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ORIGINAL ARTICLE

# Hippocampal infarction: redefining transient global amnesia

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## Abstract

**Background and aims** Transient global amnesia (TGA) is a clinical syndrome characterized by sudden anterograde amnesia not accompanied by other neurological symptoms. There is no consensus on the underlying pathophysiological mechanism. However, diffusion-weighted imaging (DWI) of the magnetic resonance imaging (MRI) has demonstrated hippocampal lesions in as many as 50% of cases. This paper describes a series of patients with TGA and hippocampal lesions.

**Methods** This study assessed vascular risk factors in patients older than age 18 admitted to the Hospital Universitario San Ignacio, Bogota, Colombia, from May 2017 to June 2020 with a diagnosis of TGA and evidence of hippocampal ischemic lesion on 3 Tesla brain MRI.

**Results** The authors identified 36 patients, 72.2% female, with mean age 62 years. Cardiovascular risk factors, most frequently high blood pressure, carotid disease, and dyslipidemia, were present in 75% of these patients. Hippocampal lesions were unilateral in 80% of cases, with median size 2.5 mm, most frequently located at the hippocampal body. Approximately 14% of patients also presented acute ischemic lesions in locations other than the hippocampus.

**Conclusions** TGA is a clinical entity previously considered to have undetermined etiology. The present study used brain MRI to identify a group of patients with hippocampal ischemic lesions, finding associated vascular risk factors in a high proportion of them.

**Keywords** Amnesia · Diffusion-weighted images · Hippocampal stroke · Hippocampus · Ischemic stroke · Transient global amnesia

## Introduction

Transient global amnesia (TGA) is a clinical syndrome characterized by a sudden onset of anterograde amnesia, with repetitive questioning the most representative symptom. The inability to store new information stands out, though some degree of retrograde amnesia may also be present. A TGA episode may last for 24 h and is not accompanied by other neurological symptoms [1].

In 1956, Bender described repetitive questioning as a typical finding in patients with a "syndrome of isolated confusion with amnesia." In 1964, Fisher and Adams coined the TGA name [2]. In 1985, Caplan was the first to propose operational diagnostic criteria; these are currently used with a 1990 modification by Hodges and Warlow [3, 4].

Reported incidence of TGA is 3 to 8 persons in 100,000 inhabitants per year, with 75% of episodes presenting in persons age 50 to 70. Recurrence rate varies from 6–10% [5, 6]. There is no consensus on a TGA pathophysiological mechanism, though recently diffusion-weighted imaging (DWI) of the magnetic resonance image (MRI) has identified small lesions in the hippocampus in as many as 50% of patients [7, 8].

In clinical practice, the DWI sequence is used to detect cytotoxic edema, generally attributed to ischemia. The DWI sequence is highly sensitive to cytotoxic edema, though it lacks specificity for ischemia, given that cytotoxic edema has been found in other conditions, such as hypoglycemia, epileptic crisis, and cortical spreading depression wave (characteristic of migraine patients) [5]. Although the finding of cytotoxic edema does not suffice to detect a common pathophysiological mechanism, it has allowed a deeper study

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of the hippocampus as a structure involved in TGA development. The CA1 zone of the Amon horn is of special interest, as it contains neurons that are selectively vulnerable to metabolic stress [8].

This paper describes a series of patients with TGA diagnosis and associated hippocampal infarction represented by MRI lesions in DWI sequence as equally as in apparent diffusion coefficient (ADC) and FLAIR sequences, ratifying its ischemic nature (Fig. 1).

## Methods

This study included patients older than age 18 admitted to the emergency room of the Hospital Universitario San Ignacio from May 2017 to June 2020. These patients had a TGA diagnosis according to 1990 criteria of Hodges and Warlow [4] and ischemic hippocampal lesion demonstrated in the brain MRI in DWI, ADC, and FLAIR sequences. Patients with TGA without hippocampal lesion were excluded. It was the first episode of TGA for all patients. The study was approved by the institutional Ethics Committee and under its guidelines. An informed consent was not required.

