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Clinical and radiologic profile of transient global amnesia in a Philippine tertiary hospital

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Abstract:

BACKGROUND: Magnetic resonance imaging (MRI) has gained increased diagnostic utility for patients with transient global amnesia (TGA), particularly for unwitnessed events or those with diagnostic uncertainty based on clinical grounds.

OBJECTIVES: The objectives are first, to determine the demographic and comorbid conditions of TGA patients; second, to determine the percentage of MRI diffusion weighted imaging (MRI DWI) hippocampal lesions, their time relationship from symptom onset, and their morphological characteristics; and lastly, to determine the dementia visual rating scale scores on neuroimaging for these patients.

METHODS: A total of 20 TGA patients in a tertiary hospital from 2018 to 2022 were included in this retrospective study, and their medical records and neuroimaging were reviewed.

RESULTS: TGA patients had a mean age of 61.4 years and a female predominance. Prevalent comorbid conditions include hypertension, dyslipidemia, and diabetes, and the majority were discharged with antithrombotic medications. An emotionally triggering event was identified in 15% (n = 3). Mean symptom onset-to-scan time was 8.33 h, and one patient (detection rate of 5%) who underwent neuroimaging after 21.7 h demonstrated typical punctate hippocampal DWI hyperintensity. None exhibited significant cortical atrophy.

CONCLUSION: TGA patients showed female predominance, occurring mostly within the 5th–6th decade, with a moderate prevalence of vascular risk factors and absence of significant cerebral atrophy based on the Dementia Visual Rating Scales. A conventional MRI protocol yielded a 5% detection rate with a delay of 21 h from symptom onset. Hence, in a resource-limited setting such as the Philippines, it may be suggested, with limited evidence, that performing the procedure in TGA patients when the event is unwitnessed or uncertain could be reasonable, as correctly diagnosing TGA has therapeutic implications. Further studies may investigate prospectively the diagnostic utility of MRI, neuropsychological profile, and estimate cardiovascular and cognitive deterioration risk.

Keywords:

Diffusion-weighted imaging, punctate hippocampal diffusion-weighted imaging hyperintensity, transient global amnesia

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Introduction

Transient global amnesia (TGA) is characterized by the abrupt onset of severe anterograde amnesia with repetitive questioning, preserved sensorium, language, and orientation to self, with no lateralizing neurologic deficits and epileptiform features.[1] These are followed by gradual recovery after a few hours and a return to baseline.[1] The estimated annual incidence of TGA is between 3.4-10.4 per 100,000 people per year, with an estimated recurrence rate of 8%.[2,3] Multiple studies[2,4,5] have demonstrated that patients with TGA have a lower prevalence of vascular risk

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factors but a higher incidence of psychiatric disease[2] and migraine headache.[2] Although previously widely believed to be secondary to an ischemic event, making a diagnosis of TGA portends a good prognosis, as this subset of patients has not been shown to have an increased risk of developing stroke, seizures, or cognitive impairment.[2]

In the age of advanced neuroimaging, magnetic resonance imaging (MRI) has gained increasing diagnostic utility for patients with TGA, especially for those with diagnostic uncertainty or unwitnessed TGA events, showing the presence of punctate hippocampal hyperintensities on diffusion weighted imaging (DWI) with features distinct from posterior circulation infarctions[6-8] usually appearing within 1–3 days from symptom onset[3] Clearly differentiating an episode of TGA from an ischemic event or non-convulsive seizure is important as these conditions will have significant therapeutic and prognostic implications.

Currently, there is a lack of local data on the clinical and radiologic profile of TGA. This study may contribute to the body of knowledge on this enigmatic condition, increase awareness, resulting in proper and timely diagnosis, and subsequent appropriate management in the Filipino context. The study aims to determine the clinical and radiologic profile of patients with TGA, specifically to determine the demographic and comorbid conditions of TGA patients, and to determine the percentage of MRI DWI hippocampal lesions, its relation with time from symptom onset and its morphologic characteristics, and to determine the Dementia Visual Rating Scale Scores for these patients which may aid in the development of dedicated imaging protocols best suited to detect these.

