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Transient global amnesia – current state of knowledge

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Abstract

Introduction and purpose

Transient global amnesia (TGA) is a sudden, short-lasting episode of anterograde and retrograde memory loss not caused by stroke or epileptic seizure. It affects 3.4-10.4 per 100,000 people per year, especially those in their 70s. There are several hypotheses regarding the etiopathology of this disorder, none of which have been unequivocally confirmed. This study aims to review the current knowledge about TGA, and to show its essence to the wider community.

Description

TGA is mainly manifested by a sudden, up to 24-hour loss of anterograde and retrograde memory, which may be accompanied by mild neuropsychological deficits. Diagnosis is based on a neurological examination and exclusion of numerous possible differential diagnoses, including laboratory and imaging tests. There is no specific treatment for this condition, and patient care is based primarily on patient education and risk factor control. They rarely recur and do not cause long-term complications.

Summary

Although TGA is considered a benign condition, its sudden onset and the temporary nature of memory loss can cause significant anxiety for both patients and their families. Increased awareness and prompt diagnosis are essential to provide reassurance and to rule out more serious conditions such as stroke or epilepsy. Expanding research on TGA is crucial to uncover potential preventive strategies and enhance our understanding of transient memory dysfunctions.

Keywords: transient global amnesia; TGA; amnesia; TGA etiopathology; transient global amnesia treatment; transient global amnesia diagnostics.

Introduction and purpose

Transient global amnesia (TGA) is a clinical syndrome characterized by the sudden onset of anterograde amnesia and retrograde amnesia, lasting up to 24 hours [1, 2]. Mild neuropsychological deficits with accompanying vegetative symptoms may persist for several days after the episode [3]. The clinical picture of TGA includes disorientation for time, with patients frequently asking repetitive questions about events from the current day. Autopsychic orientation remains intact, although allopsychic orientation may be impaired [1, 2]. TGA exhibits significant diurnal rhythm, peaking in the morning and again in the afternoon [4]. Episodes typically do not recur. However, studies indicate that about one-quarter of patients may experience a second attack in their lifetime, with very few experiencing more than two episodes in their lifetime [5].

The etiology and pathophysiology of TGA are not fully understood [3, 6, 7]. Several theories suggest potential mechanisms, including vascular (arterial and venous), epileptic, migraine-related, and psychogenic origins [1].

Vascular mechanism includes arterial ischemia and venous stasis.

The arterial ischemia hypothesis is based on similarities in clinical presentation and demographic features between TGA and transient ischemic attack (TIA). However, there are studies that argue against the ischemic hypothesis. They indicate differences in the clinical

presentation of patients with TGA and TIA, such as the absence of focal neurological deficits in TGA patients and a longer average duration of TGA episodes (4-8 hours) compared to TIA episodes (<1 hour). Additionally, no correlation has been found between risk factors for ischemic stroke and TGA occurrences. There are also no abnormalities observed in intracranial magnetic resonance angiography during TGA episodes [3, 6].

The venous stasis hypothesis posits that activities similar to the Valsalva maneuver preceding TGA can increase chest pressure, leading to retrograde pressure in the brain's venous vessels and venous stasis. However, supporting evidence for this theory is limited. Furthermore, no association was observed between TGA and other causes of venous stasis, such as congestive heart failure [3, 6].

Epileptic Mechanism.

The hypothesis suggesting that TGA results from epilepsy stems from observations that focal seizures in the temporal lobe can cause transient memory disturbances resembling TGA episodes. Nonetheless, no epileptic abnormalities have been noted during or after TGA episodes [6].

Migraine Mechanism.

Research indicates that individuals suffering from migraines are more prone to TGA [6,8]. This theory proposes that extensive cortical depression during a migraine episode may spread to the hippocampus, causing transient dysfunction and TGA. However, there is insufficient evidence supporting this theory, and patients with TGA do not typically show signs of acute migraine attack or migraine with aura during episodes [3, 6].

Psychogenic Mechanism.

