Identification of Stroke Mimics among Clinically Diagnosed Acute Strokes

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Background: *Stroke is a clinically syndrome of a sudden onset of neurological deficit in a vascular cause. Stroke mimics is the non-vascular disorders with stroke-like clinical symptoms. It is important to distinguish true stroke from mimics since treatment plan may differ.*
Objective: *To determine the incidence of the stroke mimics and identify their etiologies.*
Material and Method: *All non-contrast head CT of the patients with clinically diagnosed stroke who immediately received imaging upon arrival at the emergency department of the university hospital were retrospectively reviewed in 12-month period between January 1 and December 31, 2008. Medical records, laboratory results, MRI, and 6-month clinical follow-up records were reviewed for final diagnosis.*
Results: *Seven hundred four patients were included in this study, including 363 (51.5%) men and 341 (48.5%) women with range in age from 24 to 108 years. Amongst those, 417 (59.2%) were ischemic stroke, 80 (11.4%) were hemorrhagic stroke, 186 (26.4%) were stroke-mimics, and 21 (3%) were inconclusive. The etiologies among stroke-mimics were metabolic/intoxication (35, 18.8%), sepsis (28, 15.0%), seizure (21, 11.3%), syncope (20, 10.8%), subdural hemorrhage (14, 7.5%), vertigo (11, 6.0%), brain tumor (10, 5.3%), central nervous system infection (5, 2.7%), others (26, 14.0%), and unspecified (16, 8.6%).*
Conclusion: *Incidence rates and etiologies of the stroke mimics were similar to the western reports. However, the frequency of each mimic was not.*

Keywords: *Acute strokes, Ischemic stroke, Hemorrhagic stroke, Stroke mimics, Computed tomography*

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Stroke is a clinical syndrome of a sudden onset of focal, or sometimes global, neurological deficit in a recognizable vascular cause, with common presentations including hemiparesis, facial weakness, and aphasia. Patients who present with a suspected stroke might have a true stroke or stroke mimics[1-3]. Both ischemic stroke and hemorrhagic stroke, including subarachnoid hemorrhage, are classified as true strokes[4]. Stroke mimics is the term for non-vascular disorders with stroke-like clinical symptoms, including processes within the central nervous system (CNS) or systemic events. There are clinical variables for distinguishing between true strokes and mimics. A decreased level of consciousness and normal eye movement increases odds of mimics, however, the sensitivity and specificity of these clinical variables were only 21% and 96% respectively[5].

Imaging plays a key role in current guideline and can offer critical information for proper management of acute stroke patients[6]. Patients with computed tomography (CT) proven non-hemorrhagic stroke may be treated with thrombolytic agents within three to six hours after symptom onset[7,8]. It is important to distinguish non-hemorrhagic stroke from stroke mimics since the treatment plan may differ[9,10]. As a result, awareness of stroke mimics is essential to avoid inappropriate thrombolysis.

The aims of the present study were to determine the incidence of stroke mimics in patients with clinically diagnosed acute strokes, and identify their etiologies of stroke mimics.

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Material and Method

The present study was a single site, observational retrospective study. It was based on a 1,000-bed university hospital with a 6-bed acute stroke unit, an emergency department, and imaging modalities including computed tomography (CT), magnetic resonance imaging (MRI), catheter angiography, and carotid ultrasound. The study was approved by Ramathibodi Hospital's Institutional Review Board.

All patients arrived at the emergency department in the 12-month period between January 1 and December 31, 2008 with a clinical diagnosis of acute stroke and underwent immediate head CT scans were enrolled in the investigation. Our institution required that a Stroke Code be activated for every patient suggested to have an acute stroke irrespective of time from onset, severity of the symptom or ultimate diagnosis. A stroke alert paging system was activated by emergency physician. The pager alerted the stroke neurologist on call, and radiology service. Head CT has been a routine emergency imaging of possible acute stroke. Clinical assessment was done by a stroke-code team within an hour. If patients were admitted to the hospital, they would receive coagulation screen, blood glucose level, electrolytes, hepatic and renal function assessment. Patients who obtained medical treatment before the completion of the head CT scan were excluded from this study.