Methods

Study population

This is a descriptive retrospective cohort study. The study population consisted of adult Filipinos (more than 18 years old) admitted in a Private Tertiary Hospital in the Philippines from January 2018 to December 2022. The sample population was obtained by reviewing the hospital's inpatient census and corresponding electronic medical records of the selected patients. Patients included were the following: (1) Patients who were clinically diagnosed with TGA by a neurologist within the study period, (2) patients who underwent Cranial MRI with DWI. Patients excluded were the following: (1) Patients who were initially diagnosed with TGA but whose diagnosis changed over the course of the hospital stay, (2) patients who were diagnosed with TGA but did not undergo Cranial MRI with DWI, (3) patients who were diagnosed with TGA who underwent Cranial MRI with Dwi but had done so in another institution precluding review of the images.

Data source and analysis

Data collected from the screened participants' medical records included basic demographic data, clinical profile, such as potential triggering events, and comorbid conditions.

The MRI protocol used for all patients was through a 1.5 Tesla MRI machine with a b-value of 1000 s/mm^2 and 5 mm slice thickness. Data on the participants' radiographic profile were also included, such as the presence of DWI hyperintensities and characterization of the size, shape, location, and laterality of these findings, and the measurement of symptom onset-to-scan time. Dementia visual rating scales were determined to evaluate for the presence and degree of cerebral atrophy. Scales used were the Global Cerebral Atrophy (GCA) Scale which determines the overall degree of atrophy over 13 different cerebral regions in each hemisphere; the Medial Temporal Atrophy (MTA) Scale which evaluates changes along the choroid fissure, temporal horn of the lateral ventricle, and the hippocampus; the Posterior Parietal Atrophy scale which evaluates changes along the parietal and occipital regions; the Fazekas scale which evaluates the size and confluence of white matter hyperintense T2 lesions; the presence of strategic infarcts located along areas such as in the association areas, paramedian and bilateral thalamic, inferior medial temporal lobe, and watershed territories along the superior frontal and parietal regions. The presence of other cerebrovascular findings was also noted. A neuroradiologist reviewed the MRI of all participants to allow for a more uniform analysis of these images, as these did not include dementia visual rating scales.

This study is mainly descriptive with no comparative group. The demographic and clinical data were summarized and presented through frequencies. The radiologic data are also presented through frequencies and descriptions of the imaging findings.

Ethics

The study was approved by the Institutional Review Board of the corresponding Private Tertiary Hospital in the Philippines.

Results

Demographic and clinical profile

The sample initially consisted of 30 potential cases of TGA; however, 5 did not undergo MRI, and 5 were eventually diagnosed with another medical condition (such as cerebrovascular disease, encephalopathy, posterior reversible encephalopathy syndrome, central nervous

system infection, and traumatic brain injury), necessitating exclusion.

Twenty TGA patients met the inclusion criteria, and the majority were female (70%). Most of the cases of TGA (80%, *n* = 16) were within 50–70 years old with a mean age of 61.4 years. All of the participants were right-handed. Most did not have any identified triggering events (75%, *n* = 15). An emotional triggering event was the most commonly identified factor (15%, *n* = 3) surrounding the occurrence of the amnesic episode, such as the death of a loved one or an argument with a family member. Other identified preceding circumstances were mental exertion or exhaustion (10%, *n* = 2), successive online business meetings, and playing mahjong. Common comorbidities noted were hypertension (60%, *n* = 12), dyslipidemia (55%, *n* = 11), and diabetes (35%, *n* = 7). About 15% (*n* = 3) had a previous history of stroke, while 5% (*n* = 1) were previously treated for seizure disorder. None of the participants was previously diagnosed with TGA. The mean number of days in the hospital was 3.21 days [Table 1].

Upon discharge, 45% (*n* = 9) of patients were sent home with either a newly-prescribed antiplatelet or anticoagulant, 20% (*n* = 4) with nootropics or neuroprotective agents, and 10% (*n* = 2) with medications for headache. Among those treated with newly-prescribed antithrombotic medications, 5 had a medical indication such as the presence of ischemic heart disease, previous history of stroke, or findings of clinically silent stroke on neuroimaging [Table 1].

Neuroradiologic profile

The MR protocol used for all participants was 1.5 Tesla, b-value of 1000 s/mm^2, with 5 mm slice thickness. Mean symptom onset-to-scan time is 8.33 h (range 2.9–21.7 h). The majority of patients (75%, *n* = 15) had MRI DWI done <12 h from symptom onset. Among the 20 patients diagnosed with TGA, only 1 patient had findings of punctate hippocampal MRI DWI hyperintensity [Figure 1] in the patient with the longest symptom onset-to-scan time in this study, which was 21.7 h. This patient was eating lunch prior to the event and developed repetitive questioning and disorientation, with no lateralizing neurologic deficits on examination, and subsequent return to baseline the following day. None of the patients had findings of extrahippocampal MRI DWI hyperintensities. About 25% (*n* = 5) of patients diagnosed with TGA had findings of a previous infarct.