The psychogenic hypothesis points to emotional stress as a triggering factor for TGA episodes. Physiologically, stressful events can adversely affect the emotional learning circuitry involving the amygdala, hippocampus, striatum, and prefrontal cortex. Transient hippocampal dysfunction observed in TGA may disrupt its inhibitory influence on the amygdala, consequently affecting memory formation processes. However, research validating this hypothesis is lacking [3, 6].

Recent studies emphasize the role of hippocampal dysfunction in the pathogenesis of TGA. It has been noted that CA1 neurons' susceptibility to metabolic stress is crucial in understanding the disease's pathophysiology. Diffusion-weighted MRI has shown focal changes in the hippocampus of TGA patients [3, 6].

Interestingly, since the onset of the COVID-19 pandemic, there has been an observed increase in TGA incidence. It remains unclear whether this rise is directly linked to viral infection or related to social distancing measures during lockdowns, uncertainty about the future, and fear of infection [9, 10].

The aim of this study is to present the current state of knowledge regarding the disorder known as transient global amnesia, its potential etiology, symptoms, diagnosis, and treatment. This is an extremely important topic that still requires further research.

Material and methods

The review was based on the analysis of materials collected in the „PubMed" and Google Scholar. The following keywords were entered during the search for scholarly articles: transient global amnesia; TGA; amnesia; TGA etiopathology; transient global amnesia treatment; transient global amnesia diagnostics. A total of 27 articles published between 1990 and 2024 were considered for the study and verified for their relevance to the topic of transient global amnesia.

Epidemiology

The incidence of Transient Global Amnesia (TGA) in the general population is 3.4–10.4 per 100,000 people annually, and it increases to 23.5–32 per 100,000 people annually for individuals aged 50 and older. Most episodes occur in people aged 50 to 80, particularly during the seventh decade of life. The average age of onset for episodes ranges from 61 to 67.3 years. TGA occurs with equal frequency in both genders [1, 3].

Episodes of TGA are often preceded by a stressful physical or psychological event. Triggering events include acute illness, medical procedures, significant physical exertion, or the Valsalva maneuver. Emotional triggers can be either positive or negative [2].

A significant risk factor for TGA is migraine. Patients with migraines are at a higher risk of developing TGA compared to matched controls, with an incidence rate ratio of 2.48. No connections have been established between different migraine subtypes and TGA [1, 3]. Additionally, increased occurrences of episodes have been observed in patients with coronary artery disease and hyperlipidemia, but not in those with a history of ischemic stroke, atrial fibrillation, hypertension, or diabetes. Individuals with a history of two TGA episodes exhibited a higher incidence of carotid artery atherosclerosis and coronary artery disease compared to those with just one episode [1, 3].

Symptoms

TGA clinically typically presents as a sudden impairment of memory, characterized by a complete inability to form new memories (anterograde amnesia) and variable retrograde memory impairment (retrograde amnesia). Patients often exhibit repetitively asked numerous questions such as "Where am I?", "How did I get here?", and "What happened to me?". Typically, they do not recall events preceding the episode but retain distant memories that precede the period of retrograde amnesia. Motor function, sensory perception, and higher cortical functions such as language, visuospatial skills, reasoning, and abstract thinking remain intact. Patients remain cooperative and can perform previously learned tasks [1, 11, 12].

During an episode, mild autonomic symptoms may occur, including headache, nausea, and dizziness. Occasionally, witnesses report recent physical exertion, sudden temperature changes (e.g., hot baths followed by cold showers), sexual activity, or significant stress without loss of consciousness [11, 13, 14].

The episode typically occurs with a peak between 10 and 11 AM and a secondary peak between 4 and 5 PM, resolving within several hours, often between 4 to 6 hours, and always less than 24 hours as patients gradually begin to form new memories. The exact moment of resolution is challenging to pinpoint and leaves a persistent gap in memory [1, 4, 11, 12].

Diagnosis

The diagnostic criteria for TGA were established by Hodges and Warlow in 1990 (Table 1). To diagnose TGA, all these criteria must be met. While these criteria are currently utilized in clinical practice, there is a recognized need for their updating and enhancement. They do not account for the potential occurrence of retrograde amnesia, which frequently accompanies TGA episodes. Furthermore, available data suggest that TGA may also affect other cognitive functions, such as executive abilities and visuospatial perception, aspects that are not included in the current criteria. There are also challenges in distinctly differentiating TGA from other acute amnestic syndromes caused by ischemic incidents, hypoxia, migraines, or toxic exposures [3, 11, 15].