Patients were initially assessed by an emergency room doctor and subsequently by a neurologist. All patients had a complete blood count, glucose test, electrolytes test, and brain MRI. Also, all patients were studied for carotid disease with neck and brain vessel Doppler ultrasound or angiotomography. (Carotid disease was defined as the presence of any atherosclerotic plaque, regardless of the degree of stenosis.) Cardiac sources for embolism were studied with transthoracic or transesophageal echocardiography. A neuroradiologist assessed all brain imaging tests. The study used a 3 Tesla MRI with a 16-channel antenna. Techniques for radiofrequency pulse sequences included SE, TSE, FE, and IR.

Researchers collected data retrospectively using a standardized questionnaire that identified demographic variables and characteristics of the amnesia episode (length of the event, recurrence, and physical/emotional triggers). Data on comorbidities and cardiovascular risk factors were selected

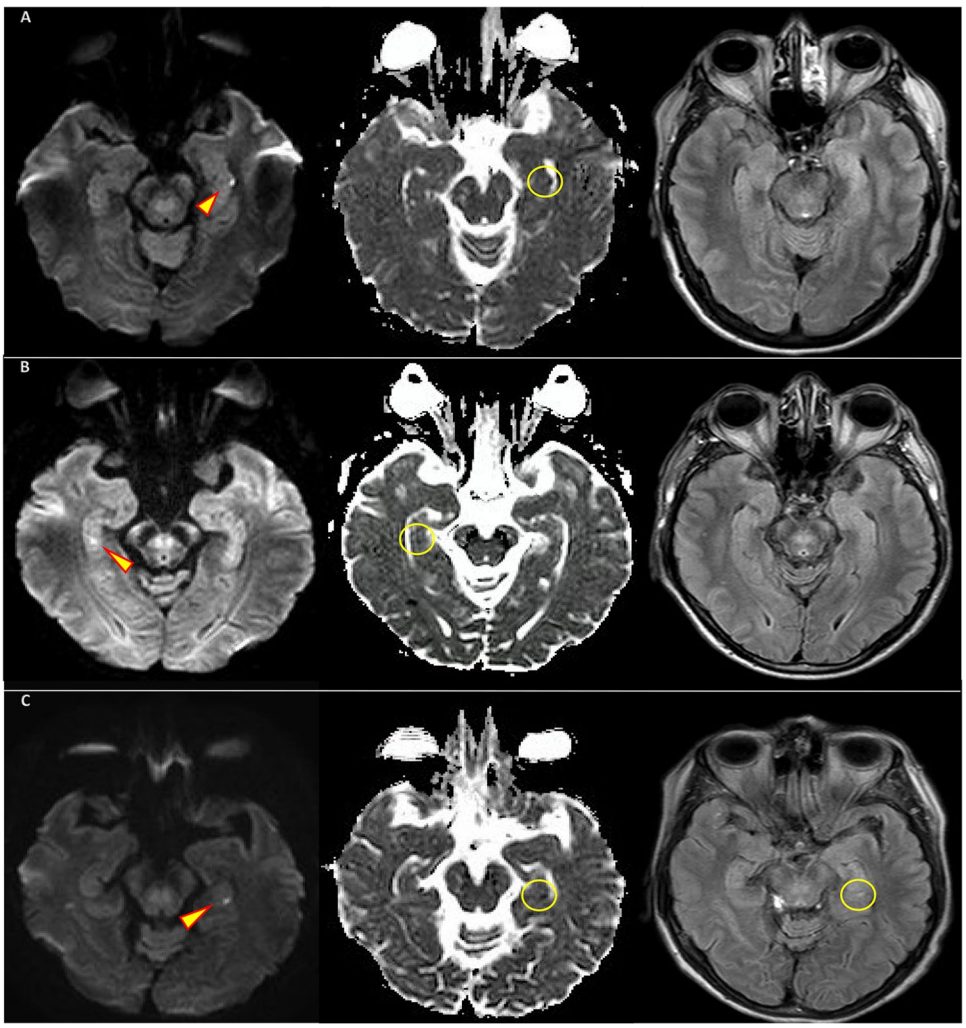


**Fig. 1** Axial MRI sequences DWI, ADC y FLAIR, **A** left hippocampal lesion without FLAIR representation, **B** right hippocampal lesion without FLAIR representation, and **C** Left hippocampal lesion with FLAIR representation

according to the definitions established in the research protocol. The ASCVD (atherosclerotic cardiovascular disease) scale [9] was used to calculate cardiovascular risk. The authors assessed the imaging characteristics of the hippocampal infarction by lesion size (which was assessed by the neuroradiologist with Kanteron System's tool), laterality, multiplicity, location in the hippocampus, concomitance of cingular infarctions, and Fazekas classification [10], in the case of leukoencephalopathy.