The dementia visual rating scales for each patient were determined which showed that 90% (*n* = 18) of patients had GCA score of ≤1 with a mean GCA score of 0.65, 90% (*n* = 18) had a MTA score of ≤1 with mean MTA score of 0.3, 90% (*n* = 18) had a Fazekas score of ≤1 with

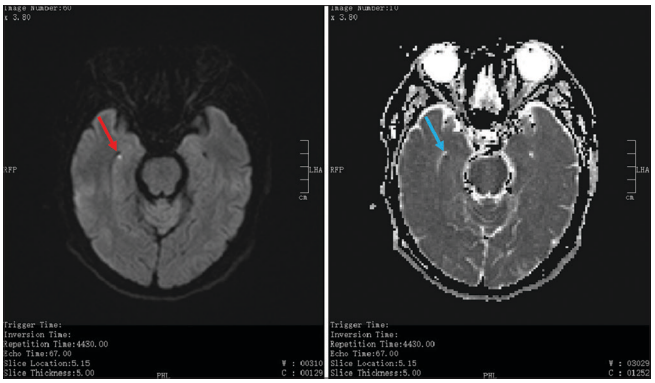

Figure 1: Punctate hippocampal diffusion weighted imaging (DWI) hyperintensity seen in a patient whose scan was done 21.7 h after symptom onset. A single round hippocampal DWI hyperintensity (red arrow) is seen on the right hippocampus measuring 0.4 cm in size, with a corresponding signal dropout on the analog-to-digital converter sequence (blue arrow)

Table 1: Demographic and clinical profile of patients diagnosed with transient global amnesia

Characteristics	Frequency, *n* (%)
Age (years), mean	61.4
Sex	
Male	6 (30)
Female	14 (70)
Triggering or precipitating factors or events	
Emotional	3 (15)
Mental exhaustion/exertion	2 (10)
Physical exertion	0
Temperature change	0
None identified	15 (75)
Comorbid conditions	
Hypertension	12 (60)
Dyslipidemia	11 (55)
Diabetes	7 (35)
Ischemic heart disease	3 (15)
Cardia dysrhythmia	2 (10)
Heart failure	0
Migraine headache	2 (10)
Previous stroke	3 (15)
Previous TIA	0
Previous TGA	0
Seizure disorder	1 (5)
Psychiatric disorder	1 (5)
Dementia	0
Other	7 (35)
Prescribed medications upon discharge	
Antithrombotic (aspirin, cilostazol, rivaroxaban)	9 (45)
Nootropic or neuroprotective agent (citicoline, piracetam)	4 (20)
Headache medication (paracetamol + orphenadrine, ibuprofen)	2 (10)
None	5 (25)

Other comorbid conditions: Bronchial asthma, allergic rhinitis, hypothyroidism, ovarian cyst, liver cirrhosis, hyperuricemia, schwannoma of the spine status post decompression and tumor excision, benign paroxysmal positional vertigo, and polycystic ovary syndrome. TGA: Transient global amnesia, TIA: Transient ischemic attack

a mean Fazekas score of 0.95, 90% (n = 18) had a Posterior Parietal Atrophy score of ≤1 with a mean PPA score of 0.55, and 90% (n = 18) had no strategic infarcts present on neuroimaging.

Discussion

To date, there has been no previous retrospective study describing the clinical and radiologic profile of patients diagnosed with TGA in the Philippines. Demographic findings in this study were consistent with previous publications showing female predominance, mean age across different studies of 61–67.3 years,[2,5,9] and occurring mostly within the 5th and 6th decades.

Collectively, different case reports demonstrated that approximately half of patients with TGA are associated with a precipitating event, mainly either physical stress or exhaustion, or emotional stress.[10] In this study, only a small proportion of the patients were identified to have a potential precipitating or triggering event surrounding the onset of the amnesic episode; relatively less in comparison to previous prospective and retrospective studies on TGA.[10] A high proportion of precipitating factors were identified in the study conducted by Quinette *et al*.,[10] reaching as high as 89.1% as they were also able to explore remote events occurring days or weeks before which they hypothesized could be a "general context of psychological pressure." These remote events might not have been elicited in this study and were also proposed by Quinette *et al*.[10] to be underreported as well as in previous studies.