Table 1- Diagnostic criteria for transient global amnesia (Hodges JR and Warlow CP 1990) [16]

Main Diagnostic Features of TGA
Attack must be witnessed
There must be anterograde amnesia during the attack
Cognitive impairment limited to amnesia
No clouding of consciousness or loss of personal identity
No focal neurological signs/symptoms or epileptic features
Attack must resolve within 24 h
No recent head injury or active epilepsy

The diagnosis and treatment of a patient suspected of having TGA should involve an interdisciplinary team, including at least a neurologist, internist, radiologist, and nurse, to effectively rule out numerous differential diagnoses. The foundation of the diagnostic process is a thorough neurological examination aimed at excluding any neurological deficits or signs of head injury that could suggest an alternative diagnosis. A detailed history must be collected from the patient and witnesses present during the episode to ascertain its characteristics. Key questions should focus on the duration and nature of memory disturbances, the presence of additional cognitive impairments, potential triggering factors, the patient's medical history, and overall health status. Those responsible for ordering additional tests should limit them to only what is necessary, avoiding excessive diagnostics [1, 7, 11].

While no laboratory tests can definitively confirm TGA, basic diagnostic tests are recommended to exclude potential differential causes. The standard range of tests should include blood glucose levels (as hypoglycemia can lead to memory deficits and should be included in the differential diagnosis) and electrolyte levels. A complete blood count with differential and a comprehensive metabolic panel—including liver function tests, C-reactive protein, ammonia levels, erythrocyte sedimentation rate, serum ethanol levels, and thyroid-stimulating hormone—should also be considered [1, 2, 11, 15].

Transthoracic echocardiography may be useful for patients with TGA presenting with elevated blood pressure upon admission. This can help assess septal hypertrophy as an indicator of chronic hypertension and determine left ventricular ejection fraction. Septal hypertrophy

(thickness >9 mm in women and >10 mm in men) aids in differentiating chronic hypertension from acute hypertension, informing decisions regarding antihypertensive medication for newly diagnosed patients [11].

Electroencephalography (EEG) is relevant in cases where clinical diagnosis is uncertain, primarily to exclude Transient Epileptic Amnesia (TEA). TEA may present as temporal lobe seizures or complex partial however, unlike TGA, they typically have shorter durations and recurrent episodes and may be accompanied by other symptoms such as oral automatisms or olfactory/gustatory hallucinations. EEG results usually show no abnormalities in TGA patients except for occasional nonspecific theta and delta waves [1, 2, 11, 15].

Magnetic Resonance Imaging (MRI) is a crucial tool in diagnosing TGA, particularly for confirming the diagnosis and excluding other potential causes such as stroke or transient ischemic attack (TIA). Imaging results vary depending on when the scan is performed, which is critical for diagnostic effectiveness. During an acute TGA episode, MRI findings often show no significant abnormalities; however, characteristic changes appear between 24 to 72 hours after symptom onset. These changes include punctate hyperintense lesions on diffusion-weighted imaging (DWI) and T2-weighted sequences primarily localized in the hippocampus, particularly in the CA1 sector known as Sommer's sector. Detection rates for hippocampal changes were 42% for the left hippocampus, 37% for the right, and 25% for bilateral changes. The average diameter of these lesions typically ranges from 1 to 5 mm. The detection rate significantly increases within 24–96 hours post-episode, reaching sensitivities between 67% to 85%, underscoring the importance of timely imaging. After 7–10 days, these changes become undetectable, indicating their transient nature [1, 2, 3, 11, 15, 17, 18, 19].

Differential diagnosis

The differential diagnosis of TGA is extensive and includes conditions such as TIA, stroke, seizures, postictal state, migraine, dissociative disorders or psychogenic amnesia, post-traumatic amnesia, concussion, metabolic or infectious encephalopathy, delirium, benzodiazepine or alcohol intoxication, encephalitis, and metabolic disturbances such as hypoglycemia. Most of these diagnoses can be ruled out after a thorough analysis of the patient's clinical condition and a comprehensive physical and neurological examination [2, 15].