A univariate analysis described the categorical variables as percentages, and the qualitative variables according to their normality distribution, assessed with the Shapiro–Wilk test. Variables with normal distribution were described as mean and standard deviation, and variables with non-normal distribution were described with median and interquartile range. Data were analyzed using the SPSS ® software Version 25 for Mac OS.

## Results

There were forty-five patients with TGA diagnosis in total from which thirty-six patients fulfilled inclusion criteria. These were 72.2% female, with a mean age of 62 years, and 75% had 1 or more cardiovascular risk factors, most frequently high blood pressure, carotid disease, and dyslipidemia (Table 1). The most frequent risk factor in females was high blood pressure; in males, dyslipidemia.

Median duration of symptoms was 7 h. In 83% of cases, no physical or emotional triggers were documented. There was a clear trigger only in 2 cases. The majority of events presented during morning hours (6 a.m.–noon).

**Table 1** Demographics and cardiovascular risk factors

| | *n* (%)<br>*n* = 36 |
|---|---|
| Age (years)* | 62.1 ± 8.5 |
| Female | 26 (72.2) |
| Cardiovascular risk factors | |
| Dyslipidemia | 15 (41.7) |
| Carotid disease | 9 (25) |
| High blood pressure | 13 (36.1) |
| Tobacco use | 2 (5.6) |
| Hypothyroidism | 5 (13.9) |
| Migraine | 2 (5.6) |
| Diabetes | 1 (2.8) |
| Coronary disease | 2 (5.6) |
| Atrial fibrillation | 0 (0) |
| Stroke | 2 (5.6) |

*Mean and SD

The laboratory tests demonstrated no alteration in electrolytes or glucose levels in any patient. A third of patients, however, had neutrophilia, half of these without leukocytosis (Table 2). The median size of hippocampal lesions was 2.5 mm (Table 3). In 80% of cases, the lesion was unilateral, most on the left side. The most frequent location was the hippocampus body, and in 2 cases, the lesions were in the anatomical limit between the hippocampus head and body.

**Table 2** Magnetic resonance imaging

| | *n* (%)<br>*n* = 36 |
|---|---|
| Lesion size (mm)* | 2.5 (1) |
| Side | |
| Left | 20 (55.6) |
| Right | 9 (25) |
| Bilateral | 7 (19.4) |
| Multiple lesions | 12 (23.3) |
| Cingular concomitant lesion | 1 (2.8) |
| Location | |
| Head | 16 (40) |
| Body | 21 (52.5) |
| Tail | 1 (2.5) |
| Head-body boundary | 2 (5) |
| Fasekas classification | |
| 0 | 28 (77.8) |
| 1 | 8 (22.2) |
| 2 | 0 (0) |
| 3 | 0 (0) |

*Median and IQR

**Table 3** Event characteristics and laboratory test results

| | *n* (%)<br>*n* = 36 |
|---|---|
| Time of onset | |
| Morning | 25 (69.4) |
| Afternoon | 6 (16.7) |
| Night | 5 (13.9) |
| Triggering events | |
| Physical | 3 (8.3) |
| Emotional | 3 (8.3) |
| Duration (hours)* | 7 (7) |
| Laboratory test results | |
| Hypoglycemia | 0 (0) |
| Electrolyte imbalance | 0 (0) |
| Leukocytosis | 5 (13.9) |
| Neutrophilia | 10 (27.8) |
| Recurrence | 3 (11.1) |

*Median and IQR

Approximately 14% of patients had acute ischemic lesions in places other than the hippocampus. In 52.8% of cases, the MRI showed white matter hyperintensities that might represent small vessel disease. The ASCVD scale documented middle or high cardiovascular risk in more than 50% of patients.

