

Transient Global Amnesia: Emergency Department Evaluation And Management

Abstract

Transient global amnesia is a clinically distinct syndrome characterized by the acute inability to form new memories. It can last up to 24 hours. The diagnosis is dependent on eliminating other more serious etiologies including toxic ingestions, acute strokes, complex partial seizures, and central nervous system infections. Transient global amnesia confers no known long-term risks; however, when abnormal signs or symptoms are present, they take precedence and guide the formulation of a differential diagnosis and investigation. In witnessed transient global amnesia with classic features, a minimalist approach is reasonable, avoiding overtesting, inappropriate medication, and medical interventions in favor of observation, ensuring patient safety, and reassuring patients and their families. This review provides a detailed framework for distinguishing transient global amnesia from its dangerous mimics and managing its course in the emergency department.

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CME Objectives

Upon completion of this article, you should be able to:

1. Define transient global amnesia and differentiate it from the most common mimics, based on clinical presentation.
2. List the features that should remove the transient global amnesia diagnosis from consideration and identify high-risk patients.
3. Counsel patients and their families about transient global amnesia and its benign implications.

Prior to beginning this activity, see "Physician CME Information" on the back page.

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Opening Cases

A 60-year-old man with no significant medical history is brought to the ED by EMS after 2 hours of sudden memory loss. His wife has been with him since the onset of symptoms, when he started asking her the same question repeatedly every few minutes. The patient is calm, cooperative, and oriented to person, place, and time, but he frequently repeats himself and does not appear to be forming new memories. He knows what he did yesterday and this morning but cannot seem to recall events of the past few hours. He is perseverating about what he was doing 1 hour ago. When you step away and subsequently return to the bedside a few minutes after the initial encounter, the patient does not recognize you and asks, "Have we met before?" The patient has no other complaints, and there are no neurologic or infectious symptoms. His vital signs are normal. You wonder if you should activate the stroke team and order an emergent CT of the brain.

A 54-year-old woman with hypertension presents with sudden memory loss. She was smoking a cigarette with her friend when she became confused and started asking, "How did we get here?" repeatedly. The witness called 911. EMS providers detected no stroke symptoms and transported her to your ED. In the ED, the patient is alert and oriented to self and place, but not to time. She demonstrates clear anterograde amnesia, failing to remember you on multiple occasions. She also complains of a mild diffuse headache, but she has no other symptoms and is found to be neurologically and, otherwise, cognitively normal. She states that she feels fine and wants to sign out against medical advice. You explain to her repeatedly that, because she is not encoding memories, she cannot be discharged, and lacks decisional capacity. You place her on hospital involuntary hold. Twenty hours later, her amnesia has fully resolved. She has not repeated any questions during the past 2 hours and remembers your latest conversations. In the meantime, she has developed new mild ataxia. She says "I'm not worried about that. I'll be fine once I have a cigarette. I'd like to sign out against medical advice." She is alert and oriented and lacks psychiatric risk factors. She consistently and accurately verbalizes, without reminders, several times over 30 minutes, the risks associated with leaving. You wonder: Should you discharge this patient against medical advice?

Introduction

Transient global amnesia (TGA) is a clinical syndrome marked by the acute onset of profound anterograde amnesia (the inability to form new memories) lasting up to 24 hours in the absence of other neurologic deficits or changes in alertness and cognition. Retrograde amnesia (the loss of pre-existing memories) can also occur. In TGA, isolated loss of new memory formation is limited to facts and events (declarative memory), and patients retain implicit and procedural memory (motor tasks

and coordination). The classic TGA patient exhibits the "broken-record" phenomenon (asking the same questions repeatedly) in the absence of other signs or symptoms. Typical questions include "How did I get to the hospital?" and asking medical staff, "Have we met?" despite several introductions.

The mean duration of symptoms in TGA events is approximately 4 to 6 hours and, in a majority of cases, symptoms resolve within 8 hours of initial onset.¹⁻³ In TGA, symptoms resolve gradually and spontaneously within 24 hours without the need for medical intervention.⁴ The definitive diagnosis of TGA cannot be made until symptoms have resolved. Failure of symptom resolution and other worrisome signs and symptoms excludes the diagnosis.