Across different studies, the most commonly identified overall comorbid conditions among patients diagnosed with TGA were hypertension and dyslipidemia,[4,5,11-14] which is consistent with our findings. The prevalence of diabetes was variable in these studies, ranging from 7.2% to 36.1% which may reflect the geographic difference in the prevalence of this condition. Our data revealed that diabetes was the third most commonly identified comorbid condition among patients with TGA. Data from the Department of Science and Technology – Food and Nutrition Research Institute[15] showed that the incidence of elevated fasting blood sugar (>126 mg/dL) among Filipinos has been increasing over the past 2 decades and has more than doubled in a span of 7 years, from 4.3% in 2013 to 9.1% in 2019, which may explain the relatively high proportion of participants with diabetes in this study. The proportion of participants with comorbid migraine headache was also low, despite being considered the only factor significantly associated with an increased risk of TGA as compared to normal individuals.[9,10] It was also pointed out in previous studies that patients diagnosed with TGA had a higher incidence of psychiatric comorbidities[2,14] when compared to patients with transient ischemic attack (TIA). In our study, only one patient was diagnosed with a psychiatric disorder (Adjustment Disorder).

The exact etiology and pathophysiological mechanisms underlying TGA still remain unknown, with several proposed hypotheses. Based on similarities with other transient conditions, focal ischemic-hypoxic mechanisms on the hippocampus, abnormal cerebral venous outflow within the temporal lobes, cortical spreading depression, mesial temporal lobe seizures, and psychogenic mechanisms affecting the affective learning circuitry within the limbic system have been suggested.[16] However, to date, no strong evidence supports one mechanism over the other.[16]

Hodges and Warlow[1] in 1990 had already postulated that patients with TGA had a good prognosis, with only a low per annum rate of major vascular events at 0.6%. This was later confirmed by Mangla *et al*.[17] who determined that the cumulative 1-year rate of ischemic stroke in patients with TGA and TIA was 0.54% and 4.72% respectively; and by Romoli *et al*.[4] who, after a 7-year follow-up period on patients with TGA, found a pooled annual risk of stroke of 0.6%, not greater than the expected stroke risk in the general population, as long as the accompanying cardiovascular risk factors were being treated. Contrary to these findings, however, in a recent study by Lee *et al*.,[11] it was proposed that TGA patients with hypertension, diabetes, and atrial fibrillation had an increased risk for developing ischemic stroke but not hemorrhagic stroke, as these vascular risk factors are also independently established risk factors for ischemic stroke, although the annual risk for stroke was not determined. But consistent across multiple studies is that TGA had significantly lower prevalence of cardiovascular risk factors, future stroke risk, and other major cardiovascular events when compared to patients with TIA.[2,4,9-11,14,17] Despite this, the majority of patients included in the study were started on antithrombotic medication upon discharge as deemed appropriate by their respective attending neurologists, probably after individual assessment of vascular risk factors.

This study yielded 1 participant with punctate hippocampal DWI-hyperintensity, with a detection rate of 5%, without any accompanying extrahippocampal DWI hyperintensities, consistent with TGA. The absence of extrahippocampal DWI-hyperintensities would support the diagnosis of TGA rather than a stroke, as isolated hippocampal infarcts are rare,[6] and in a study conducted by Szabo *et al*.,[18] virtually all patients with infarcts involving the hippocampus also showed DWI hyperintensities of extrahippocampal tissue supplied by the posterior cerebral artery. Several factors have been identified that may increase the detection rate of

these punctate hippocampal DWI-hyperintensities, and these include delayed symptom onset-to-scan time,[7,8,19,20] increased magnetic field strength,[19,21] a slice thickness of 3 mm or less,[7,19-24] b-values of 2000 or 3000 s/mm^2,[7,19,22,24] and those presenting with recurrence of TGA.[25] Among these identified factors, only the symptom onset-to-scan time is variable in this study, as all patients underwent the same MRI protocol.