Ischemic or Hypoxic Events

Ischemic stroke and TIA rarely present with isolated amnesia. A characteristic imaging feature of TGA is the reversibility of changes on DWI and the absence of permanent alterations in T2-

weighted and FLAIR sequences. This differentiates TGA from ischemic strokes, where changes are permanent, involve larger areas of the brain, and are associated with irreversible tissue damage. The ischemic nature of changes may suggest involvement of the hippocampus, thalamus, or structures in the medial temporal lobe (MTL), complicating differential diagnosis. In atypical cases of TGA or when clear symptoms are absent, further diagnostics such as MRI angiography may be necessary to evaluate the condition of cerebral vessels.

The etiology of hippocampal strokes manifesting as amnesia is most often cardioembolic or related to large vessel disease in the vertebrobasilar system. Patients with ischemic amnesia are typically older, have vascular risk factors, and lack emotional triggers. They often present with focal neurological deficits and other cognitive symptoms such as mild anomia or executive deficits.

TGA can also be triggered by vasospastic events, the use of contrast agents, or vascular procedures. In patients presenting with amnesia symptoms alongside cardiovascular changes (e.g., asymmetric blood pressure), aortic dissection should be considered as it may present similarly to TGA. In these cases, CT angiography is indicated to rule out this possibility [2, 3, 11, 15, 17].

Transient Epileptic Amnesia (TEA)

TGA is characterized by single episodes lasting several hours, predominantly involving anterograde amnesia with partial retrograde amnesia, accompanied by anxiety and repetitive questioning. TEA consists of recurrent short episodes (<1 hour), often occurring in the morning, which may include both forms of amnesia. TEA presents with symptoms associated with the temporal lobe, such as olfactory hallucinations, déjà vu, oral automatisms, and autobiographical and episodic memory deficits that may persist between episodes. Diagnosis of TEA relies on EEG findings; however, EEG can be normal in 30-43% of patients. TGA rarely recurs and resolves spontaneously without lasting complications, whereas TEA has a high recurrence rate and requires antiepileptic treatment that significantly improves outcomes. Differences in duration, accompanying symptoms, and treatment response are critical for distinguishing between these two disorders [3, 11].

Migraine

Migraines and TGA share common features such as episodicity and triggering factors. Some studies have indicated that TGA may occur in association with migraines as an aura or during severe headache attacks. A French retrospective study involving 8,821 migraine patients identified six cases of TGA occurring during migraines accompanied by intense vomiting. It is

suggested that the Valsalva maneuver associated with vomiting leads to elevated venous pressure in the brain, potentially triggering TGA. Although migraines and TGA are connected by their transient nature and neurological symptoms, their underlying mechanisms may differ.The relationship between them remains ambiguous; studies show both instances of co-occurrence and a lack of significant correlations [3, 11].

Dissociative Amnesia

TGA differs from dissociative amnesia (DA) in terms of duration and underlying causes. TGA resolves spontaneously within hours, while DA—resulting from intense psychological stress—can last days or longer. DA is characterized by retrograde loss of autobiographical memory often without anterograde amnesia and typically affects individuals aged 20-40 years. It may be preceded by psychological or physical trauma, with patient responses ranging from anxiety to indifference. If distinguishing between TGA and DA proves challenging, neurological and neuropsychological testing may be helpful in clarifying the diagnosis [2, 3, 11].

Treatment

Treatment for TGA primarily focuses on patient care, as there is no specific therapy required or available. The cornerstone of care involves thorough patient education regarding the condition. It is essential to emphasize the benign and typically isolated nature of TGA, along with its favorable prognosis, which generally does not lead to long-term consequences. Patients must be informed about the necessity of conducting comprehensive examinations during an acute episode to confirm the diagnosis and exclude other causes of amnesia, such as stroke, seizures, or transient ischemic attacks (TIA) [1, 3, 20].

Patients should be educated on stress reduction techniques and managing vascular risk factors, including hypertension and hyperlipidemia, which may help reduce the risk of recurrence. A healthy lifestyle is recommended, which includes: weight management, smoking cessation, avoiding excessive alcohol consumption, adhering to medical advice, regular follow-up appointments.