Descriptions of TGA first appeared in the medical literature in the 1950s.⁵⁻⁷ In the following decades, consensus emerged regarding the clinical features required for diagnosis. Classically, the syndrome occurs in a functional middle-aged or elderly patient; fewer than 10% of cases present in patients aged < 50 years.³ TGA patients are notable to the clinician and their family and friends for repetitive questioning, but patients remain oriented to person and place. In uncomplicated cases (ie, in which TGA is not coincidentally presenting with another unrelated process), TGA patients maintain hemodynamic stability and are otherwise cognitively intact, retaining the ability to perform complex tasks. Often, the patient will have little to no memory of the entire event.^{8,9}

In order to confidently make the diagnosis of TGA, a prehospital witness is helpful in providing a reliable history of the symptoms, including a description of the onset. By convention, patients with head trauma in the preceding 72 hours, those with known epilepsy, patients with any other neurologic abnormalities (motor deficits, aphasia, dysarthria, cognitive impairment, loss of attention span, etc), and patients with known psychiatric disorders are excluded from the diagnosis of TGA.^{8,9}

While diagnostic criteria are clear, disagreement as to the causes and etiology of TGA continues. In the past 2 decades, neuroimaging and other testing modalities have suggested potential etiologies, but controversies among researchers persist amid limited and conflicting evidence. From the standpoint of the emergency clinician, however, a definition of the clinical syndrome and the required features for diagnosis are now widely agreed upon, and TGA is a relatively easy diagnosis to make when it presents with classic features; however, upon initial presentation, rare mimics must be considered and any unusual features and symptoms must be investigated. (See **Table 1, page 3.**) It is the role of the emergency clinician to recognize TGA syndrome and to exclude other more dangerous conditions that present with an amnesic component among a constellation of other features that, though exceedingly rare, can mimic TGA.^{8,10}

Once definitively diagnosed, patients and their families need education on the diagnosis and its benign prognosis. This issue of *Emergency Medicine Practice* provides a comprehensive review of the literature on TGA and its diagnostic criteria. We also suggest a simplified workflow in order to facilitate decision-making and minimize unnecessary diagnostic testing.

Critical Appraisal Of The Literature

A PubMed literature search was performed using the search terms *transient global amnesia* as well as *amnesia* and *emergency*. The National Guideline Clearinghouse (www.guideline.gov), and the Cochrane Database of Systematic Reviews were searched with the term *amnesia*; this search yielded only mentions of amnesia in the context of differential diagnoses for stroke syndromes, but no guidelines, policies, or reviews focused primarily on amnesia were found. We also searched for guidelines released by the American College of Emergency Physicians and the American Academy of Neurology. There are no known published guidelines by these bodies related specifically to amnesia.

Little high-quality research on TGA exists. Most of the literature is comprised of case reports and case series. Unfortunately, close reading of many case reports of TGA with unusual features reveals a frequent, incorrect conflation of TGA as a distinct clinical entity and its *sine qua non* symptom, anterograde amnesia. However, much has been learned through higher-quality retrospective chart reviews, retrospective cohort studies, prospective case-control studies, and prospective cohort studies. Case-control studies have been performed in an attempt to understand the etiology of TGA. These studies primarily focused on imaging and electroencephalogram (EEG) studies in an attempt to elucidate the etiology of TGA. Several studies have attempted to link TGA to serologic markers, and the results have been marked by minimal success. Retrospective studies designed to identify risk factors for the development of TGA have been conducted and have also yielded mixed results. No trial assessing

Table 1. Transient Global Amnesia Mimics

- Seizure/transient epileptic amnesia
- Stroke, cardiovascular accident (rare)
- Atypical migraine
- Head injury/occult trauma/concussion syndrome
- Medication and recreational drug side effect
- Herpetic encephalitis
- Early neurosyphilis
- Human immunodeficiency virus dementia
- Alcohol psychosis
- Alcohol blackout

any medical intervention has ever been reported in the medical literature.