Imaging

All patients received CT scan, which was performed on 64-slice multi-detector scanner (Somatom Sensation Cardiac 64, Siemens, Germany). The routine protocol was non-contrast CT (NCCT) of the whole brain with 120 kVp; effective 500 mAs; 3.0-mm slice thickness and 0.6-collimation. Images were acquired in the orbitomeatal plane from the skull base to the vertex. The contrast enhanced CT (CECT) and/or CT angiography, CT perfusion, and MRI with DWI were performed in some patients at the discretion of attending radiologists and/or neurologists who monitored the examinations.

All head NCCT scans finalized by attending radiologists were retrospectively reviewed with unawareness of the final diagnosis and categorized based on the imaging findings into acute ischemic stroke, hemorrhagic stroke, subdural hemorrhage, focal mass lesion, old infarction, negative result, or a combination of these. Ischemic strokes were defined by identification of acute thrombus in the intracranial arteries or parenchymal abnormalities visualized as hypoattenuation in the vascular territories. Hemorrhagic strokes were subdivided into compartment of hemorrhages such as intraparenchymal, intraventricular or subarachnoid hemorrhages. Subdural hemorrhage was defined as a crescent-shaped extra-axial collection. Focal mass lesions were those with solitary or multiple masses/nodules in the brain. Old infarction was defined as area of well defined hypoattenuation with evidence of volume loss.

Determination of the final diagnosis

The authors retrospectively reviewed medical records of all patients to identify the final diagnosis, which was defined as the most probable diagnosis at discharge or 6-month clinical follow-up. The final diagnosis was documented by attending neurologists on the basis of laboratory results, neuroimaging, or histopathology. Each patient was classified as ischemic stroke, hemorrhagic stroke, stroke mimics, and inconclusive diagnosis. Ischemic stroke patients were further subdivided into transient ischemic attack (TIA) or acute infarction. TIA was reserved for transient neurological deficit less than 24-hour duration without imaging evidence of infarction. The ischemic strokes with negative head CT were documented by follow-up head CT or MRI brain with DWI. The subtypes of hemorrhagic stroke were finally diagnosed on basis of clinical history, characteristic findings on neuroimaging or vascular diagnostic work up. Stroke mimics were defined as disorders suggestive of acute brain dysfunction of non-vascular origin. Inconclusive results were those with incomplete medical records, lack of hospitalization, and no evidence of acute infarction or hemorrhage on head CT.

The investigators separated mimics into the following categories: toxic/metabolic disease, sepsis, seizure, syncope, vertigo, subdural hemorrhage, brain tumor, CNS infection, other, and unspecified. Toxic/metabolic category was documented by laboratory investigations during admission. Sepsis was diagnosed by clinical and laboratory results including hemoculture. The syncope, seizure, and vertigo were transient medical illnesses that were severe enough to require brief hospitalization. Regarding brain tumor category, primary brain tumors were defined by histopathology, whereas patients with metastatic brain tumors were diagnosed by evidence of solitary or multiple nodules on neuroimaging with history of known primary cancers. CNS infections were diagnosed based on stereotactic aspiration and/or microbiology.

The incidence and etiologies of stroke mimics were estimated in relation to final diagnosis, presented as percentage.

Results

Seven hundred four patients were included in the present investigation. There were 363 (51.6%) men and 341 (48.4%) women with range in age from 24 to 108 years. Incidence, mean age, and gender of the patients with ischemic stroke, hemorrhagic stroke, and stroke-mimics, were listed in Table 1.

Among 374 cases of ischemic stroke, 198 (53%) were diagnosed by NCCT that demonstrated imaging findings of acute cerebral infarction (Fig. 1). The negative results on NCCT in the remaining patients (176, 47%) were diagnosed by follow-up head NCCT or MRI brain with DWI (Fig. 2). All patients with hemorrhagic stroke were detected by the NCCT, and 45 of 80 patients (56.3%) were specified as hypertensive hemorrhage by history of hypertension and classic locations of intracerebral hemorrhage, such as in the basal ganglion, thalamus, external capsule, pons, and cerebellum with or without rupture into the ventricular system (Fig. 3). The remainder was amyloid angiopathy, cavernoma, arteriovenous malformation, coagulopathy, and vasculopathy in order of decreasing frequency. Among six patients (7.5%) with subarachnoid hemorrhage, four patients had documented aneurysms on CT angiography or conventional cerebral angiography.