In terms of the symptom onset-to-scan time, maximum detection rates across multiple studies with varying slice thickness and b-values would range from 12 h[7] to 84 h[19] with a drop in the detection rate after 96 h and virtually undetectable by 192 h after symptom onset.[19] Among those studies with high detection rates (>70%), the presence of punctate MRI DWI-hyperintensities would maximally be seen within 48–72 h.[7,8,20-24] In a meta-analysis by Wong *et al*.,[6] the computed pooled DWI-MRI sensitivity for imaging done within 12 h for TGA is 15.6% which corresponds to 75% of participants in this study. The highest pooled sensitivity was for MRI DWI imaging done within 48–60 h, with 82.8%. In our current study, the only participant who yielded positive DWI findings had the longest symptom onset-to-scan time of 21.7 h. A study done in Korea[26] with a similar MRI protocol and median symptom onset-to-scan time of 6 h, but with a higher study population of 203 TGA patients, obtained a comparable detection rate of 7.9%. Cited possibilities as to why these findings are usually not seen within the first few hours from symptom onset are subtlety of the hyperintense signal during the hyperacute stage, and possibly delayed neuronal injury affecting the hippocampus, hence manifesting on MRI DWI at a later time.[27] Furthermore, a slice thickness of 5 mm is not ideal as hippocampal lesions seen in TGA are typically 1–3 mm in size, and this may increase the chances of missing it in between cuts.[24]

None of the study participants were previously diagnosed with dementia, nor were taking any medications used for dementia (e.g. Donepezil, Rivastigmine, Memantine). Overall, patients included in this study collectively did not demonstrate significant brain atrophy with mean atrophy scores of <1 across all dementia visual rating scales, and 90% of participants had a score of either 0 or 1. A previous small-scale study by Kwan *et al*.[28] in Hong Kong SAR, China demonstrated that patients with TGA had a significantly lower prevalence of subcortical and periventricular white matter hyperintensities, cerebral microbleeds, and medial temporal lobe atrophy as compared to age-matched cognitively normal controls, and 8% had an MTA scale of >1 which is comparable to the result in our study. However, radiologic assessment of global atrophy, posterior parietal atrophy, and the presence of strategic infarcts were not included in Kwan *et al*.'s[28] study.

Several previous studies have attempted to determine the cognitive sequelae of patients who have had an episode of TGA with mixed results. A meta-analysis of 25 studies addressing the recovery of cognitive function in TGA patients demonstrated that all-in-all there was no significant difference between TGA patients and controls in anterograde and retrograde long-term memory, executive function, short-term memory, and semantic memory 5–30 days after a TGA episode, concluding that it is not likely that TGA will lead to permanent memory changes down the line.[29] Likely reasons postulated for the variability of results across different studies are the heterogeneous tests used for assessment of different cognitive functions, unsatisfactory matching of patients and controls, and lack of information on prior cognitive performance prior to the development of TGA.[29]

There are several limitations for this study, namely the relatively small sample size, conducted in a single center, with a retrospective study design. In addition, the MRI specifications used, specifically the low b-value and thicker slice thickness, were less than ideal to maximize the yield of appreciating the punctate hippocampal DWI hyperintensities, which likely have contributed to the low detection rate in this study. Also, since there are no clear protocols or guidelines that guide clinicians on approaching a patient suspected of having TGA, the diagnostic workup to rule out confounding factors to explain the amnesic episode may differ from case to case. Finally, data on neuropsychological testing on included participants were lacking, and no follow-up data were available to correlate identified risks and events.

Conclusion and Recommendations

In conclusion, this is the first retrospective study on TGA in the Philippines as of writing, showing female predominance, occurring mostly in the 5th–6th decade with moderate prevalence of vascular risk factors, and absence of significant cerebral atrophy. In the local setting, migraine headache and psychiatric comorbidities were not prevalent, and most did not present with a triggering event. Typical punctate hippocampal DWI-hyperintensity may be detected by conventional MR protocols through delaying symptom onset-to-scan time by at least 21 h. Hence, in a resource-limited setting such as the Philippines, it may be suggested, based on limited evidence, that performing the procedure in TGA patients when the event is unwitnessed or uncertain could be reasonable, particularly if the patient arrives beyond the therapeutic window for thrombolysis. Correctly diagnosing TGA has therapeutic implications, has management of TGA is different from TIA and transient epileptic amnesia.

Further studies may prospectively investigate the diagnostic utility of DWI-MR imaging in the Philippine setting, explore the neuropsychological profile of TGA patients, and estimate the cardiovascular and cognitive deterioration risk in the local setting through a follow-up study.

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Nil.

Conflicts of interest
There are no conflicts of interest.

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