Epidemiology

The incidence of TGA ranges from 5.2 to 10 per 100,000 patients per year.¹¹⁻¹⁴ The mean age of TGA patients is 65 years. The highest risk factor for TGA appears to be the occurrence of a past TGA event, with yearly recurrence rates ranging from 2.5% to 5.8%, representing an approximately 1000-fold increased risk over the baseline population. Longer-term recurrence rates of up to 14.1% have been reported.¹⁵ Other risk factors include advanced age and/or a history of migraine headaches.^{8,16} In older persons, the incidence of TGA ranges from 23.5 to 32 per 100,000 patients per year, representing a 4.5- to 6-fold increase. There are no gender or ethnic predispositions currently identified. While the increased incidence of TGA coincides with age groups in which cardiovascular risk factors are higher than average, studies have demonstrated that the presence of vascular risk factors does not confer a higher rate of TGA.¹⁷⁻²⁰ Several case reports and case series have suggested an increased predisposition to TGA based on a yet-to-be identified genetic inheritance.²¹ These reports describe several first-degree relatives, each of whom experienced TGA at different times in their lives. However, no predisposing gene has been identified, and it is unclear what fraction of TGA cases may reflect any underlying genetic disorder.²²⁻²⁵

Inciting Factors

Several inciting factors for TGA events have been described. While no associations have been validated in large-scale studies, correlations with certain historical features have been observed, including emotional stress directly before onset (18%-29% of cases) and intense physical effort (25%-31% of cases).^{3,26} Other "everyday life" factors such as sexual activity, recreational substance use, and rapid changes in temperature have been described, but less frequently.^{3,27-33} Some of these, including swimming, sexual activity, and intense emotional arousal, are associated with Valsalva-like maneuvers.^{3,34,35} Finally, medical procedures such as stress saline-contrast echocardiography, cerebral angiography, trigeminal neuralgia treatments, and enemas have been reported as precipitating TGA events.^{6,36,37} Among a variety of concurrent symptoms that have been described in TGA, headache, nausea, and vomiting appear to be the most common, occurring in approximately 17% of patients.¹¹

Pathophysiology

The pathophysiology of TGA remains controversial, and several theories have been proposed.

One theory suggests that TGA is a manifestation of short-lived, reversible ischemia resulting from temporary intracranial venous stasis surrounding the hippocampus, a region found in the medial temporal brain known to be crucial for memory storage. Supporting this theory are case reports of TGA events with abnormal hippocampal findings on magnetic resonance imaging (MRI), some of which resolved on follow-up imaging.³⁸⁻⁴³ The reversible ischemia hypothesis is further supported by the observation that Valsalva-like maneuvers (which can cause temporary cerebral ischemia) have been seen as inciting features of TGA.^{3,44,45} In some studies, duplex ultrasonography of TGA patients demonstrated an increased prevalence of insufficient jugular vein valves in comparison to healthy controls; however, other studies failed to find an effect compared to age- and sex-matched controls.^{26,34,35,46-53}

Arterial insufficiency has also been proposed as etiology, but recent evidence shows no difference between TGA patients and controls.^{54,55} An embolic etiology for TGA based on the observation of increased rates of patent foramen ovale in a study of TGA patients was not confirmed, and the lack of ischemic findings in most patients with TGA further undermines this explanation.^{51,56}

Another explanation for TGA is spreading cortical depression, in which a reduction of brain activity following hyperstimulation triggers sudden memory loss.⁵⁷ This theory is supported by the prevalence of migraine headaches in patients with TGA, and observational studies linking TGA and emotional events.⁵⁸⁻⁶⁰ A further proposal suggests a nonconvulsive epileptic etiology. This theory is supported by studies showing abnormal EEGs in TGA patients both during TGA attacks and at follow-up. One study found that as many as 7% of TGA patients manifested EEG signs of epilepsy within 1 year, a substantial increase over baseline population estimates for new-onset epilepsy (which range between 77 and 237 per 100,000 patients per year).^{18,61-63} However, other studies showed no increased risk of epilepsy in TGA patients at follow-up compared to the baseline population, and others have noted that many of the EEG findings in a subset of acutely amnesic patients do not constitute epilepsy.^{14,64,65}

The disagreements in the data from various studies have not been explained, and no theory is without contradictory evidence. Disagreement may stem from study population differences or inclusion/exclusion criteria. Alternatively, TGA may simply turn out to be a pathophysiologically heterogeneous condition comprised of components of several of these proposals, or another model yet to be elucidated.

Differential Diagnosis

TGA is such a distinct clinical entity that when it is observed in the absence of other symptoms and changes, there are exceedingly few mimics that should plausibly be included in a differential diagnosis. When other abnormal signs or symptoms are present, those symptoms should take precedence and guide the formulation of a differential diagnosis and investigation. Rarely, however, certain other entities can present as TGA.