Nearly all patients with stroke-mimics received diagnoses of one of many specific etiologies as shown in Table 2. The most frequent mimic was toxic/metabolic disturbance, which consisted of hypoglycemia, hyperglycemia, hyponatremia, hepatic encephalopathy, uremia, diabetic ketoacidosis, or drug-induced encephalopathy, and most patients presented with alteration of consciousness. The next most frequent mimic was sepsis. Twenty-six of 28 (93%) patients in this group were documented from urinary tract infections. The remaining diagnoses were seizure, syncope, vertigo and others, e.g., conversion disorder, myasthenia gravis, migraine, multiple sclerosis, and posterior reversible encephalopathy syndrome. Twelve of 21 (57%) patients with seizure had experienced a previous stroke, and had an area of old infarction shown on head CT. All patients with syncope and

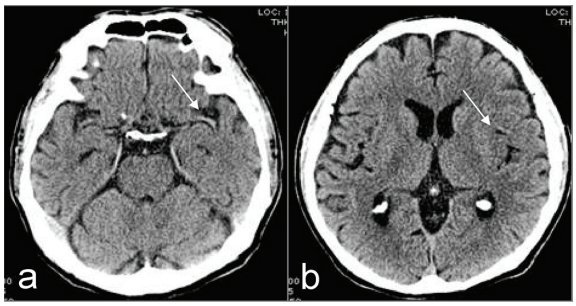

Fig. 1 A 73-year-old man with acute infarction.
a, b) non-enhanced CT (NCCT) scan revealed acute thrombus in the horizontal left middle cerebral artery (arrow in a) and loss of gray-white differentiation at the left insular cortex (arrow in b).

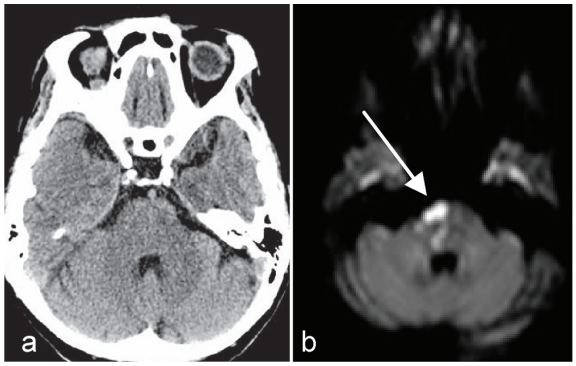

Fig. 2 An 82-year-old woman with acute infarction.
a) NCCT scan is unremarkable.
b) DWI image shows small lesion at the right-sided of pons with restricted water diffusion shown as bright signal intensity on DWI (arrow) and low signal intensity in ADC image (not shown).

Table 1. Incidence of patients with clinically diagnosed acute stroke

Diagnosis	N	%	Mean age (range)	Gender (% male)
Ischemic stroke/TIA	417	59.2	65.7 (27-93)	54
Hemorrhagic stroke	80	11.4	62.6 (28-92)	56
Mimics	186	26.4	66.3 (24-108)	42
Inconclusive	21	3.0	NA	NA
Total	704	100.0		

N = number of patients; TIA = transient ischemic attack; NA = non available

Table 2. Stroke mimics diagnosis

Diagnosis	N	%
Toxic/Metabolic	35	18.8
Sepsis	28	15.0
Seizure	21	11.3
Syncope	20	10.8
Vertigo	11	6.0
Subdural hemorrhage	14	7.5
Brain tumor	10	5.3
CNS infection	5	2.7
Other	26	14.0
Unspecified	16	8.6
Total	186	100.0

N = number of patients

vertigo, head CT were negative. The patients with syncope were awake by the time they got head CT examination or received brief hospitalization without neurological deficit. Finally, there were unspecified results in 16 patients (8.6%) by clinical, complete laboratory tests, and brain imaging.

Among patients with CNS space occupying lesions (29 of 186 patients, 15.5%), the most frequent cause was subdural hemorrhage (SDH), which was identified as acute/subacute stages in nine patients (Fig. 4), and multistage in five patients (Fig. 5). The next most frequent etiology was brain tumors. The majority of them (8 of 10 patients) were metastasis (Fig. 6), whereas two patients had pathological proved glioblastoma multiforme and meningioma respectively. The least frequent etiology was CNS infection, which was specified with focal cerebritis in four patients, and brain abscess in one patient (Fig. 7). Almost all CNS space-occupying lesions were detected by head CT except one case with cerebritis.