## Discussion

This observational, descriptive, cross-sectional study included 36 adult patients with TGA and hippocampal infarction in a referral hospital in Bogotá D.C., Colombia, in a 3-year period. Of these patients, 75% had one or more cardiovascular risk factors. Only in 16% of patients was a physical or emotional trigger identified, and the only relevant laboratory finding was neutrophilia. Median size of the hippocampal lesion was 2.5 mm, and more than 50% of patients had middle or high cardiovascular risk documented with the ASCVD scale.

Pathogenic mechanism of TGA was not fully described, although MRI has documented a signal abnormality in the DWI sequence of the hippocampus [7] in approximately 50% of patients that met Hodges and Warlow diagnostic criteria (Fig. 1). The DWI sequence is highly sensitive for detection of cytotoxic edema (generally attributed to ischemia), though it lacks specificity, given that the same edema has been found in other conditions, such as hypoglycemia, epileptic crisis, and in the cortical spreading depression wave in patients having migraine with aura [6]. The cytotoxic edema does not sufficiently indicate a common pathophysiologic mechanism, though it has allowed a deeper study of the hippocampus, most specifically the CA1 area, as a structure involved in TGA development. The CA1 area has neurons that are very vulnerable to metabolic stress, specifically in the context of hypoxia and ischemia [11]. This vulnerability may be explained by a sensitivity to glutamate cytotoxic release and trapping, or by the vascular anatomy of the hippocampus that may encompass a boundary zone between two principal arteries [12].

The TGA occurs in an estimated 5.2 to 10 persons per 100,000 each year, and incidence increases after age 50 [5, 13]. In this study, 72.2% of patients were female, though most studies indicate no significant differences in TGA occurrence by gender [13–15]. Mean age of presentation was 62 years, similar to other reports [6, 16, 17]. Some studies suggest that female gender is a risk factor for TGA recurrence, though in this cohort only 1 woman (of a total of 3) had a recurrence [18].

More than 70% of patients in this series had at least a classic cardiovascular risk factor, most frequently dyslipidemia, blood hypertension, and carotid disease. Also, 85% of patients with documented carotid disease had dyslipidemia concurrently, a percentage similar to that reported in a retrospective study in Poland [19].

A study based in a national sample of in-hospital patients in the USA determined that patients with TGA have almost twice the probability of high blood pressure and three times the probability of dyslipidemia compared to the general population of hospitalized patients [20]. Several other studies, however, found no differences between TGA patients and controls in the prevalence of cardiovascular risk factors, based on age and gender [13, 21, 22].

A case–control study compared 293 TGA patients, 632 patients with transient ischemic attack (TIA), and 293 controls paired by gender and age. The study found that patients with TGA have a higher probability of TIA, ischemic cardiomyopathy, and dyslipidemia. Patients with TGA compared to patients with TIA, however, have a lower probability of diabetes, blood hypertension, atrial fibrillation, and ischemic stroke [23, 24]. The authors of the present study consider that the strategies of primary and secondary prevention should be similar in patients with TGA and TIA, since the long-term effects of TGA are not clearly known. A cohort study demonstrated that TGA does not confer a higher risk for stroke or major cardiovascular events when cardiovascular risks are treated by primary prevention guidelines [25]. Several studies [13, 20, 26] report migraine as a factor associated with TGA. The present study, however, documented migraine in less than 10% of patients.

Among the triggering events, Valsalva's maneuver is the most frequent [6]. It is believed that the maneuver increases intrathoracic pressure and reduces blood return to the heart. This increases brain venous pressure, with hippocampal vein congestion and further ischemic injury [27, 28]. Also, many patients with TGA have been found to have incompetent jugular valves, though this factor is not universally accepted as a triggering event due to the lack of high rates of recurrence in these cases [28]. The literature reports physic or emotional events preceding a TGA episode in 30–90% of patients [6, 21]. A study in Ferrara, Italy, documented that highly urbanized places had more TGA cases. Such an increase may be directly related to stress levels in daily life [29]. The present study documented only 2 cases of a physical or emotional triggering event. Such variability in results may be due to lack of a systematic evaluation in the anamnesis from patient to patient.

In this series, 55.5% of patients had symptoms onset between 6 a.m. and noon, similar to results in several other reports in the literature [6, 24]. Some studies report a peak of presentation around 8 a.m. [24]. A theory for TGA pathophysiology proposes that metabolic stress may induce a high cortisol level that would directly affect the hippocampus function [30]. Cortisol peak is known to occur between 6 and 8 a.m., which may explain why most TGA cases present at that time. Also, several studies have found increased

cortisol in saliva during TGA episodes. This may support the theory that stress plays an important role in the pathophysiology of TGA [30, 31].