Transient epileptic amnesia may present as repeated short amnesic episodes lasting < 1 hour, while TGA rarely resolves in this time frame.^{8,9,66} Exceptionally rare stroke syndromes have been reported that may appear like TGA.⁶⁷⁻⁶⁹ Importantly, none of the patients in these reported cases were found to have concerning National Institutes of Health Stroke Scale (NIHSS) findings that would have necessitated immediate guideline-driven management. More commonly (though still rare), anterograde amnesia can present as a coinciding symptom among other worrisome stroke symptoms that, by themselves, immediately warrant urgent investigation and, by definition, rule out TGA as the definitive diagnosis.^{68,70}

Although rare, other cerebrovascular problems, such as subarachnoid hemorrhage or cerebral hematoma, have coincided with amnesia. In these unusual cases, other neurologic deficits and abnormal features (such as nystagmus or syncope) were always present, and thus need not be normally included in the differential for acute amnesia.⁷¹⁻⁷³ A comparison of TGA to transient ischemic attacks (TIAs) has been discussed, but, as with rare strokes, the presence of other acute neurologic symptoms reflexively removes TGA from diagnostic consideration. Further, risk profiles comparing TGA and TIA have demonstrated that patients who experience TGA have cardiovascular and cerebrovascular risk profiles similar to the baseline population, while TIAs confer higher future risks.^{17,74} Thus, TGA should not be considered as a rare subtype of TIA.

In certain at-risk patients, herpes encephalitis presenting with anterograde amnesia can be considered in the differential for TGA; however, in herpes encephalitis, the amnesia fails to resolve within 24 hours and other symptoms usually occur.⁷⁵ While anterograde amnesia may be an early finding in neurosyphilis, other findings should emerge on examination and observation.⁷⁶

Patients presenting after witnessed head injury may present with anterograde amnesia, but amnesia caused by trauma should be investigated and managed as a concussion/traumatic brain injury, rather than as TGA.

Rare cases of medication side effects (the gamma-Aminobutyric acid [GABA]-agonist zolpidem

[Ambien®, Edluar®, Intermezzo®, Zolpimist®]) and recreational drug use (marijuana) causing TGA have been described.^{29,77} Korsakoff syndrome caused by chronic alcoholism can cause anterograde amnesia, but only in a pattern of other neurologic and cognitive changes.^{78,79} “Alcohol blackout” is a rare, but distinct, entity characterized by anterograde amnesia during intoxication in the absence of loss of consciousness or other skill deficits.⁸⁰ A psychogenic cause of TGA without other cognitive changes has never been described.

Prehospital Management

Frequently, a family member or an acquaintance activates emergency medical services (EMS) regarding a patient displaying sudden amnesic features. EMS providers should elicit corroborative history from any witnesses and, when possible, transport family, friends, and/or relevant witnesses to the hospital with the patient. EMS providers should attempt to obtain medication history and bring any medication containers a patient may have had at the scene, as the acutely amnesic patient is at risk of having unintentionally overdosed due to unwittingly taking their medications repeatedly. If the patient has changed locations or was unwitnessed at onset, family and friends should be engaged to search those locations for medications the patient may have unintentionally overdosed.

EMS providers should be educated about TGA, but their primary role is to consider the diagnoses of stroke or seizure and to transport the patient to the most appropriate medical center if such signs are present. EMS providers should perform a basic neurologic examination, including a validated prehospital stroke scale. EMS providers may help delineate whether the patient has aphasia versus amnesia during the initial evaluation.

EMS should obtain a point-of-care glucose level. An electrocardiogram (ECG) is indicated only when the patient has other symptoms, such as chest pain, palpitations, or shortness of breath. While TGA has been reported after acute myocardial infarctions (AMI), there are no known cases of TGA as the sole presenting symptom of AMI in the presence of completely normal vital signs.^{81,82}

Emergency Department Evaluation

History

The emergency clinician’s task is to recognize TGA and to consider other conditions that may mimic TGA as well as those that co-present with amnesia and other symptoms. When other symptoms are present, those symptoms—and not the amnesia alone—should guide the investigation.

There are 8 diagnostic features required for the

diagnosis of TGA. (See Table 2.) While isolated anterograde amnesia in the absence of other signs, symptoms, or concerning historical features may quickly render TGA as the leading diagnosis, TGA cannot be definitely diagnosed unless and until all symptoms have resolved in under 24 hours.