Discussion

Acute stroke syndrome is the abrupt onset of neurological deficit. The bedside diagnosis is still problematic because there are several subtypes. The use of NCCT for stroke evaluation is widespread, since it is fast, readily available in an emergency setting and easily performed in any patients. NCCT is highly accurate for diagnosis of intracranial hemorrhage but is relatively less sensitive for the detection of early signs of cerebral infarction with sensitivity ranging from 16 to 61%[10,11], which depends on the time of investigation. In the present study, the sensitivity was

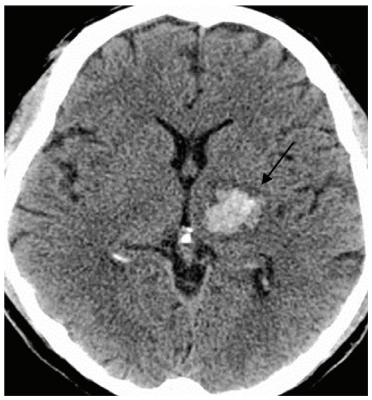

Fig. 3 A 53-year-old man with hypertensive hemorrhage. NCCT scan shows acute hemorrhage at the left thalamus (arrow).

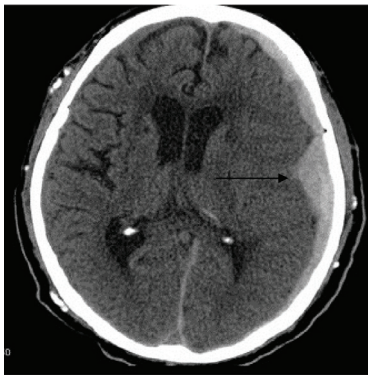

Fig. 4 A 66-year-old man with acute subdural hemorrhage. NCCT scan showed a crescent hyperattenuating collection at the left cerebral hemisphere (arrow).

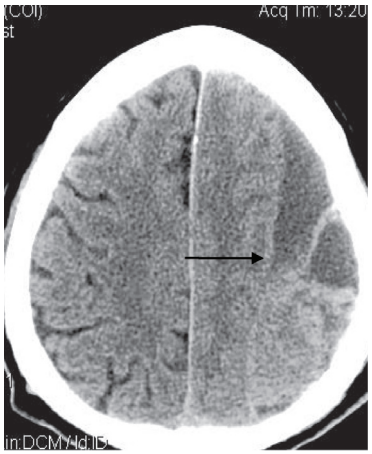

Fig. 5 A 76-year-old man with multistage subdural hemorrhage. NCCT scan showed crescent mixed hypo-hyperattenuating collection with fluid level at the left fronto-parietal region (arrow).

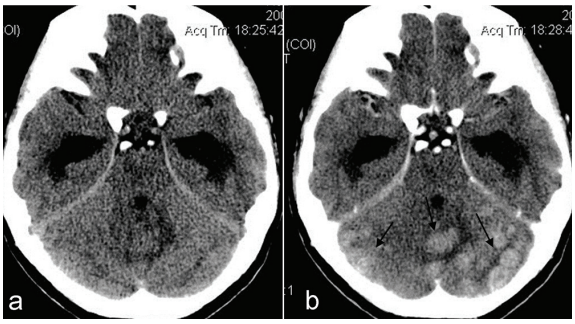

Fig. 6 A 47 year-old-woman with brain metastasis. a) NCCT and b) contrast enhanced CT (CECT) scans showed multiple small enhancing isoattenuating nodules at bilateral cerebellar hemispheres (arrows) with obstructive hydrocephalus.

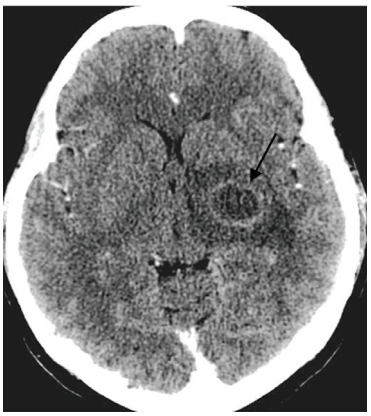

Fig. 7 A 33 year-old-man with brain abscess. CECT scans show a smooth thin-rim enhancing lesion at the left thalamus with surrounding edema (arrow). Stereotactic aspiration revealed flank pus with positive *Klebseilla* sp.

