choice of treatment.

ACKNOWLEDGEMENTS

We thank Professor R von Kummer for his helpful advices together with some reprints and Dr.Pitchaya Tantiseranee for many good comments.

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