Some researchers and clinicians require the presence of a corroborating witness in order to make the diagnosis of TGA, as there have been incorrect TGA diagnoses in some cases lacking a witness at onset, and rare cases where witnesses present at onset reported features such as pallor, dizziness, or even forgotten chest pain that led to alternative diagnoses.^{8,9,83} In the absence of a witness at onset, a well-defined “last seen normal” can be helpful, but does not eliminate the possibility of unwitnessed symptoms or trauma at onset that would require the appropriate workup. Further, symptoms lasting longer than 24 hours excludes the TGA diagnosis, by definition. We therefore emphasize the importance of a reliable witness at onset. In the absence of a witness at onset, an extensive and broader emergency department (ED) workup may be warranted in accordance with the patient’s other symptoms (eg, dizziness, headache, or chest pain) as well as any other underlying individual risks. On the other hand, in witnessed TGA, a remarkably minimalist approach is equally reasonable.

Historical Features Of Transient Global Amnesia

While TGA is conspicuous, once recognized, certain historical features should be ascertained:

- **TGA history.** Prior TGA events are the most likely risk factor for future events. However, uncovering a history of multiple or closely spaced events (even in TGA cases that do not coincide with other unusual symptoms) may trigger a broader workup that may reveal an undiagnosed alternate condition, most often a seizure disorder.
- **Headache history.** Literature points to a stronger consideration of TGA in patients with headache during a TGA event (which may occur in 10%-40% of patients) or in patients with long-standing headaches or who carry a migraine headache diagnosis.⁸⁴

Table 2. Diagnostic Features Of Transient Global Amnesia⁹

- Witnessed at onset and during attack
- Must have anterograde amnesia
- No focal neurological symptoms or signs during or after
- No epileptic features
- No clouding of consciousness, no loss of personal identity, and no cognitive impairment other than amnesia
- No head injury in the past 72 hours
- No seizures in the last 2 years, and not on medication for epilepsy
- Must resolve in 24 hours

- **Neurologic history.** Any seizure activity or known history excludes the diagnosis of TGA. However, other neurologic history or recent other symptoms should be elicited, as TGA can, rarely, present as a concurrent symptom of other processes.
- **Medication list.** For obvious reasons, unintentional overdoses are possible in TGA patients who may have repeatedly taken their medications. Determine any medications a patient may have overdosed. If there is significant concern, this may mean asking family or friends to search for pill bottles in the place where the event started, or in the patient's belongings (purse, handbag, pockets). Isolated case reports of a small number of medication classes (such as benzodiazepines) have been reported to induce a TGA-like syndrome.^{77,85} While not described in the literature, an unintentional overdose of multiple daily aspirin tablets or acetaminophen would necessitate commensurate workups.
- **Psychiatric history.** TGA is generally differentiated from psychosis by its isolated anterograde amnesic features without other changes or psychiatric features. Substance abuse may cause amnesia, but it would similarly be expected to cause other symptoms not seen in TGA.
- **Triggers or inciting features.** In addition to the diagnostic features required for TGA (see Table 2, page 5), other features may raise suspicion of TGA. (See also the "Epidemiology" and "Pathophysiology" sections, pages 3 and 4.) These include triggering events such as strong emotions, intense physical exertion, changes in posture, medical procedures, high altitude, contact with water, and changes in body temperature. Other features seen include pain, agitation, anxiety, and, in some cases, nausea, paresthesias, and nonspecific dizziness.^{3,8,9,18}
- **Historical red flags.** Repeated TGA events may represent an undiagnosed neurologic condition, including a seizure disorder. Elicit history of immune compromise, especially in older patients and those with a history of alcohol or intravenous drug abuse, as these patients also warrant more intensive workup. Some researchers have suggested that patients with hypercoagulable states be given extra consideration as well.^{79,86-89} We believe that patients aged < 50 years warrant special consideration, as TGA is rare in young individuals.

Physical And Cognitive Evaluation

As with any neurologic complaint, a thorough neurologic and physical examination should be performed. However, the focus of the evaluation of the patient with suspected TGA is to accurately determine the type of memory loss the patient is

experiencing and to test cognition. (See Table 3.) Patients with TGA experience only the loss of memory pertaining to new facts and events, so-called *declarative* or *explicit* memory loss (mainly stored by the hippocampus). By contrast, nondeclarative, implicit, or procedural memories (stored in the cerebellum and the basal ganglia) should not be compromised. For example, a TGA patient retains the ability to operate a car or the ability to open a door with a key. Patients also retain all other cognitive functions.