Additionally, it is crucial to inform patients about symptoms requiring urgent medical attention, such as new neurological deficits or episodes of disorientation. This knowledge enhances their ability to respond appropriately and seek medical help [1].

Psychological support can be beneficial for both the patient and their family, as they often experience stress related to the sudden onset of TGA. Healthcare providers should reassure patients and their relatives about the self-limiting and mild nature of this condition [11].

During an acute episode of TGA, it is advisable to avoid physical activities that could increase intrathoracic venous pressure until symptoms resolve. In certain cases, intravenous thiamine administration may be considered, especially when a deficiency is suspected [1, 15].

If alternative diagnoses such as seizures or ischemic stroke/TIA are suspected, appropriate treatment and secondary prevention according to established guidelines must be initiated. Patients at elevated risk for serious cardiovascular events should receive treatment based on primary prevention principles [11].

Currently, there are no established evidence-based strategies for preventing TGA recurrences. However, studies suggest a potential link between acute spikes in blood pressure and the occurrence of TGA in patients without prior adaptation to chronic hypertension (e.g., without microangiopathy or left ventricular hypertrophy). In such instances, avoiding sudden increases in blood pressure is advised [11].

Periodic instrumental examinations, such as EEG or cardiological diagnostics, may be considered if another pathology is suspected. After memory deficits have resolved, there are no restrictions on daily activities, including driving [11, 21].

Prognosis and long-term outcome

TGA is currently defined as an isolated event that resolves within 24 hours. Symptoms completely resolve without long-term complications in the vast majority of patients. However, the episodic occurrence of this condition may lead to stress and anxiety for both patients and their families during the acute episode. Some studies indicate the possibility of recurrences, with incidence rates ranging from 2.9% to 26.3%, depending on the definition used for identifying TGA cases and the duration of follow-up, a ten-year study reported a recurrence rate of 6.3% [11, 22].

A study involving 340 German patients found that those with recurrent TGA episodes were significantly younger (recurrent vs. single episode: 63.6 ± 8.6 years vs. 67.3 ± 10.5 years) and exhibited less extensive microangiopathic changes in the brain compared to patients with isolated TGA episodes [11, 23].

The duration of TGA itself does not seem to influence the occurrence of complications. In a study of 639 patients with TGA, no differences were found in the risk of seizures or major cardiovascular events between patients with episodes lasting less than one hour and those with episodes lasting more than one hour [24].

Some earlier clinical studies suggested an increased risk of stroke. However, current reports indicate that patients with TGA have a similar risk of stroke, myocardial infarction, and peripheral artery disease as the general population [11]. A study conducted on 525 patients with TGA demonstrated a cumulative stroke risk of 0.6%, indicating that TGA is not associated with an increased risk of cardiovascular events [25]. Additionally, a cohort study analysing 27,266 hospitalizations with a diagnosis of TGA seems to confirm these data, showing that the risk of readmission for ischemic stroke did not differ significantly from the control group during a mean follow-up period of 192.2 (SD 102.4) days [26].

A recent cohort study from Korea covering the years 2002-2020 suggests a potential risk of epilepsy following episodes of transient global amnesia, with an adjusted hazard ratio of 1.46 [1, 27].

Prevention methods

There are no known or certain methods for preventing episodes of TGA. Education plays a fundamental role in informing patients about the benign and transient nature of symptoms, emphasizing its isolated nature and favorable prognosis. Patients should be advised on the necessity for medical care during acute episodes to rule out other potential causes. Lifestyle changes, reduction of vascular risk factors, and stress reduction may potentially decrease the risk of recurrences [1].

Summary

TGA is a serious health problem that causes significant anxiety for patients and their families, despite its short-term and mild nature. This problem affects a significant number of people worldwide and an increasing number of studies are focused on exploring this topic in detail.

Further understanding of the pathophysiology of TGA, risk factors, prevention strategies, diagnostics and treatment options is necessary. Further studies are needed to determine the etiology of this condition and the role of ischemic and non-ischemic factors in its pathogenesis.

DISCLOSURES

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