53%, which was within reported ranges. The authors found that CT signs of early infarction were detected in large arterial territory, while many patients with mild ischemic strokes did not develop a visible infarction on CT, similar to the previously reported studies. NCCT could detect all acute hemorrhagic stroke of various etiologies.

The incidence of stroke-mimics in the present study (26.4%) was similar with western reports, within the range of 19 to 31%[3,5,12-14]. The mean age and range in patients with true stroke and mimics were similar in the present study. However Patrick CA et al[3] demonstrated that there was difference in incidence of the stroke mimics between patients under and above the age of 50 years with 21% and 3% respectively, migraine and conversion disorder were the predominant stroke mimics in the younger age group. Although etiologies of stroke mimics in the present study were not different from the previous reports[2,3,12,13], the frequency of each mimic were. The causes of stroke mimics in the present study were toxic/metabolic disturbance, sepsis, seizure, and syncope in the following frequency. In contradiction to the study of Libman et al[5] that demonstrated unrecognized seizure with postical deficits, systemic infection, brain tumor, and toxic/metabolic in the following frequency. The reason was unclear, whether the duration of the postictal deficit can be short, misdiagnosed as TIA[15,16]. The investigators found that there was association of prior stroke deficits and seizure in patients with stroke mimics. Stroke survivors may present inaccuracy in initial history taking or increased awareness of stroke-like symptom.

Hypoglycemia was the most frequent etiology among metabolic condition in the present study. The clinical manifestations can be considered in two patterns that are alteration of consciousness or focal neurological deficit, so-called hypoglycemic hemiplegia as a result of hypoglycemia-induced brain injury[17]. CT brain is usually unremarkable. Although uncommon, hypoglycemia should be listed in differential diagnosis of acute stroke. Blood glucose determination is a simple, fast, and inexpensive test, and is an effective screening procedure for diagnosis.

The incidence of brain tumors in the present study was much lower than in the previous reports. Giglio P et al[18] showed 6% of patients with brain tumors presented with symptoms less than one-day duration and were associated with hemorrhage into the tumor, developing obstructive hydrocephalus, vascular steal, or secondary mass effects on cerebral vasculature. The most common brain tumor in the present study was metastasis whereas another two were glioblastoma multiforme and meningioma, similar to the study of Hatzitolios A et al[2]. Contradicting with a large multicenter trial of the UK TIA study group[19], which revealed only 10 out of 2,499 patients with transient neurological dysfunction had intracranial neoplasm, half of them were malignant glioma whereas the other half had meningioma.

SDH was the most frequent condition among CNS space taking lesions in the present study. In elderly population, brain atrophy plays a role in the development of SDH, bridging veins can be sheared off by violent head motions even in the absence of head trauma. These lesions can have various clinical presentations, e.g., dementia or focal neurological

deficits[20]. Even though chronic SDH has been reported as a cause of stroke with TIA-like symptoms[21], there was no isolated chronic SDH in the present study. Interestingly, the investigators found a small number of patients with a clinically diagnosed acute stroke that eventually were CNS infection including cerebritis and brain abscess whereas the previous study had not been reported. Kocaeli et al[22] identified 11 of 116 cases of intracranial abscess with unusual presentations were related to rupture of the abscess, hemorrhage into the abscess, associated with arterial or dural sinus thrombosis.

The present study had a few number of limitations. The entry criteria may have been less restrictive. Stroke Code is activated for every patient who arrives with acute neurological deficit with irrespective time onset, severity of symptom or ultimate diagnosis, may be increasing the incidence of mimics. The investigator retrospectively identified final diagnosis by medical record reviews that depended on neuroimaging, laboratory result, and clinical impression, expressed by attending neurologists. Some discharge diagnosis may be mislabeled.

Conclusion

The present study provides the data support that several nonvascular conditions can be produced stroke-like clinical picture. The incidence and etiologies were similar to the western reports, however the frequency of each mimic was different. Even though small number of the patients with stroke mimics in the present study revealed CNS space taking lesion demonstrated by NCCT. Awareness of stroke mimics and proper investigations are essential to avoid inappropriate stroke management.

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Potential conflicts of interest

None.