For cognitive evaluation of patients suspected of TGA, we recommend the following tests:

1. **Test immediate versus delayed recall.** Immediate recall (eg, recalling a digit span, repeating back a series of numbers, or remembering 3 words) remains intact in TGA patients, while delayed recall (after 5 minutes) is impaired.
2. **Assess attention span.** Executive function abilities such as "serial 7s" or spelling the word "world" backwards remains intact during TGA.
3. **Test procedural memory.** TGA patients retain task memory (eg, making a paper airplane).
4. **Test cognitive functioning.** While the minimal state examination (MMSE) is generally impractical in the ED, certain components may be used.^{90,91} Testing for alexia (inability to read), anomia (inability to name objects), and apraxia (loss of spatial relationships, ie, drawing a clock) may be useful, as these have rarely been observed to coincide with amnesia in rare stroke syndromes that exhibited amnesic features but, by virtue of these findings, were not true TGA events.⁷¹

Serial evaluations should be performed to monitor memory improvement and to detect new symptoms. Generally, TGA patients regain anterograde memory gradually. They will not regain memories from the episode, and this is not a requirement when considering symptom resolution.

Table 3. Types And Features Of Memory Loss

Type of Memory Loss	Features
Anterograde	Inability to form new memories (eg, patient is unable to remember how he/she arrived at the hospital).
Retrograde	Inability to recall memories from the past (eg, patient is unable to recall his/her city of birth).
Dissociative (psychogenic)	Inability to recall important personal information (beyond normal forgetfulness) not due to a "dissociative identity disorder, dissociative fugue, posttraumatic stress disorder, acute stress disorder, or somatization disorder" (eg, patient does not know his own name and is unable to provide historical information). Must not be related to substance abuse ⁸³

Diagnostic Studies

The workup required in acutely amnesic patients depends greatly on the patient. In patients with entirely witnessed TGA without concurrent physical signs or symptoms and normal vital signs (ie, not high-risk), the differential diagnosis and diagnostic testing should remain limited. It is not the same as the broader differential for altered mental status. Conversely, acute memory loss in the presence of either concerning vital signs or any other physical, neurologic, or cognitive complaint excludes TGA and requires diagnostic testing tailored to those symptoms. Additionally, despite a paucity of definitive evidence, we believe that certain high-risk patients may necessitate a broader workup. Such patients include persons aged < 50 years, immune-compromised patients, and intravenous drug users/substance abusers. (See Table 4.)

Non-high-risk individuals presenting with TGA syndromes without concurrent symptoms or abnormal vital signs can, realistically, be managed with minimal testing. Limiting management to serial examinations, observation, and patient/family education until symptoms resolve may even be a reasonable strategy in some classic cases. Unless other concerning historical features, unusual physical examination findings, or atypical symptoms are present, the yield of extensive diagnostic testing in search of occult etiologies is low.

Basic Laboratory Studies

There are no particular abnormal laboratory findings that are associated with isolated amnesic syndromes. Therefore, in classic TGA events lacking unexpected other symptoms, forgoing blood or urine testing may be appropriate. However, many hospital protocols require some basic laboratory testing for admission or observation that may include serum glucose, complete blood count, comprehensive metabolic panel, and coagulation testing. If chronic alcoholism is suspected, a thiamine level may be useful.⁷⁸

Toxicology Studies

No specific blood or urine tests are useful in the diagnosis or management of TGA. Some groups recommend a toxicology screen, but others have noted that there is no evidence for any toxin that consistently produces a TGA-like picture.^{2,92}

Table 4. High-Risk Features In Patients With Suspected Transient Global Amnesia

- Age < 50 years
- Immunocompromise
- History of drug or alcohol abuse
- Abnormal vital signs on presentation

Cardiac/Vascular Testing

While rare cases of amnesia have been reported as the sole presenting feature of aortic dissection and myocardial infarction, these are infrequent enough to be “case-report worthy” and always presented with unusual features, including abnormal vital signs, chest pain, pallor at onset or during the event, or recent acutely worsening anginal symptoms.^{82,93-96} A patient or witness reporting any unusual history including (but not limited to) syncope, dizziness, severe headache, chest pain, back pain, or pallor should prompt further investigation. Evidence of hypertension, hypotension, unequal pulse deficits, aortic regurgitation murmur, bradycardia, or heart failure can be evaluated by physical examination, with close attention to vital signs, a screening ECG, and chest radiographs. Laboratory testing may include cardiac biomarkers, and advanced imaging for dissection or other processes if clinical suspicion warrants it.