No changes were documented in electrolytes and serum glucose in any patient. A few studies have assessed these variables, and some case reports have shown a relationship between hypokalemia and TGA [32]. On the other hand, 15% of patients had leukocytosis and 27% had neutrophilia, similar to results of a retrospective study by Guohong Li with 203 patients [33]. The role of polymorphonuclears in TGA pathophysiology is not clear; it is proven, however, that the brain responds to ischemic injuries with acute and prolonged inflammation. Such inflammation is characterized by a fast activation of microglia, production of pro-inflammatory factors, and infiltration by several inflammatory cell types [34]. There is also a relationship between early neutrophilia and the volume of ischemic tissue in acute ischemic strokes [35].

Clinical symptoms of TGA suggest that the primary sites of neurological involvement are the medial areas of the temporal lobe and the hippocampus. (These areas play a role in the formation and evocation of the episodic memory [5].) This study documented hyperintense punctiform lesions in the DWI sequence and a mean size for hippocampal infarction of 2.5 mm. These results are similar to those in other studies [16, 36]. In this study, 55.6% of patients presented left hippocampal lesions, as in most other studies reviewed by the authors [13, 16, 37]. More studies are required, however, to determine the cause for such superiority. Conversely, 13.8% of patients simultaneously had acute or subacute ischemic lesions outside the hippocampus, mainly in the temporal, occipital, and frontal lobes. The majority of microinfarctions were located in the hippocampal body. As the scope of such findings is currently unknown, more research is required to better understand underlying pathophysiological mechanisms.

Some techniques have been reported to enhance the visibility of lesions in the MRI, such as the use of high-resolution DWI sequences, thin cuts (2–3 mm), and waiting 48–72 h between symptoms onset and radiologic study [38]. In this study, however, the median time between symptoms onset and MRI was 20.3 h. Some studies suggest that the time for lesions to appear ranges from 24 to 48 h [39].

There were incipient findings of small-vessel disease in 52.8% of patients. Some studies have shown a strong association between the hyperintensities in the white matter and cognitive deterioration in patients with subcortical vascular dementia [40]. Other studies suggest that even with prompt resolution of symptoms, TGA effects may be lengthier and less benign than previously thought. One study showed that a year after their TGA episodes, 55 patients performed worse in neuropsychological tests of memory and attention than a group of 80 controls of the same age. Also, one third of patients with TGA fulfilled criteria for mild cognitive deterioration [13]. More studies with long-term follow-up are required for definitive conclusions.

## Conclusions

The TGA has historically been considered to have a functional nature and good prognosis, and a deeper etiologic study was not considered necessary. The present study, using MRI systematically in the emergency room, identified a high prevalence of ischemic injuries in the hippocampus. The descriptive analysis of the TGA cases found a high proportion of patients with associated vascular risk factors, such as blood hypertension, dyslipidemia, and carotid disease. Another relevant finding of this study, consistent with the literature, is the presentation of this clinical condition in morning hours, supporting the pathophysiologic theory regarding cortisol secretion.

The authors propose that brain MRI technique using thin sections from the temporal lobe and using other planes for hippocampus visualization would increase neuroimaging efficiency, improving the probability of finding a hippocampal lesion in TGA patients. In the future, the long-term sequelae of hippocampal amnesia as a predictor for stroke and for cognitive deterioration must be studied.

Patients with TGA should be advised to lose weight, discontinue tobacco use, remain compliant with their medications, and refrain from alcohol use [27].

## Limitations

A small sample is among the one limitation of this study as well as the retrospective design and the lack of a control group; comparison with a group in which ischemic lesions are absent is suggested. Also, the ASCVD scales used to measure cardiovascular risk are not validated in Colombia. It is, however, the only scale available to measure risk of stroke. Further studies should include MRI follow-up data and timing of MRI scanning.

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## Declarations

**Ethical approval** None.

**Informed consent** Informed consent was obtained from all individual participants included in the study.

**Conflict of interest** The authors declare no competing interests.

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