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การวินิจฉัยภาวะอัมพาตเลียนแบบในกลุ่มผู้ป่วยอัมพาตเฉียบพลัน

โลจนา ตันติยาทร, พิชญา ศักดิ์ศรชัย, สุพจน์ ตุลยเดชานนท์

ภูมิหลัง: *โรคอัมพาตเฉียบพลัน (acute stroke) เป็นโรคที่เกิดจากการที่เนื้อสมองขาดเลือดอย่างฉับพลัน ทำให้เกิดอาการอัมพาตของร่างกายส่วนนั้น ๆ ภาวะอัมพาตเลียนแบบ (stroke mimics) เป็นกลุ่มอาการที่ไม่ได้มีสาเหตุจากภาวะสมองขาดเลือดแต่มีอาการทางคลินิกแบบเดียวกันซึ่งการรักษามีความแตกต่างกัน การตระหนักถึงภาวะอัมพาตเลียนแบบจะช่วยแพทย์หลีกเลี่ยงการรักษาที่ไม่เหมาะสมได้*

วัตถุประสงค์: *เพื่อศึกษาอุบัติการณ์และสาเหตุของภาวะอัมพาตเลียนแบบในผู้ป่วยที่ได้รับการวินิจฉัยทางคลินิกว่าเป็นโรคอัมพาตเฉียบพลัน*

วัสดุและวิธีการ: *เป็นการศึกษาแบบพรรณนาโดยเก็บข้อมูลย้อนหลังจากภาพถ่ายเอกซเรย์คอมพิวเตอร์สมองแบบไม่ใช้สารทึบแสง (non-contrast CT; NCCT) ในผู้ป่วยที่ได้รับการวินิจฉัยทางคลินิกว่าเป็นโรคอัมพาตเฉียบพลัน ซึ่งผู้ป่วยได้รับการตรวจ NCCT สมองทันทีหลังจากเข้ามาตรวจที่ห้องตรวจฉุกเฉินของโรงพยาบาลมหาวิทยาลัยแห่งหนึ่ง มีระยะเวลาที่ทำการศึกษาติดต่อกัน 12 เดือน การแปลผลภาพ NCCT สมองทุกรายกระทำโดยรังสีแพทย์ การวินิจฉัยขั้นสุดท้ายได้จากการทบทวนเวชระเบียน ผลการตรวจทางห้องปฏิบัติการ ผลการตรวจการตรวจด้วยเครื่องสร้างภาพจากคลื่นแม่เหล็กไฟฟ้าพื้นฐาน (MRI) การติดตามโดยภาพ CT หรือการติดตามอาการทางคลินิกอย่างน้อย 6 เดือน*

ผลการศึกษา: *มีผู้ป่วยทั้งสิ้น 704 ราย เป็นชาย 347 ราย (51.5%) หญิง 327 ราย (48.5%) ช่วงอายุ 24-108 ปี ได้รับการวินิจฉัยขั้นสุดท้ายว่าเป็นโรคอัมพาตจากการขาดเลือด 417 ราย (59.2%) จากภาวะเลือดออกในสมอง 80 ราย (11.4%) ภาวะอัมพาตเลียนแบบ 186 ราย (26.4%) และภาวะที่ไม่สามารถสรุปได้ 21 ราย (3%) สาเหตุของภาวะอัมพาตเลียนแบบเกิดจากโรคเมตะโบลิค หรือ การได้รับสารพิษ 35 ราย (18.8%) การติดเชื้อในกระแสเลือด 28 ราย (15.0%) การชัก 21 ราย (11.3%) เป็นลม 20 ราย (10.8%) ภาวะเลือดออกในเยื่อหุ้มสมอง 14 ราย (7.5%) เวียนศีรษะ 11 ราย (6.0%) เนื้องอกสมอง 10 ราย (5.3%) การติดเชื้อในสมอง 5 ราย (2.7%) และอื่น ๆ 26 ราย (14.0%) เช่น conversion disorder, myasthenia gravis, multiple sclerosis, posterior reversible encephalopathy syndrome และอื่น ๆ ที่ไม่สามารถจัดกลุ่มได้ 16 ราย (8.6%)*

สรุป: *อุบัติการณ์โดยรวมและสาเหตุของภาวะอัมพาตเลียนแบบไม่แตกต่างจากการศึกษาในประเทศตะวันตก แต่ความชุกของแต่ละสาเหตุนั้นแตกต่างกัน การตระหนักถึงภาวะนี้และการตรวจเพิ่มเติมเพื่อค้นหาสาเหตุจะช่วยหลีกเลี่ยงการรักษาที่ไม่เหมาะสมได้*