Imaging

Imaging is not required for the diagnosis of TGA; however, in patients unwitnessed at onset or with unusual features, imaging to rule out trauma or a cardiovascular accident is warranted. Also, patients with a duration of symptoms approaching 24 hours may also warrant imaging, though no strict cutoff time has been proposed. While MRI with and without contrast with diffusion-weighted imaging (DWI) is the modality most likely to yield information, this modality is not required for the diagnosis of TGA, and its diagnostic utility has not been consistently demonstrated.^{49,97,98} Thus, the purpose of obtaining an MRI with DWI in the emergency setting is not to rule in TGA, but to rule out an unusual neurological syndrome if amnesia does not resolve within the typical time course or if other high-risk features exist. If there is any suspicion of an acute ischemic stroke, immediate computed tomography (CT) should be obtained and a normal stroke workup should take precedence.

Neurologic Testing

The presence of any seizure-like symptoms or a history of repeated short TGA-like events may prompt the need for EEG, either in the ED or inpatient setting, as some short and repeated purely amnesic events can, rarely, be signs of undiagnosed epilepsy.^{78,99} While certain EEG findings (such as spikes and sharp waves with left-sided predominance) have been described in some TGA patients, no diagnostic utility has been demonstrated, and the significance of specific epileptiform patterns described in this subset remains unclear. Similar to imaging, the purpose of EEG testing in the setting of acute memory loss remains to rule in seizures, not to rule TGA in or out.¹⁰⁰

Finally, an otherwise asymptomatic TGA syn-

drome that does not resolve in 24 hours requires a broader investigation, admission, and neurologic consultation. At this point, testing may include an EEG, advanced imaging, and even cerebrospinal fluid testing, as seizure foci from infections or other lesions must be considered at this point.^{75,101}

Treatment

The guiding principle in the management and treatment of TGA is to avoid the inappropriate administration of medications and medical interventions. It may be prudent to withhold medications the patient may normally take that may have amnesic side effects, including benzodiazepines. The mainstay of TGA management is observation and serial neurologic examinations until symptoms have completely resolved and the patient returns to baseline. Reassurance and education of the patient and family is useful and necessary in mitigating discomfort during the natural course of TGA, which can be disturbing, often mostly to patients' families and friends.

Special Considerations

Decisional Capacity

While patients with TGA may appear to have a normal level of alertness, their inability to remember and contextualize new information renders them without decisional capacity, despite the fact that they may be oriented to time, person, place, and have no psychiatric risk factors. Therefore, until the amnesic episode fully resolves, TGA patients cannot be discharged against medical advice.

Once symptoms resolve and capacity has been re-established (as judged clinically by a physician), a patient regains the right to refuse further testing that may have been planned, provided the patient fully understands and believes what has occurred, despite having no memory of the event. When in doubt, capacity can be assessed using CURVES mnemonic (Choose/Communicate, Understand, Reason, Value, Emergency, Surrogate). Patients should demonstrate the ability to choose and communicate their choice; understand the benefits and risks and alternatives; and make logical, rational choices that are consistent with their values.¹⁰² While symptomatic TGA patients may be able to convincingly demonstrate immediate capacity, it is important to revisit these conversation 5 to 20 minutes later to ensure that the condition has truly resolved and the patient has regained capacity. This ensures that the patient is processing, assimilating, and remembering information, rather than simply repeating it.¹⁰²

At-Risk Subgroups

There are no definitive differences in testing thresholds for imaging modalities for patients of various

age or risk groups. While some cases of TGA have coincided with hypercoagulable conditions, no validated studies have shown different risks than the baseline population.^{86,87} However, despite rigorous evidence, we feel that certain situations warrant a lower threshold for imaging and laboratory testing, even in uncomplicated TGA syndromes, including:

1. **Patients aged < 50 years.** Fewer than 10% of TGA patients are younger than 50 years old.² Therefore, younger patients presenting to the ED with acute amnesia should lower the threshold for investigating alternative diagnoses. However, younger patients experiencing TGA are also more likely to have migraine disorders.⁵⁹ Therefore, in these patients, it is important to get a full headache history to avoid exposing patients to unnecessary radiation associated with imaging.
2. **Individuals with risk factors for encephalitis.** In particular, herpes simplex virus infections should be considered in anyone with high individual risk or a known prior infection.⁷⁵ For these patients, even in otherwise typical TGA syndromes, we recommend early consideration of imaging and a broader infectious disease workup, which may include neuroimaging and a cerebrospinal fluid analysis. We believe the same approach may be reasonably applied to substance abusers and immune-compromised patients presenting with amnesia.
3. **Chronic alcohol abusers or patients with chronic malabsorptive syndromes.** Korsakoff syndrome may be considered in these patients. This may necessitate neuroimaging and administering intravenous thiamine in addition to obtaining basic laboratory studies.⁷⁸

Controversies And Cutting Edge

What is the role for advanced imaging?

There is no clinical role for advanced imaging positron emission tomography (PET) or single-photon emission computerized tomography (SPECT). An MRI with DWI can be used to exclude acute ischemia syndromes and to rule out malignancy or a space-occupying lesion when suspicion is elevated by the presence of relevant symptoms. While PET and SPECT imaging have been used by researchers in an attempt to establish the etiology of TGA, these modalities are research-centered and do not contribute to the diagnosis of TGA in the ED.

Are there any serologic biomarkers that have been investigated in association with TGA?

Some studies have suggested that patients with TGA are at increased likelihood to have abnormalities in certain blood markers, including aspartate aminotransferase, alanine aminotransferase, lactate dehydrogenase, and glutamate,¹⁰³ and there is a case

report of an increased troponin T level.¹⁰⁴ However, none of these has been validated, and none is necessary or sufficient for the diagnosis of TGA.

What is the appropriate use of EEG in patients with acute amnesia?

Despite the fact that some investigators have found abnormal EEG results in TGA patients, findings are inconsistent and do not aid in the diagnosis of TGA. A bedside or complete EEG has not been found to have consistent utility. An EEG may be considered in cases at temporal extremes. For example, in amnestic events lasting < 1 hour, transient epileptic amnesia is more likely than TGA, and an EEG may be of use.^{22,91} When anterograde amnesia has not resolved after 24 hours, TGA is excluded, and an EEG may be warranted, among other tests.

Is there a role for ED lumbar puncture in the diagnosis of TGA and for ruling out dangerous TGA mimics?

There is no need for normal lumbar puncture findings in order to diagnose TGA or to rule out dangerous etiologies occurring in typical, non-high-risk patients. (See Table 4, page 7.) No cerebrospinal fluid abnormalities consistent with TGA have ever been identified. Lumbar puncture has extremely limited utility in ruling in or ruling out other etiologies on the differential diagnosis for TGA. There have been rare cases of isolated amnesia in patients with subarachnoid hemorrhage and ruptured arteriovenous malformations, but these cases contained unusual signs and symptoms and thus did not present purely as TGA.^{73,94} In instances of TGA symptoms that coincide with other neurologic findings, severe headache, and/or abnormal vital signs, lumbar puncture can be considered as part of a wider differential diagnosis. In particular, the presence of symptoms indicating a broader cognitive deficit (ie, more than simple declarative memory loss), a longer course of symptoms, the presence of immunosuppression, and other infectious signs and symptoms such as fever, neck stiffness, severe headache, or photophobia should direct the emergency clinician to perform a lumbar puncture as part of a broader differential diagnosis that may include encephalitis and intracranial bleeds.

Are there any medications that have been shown to have efficacy in the treatment of TGA in the ED or prevention of recurrence in the outpatient setting?

In non-peer-reviewed literature, empiric thiamine has been suggested in the early management of TGA. This recommendation is a supposed safeguard against alcohol-related vitamin deficiency that may have led to an acute amnestic event with a clinical picture similar to TGA; it is not suggested as a TGA-specific intervention. Moreover, amnesia from

alcohol-related effects almost always appears with other features that exclude the diagnosis of TGA, and are thus of exceedingly low utility. In individuals with a history of heavy or chronic alcohol consumption, intravenous thiamine administration may be considered.

Does TGA impart a higher risk of future cerebrovascular disease, cardiovascular disease, or vascular death?

No. In fact, compared to age-matched amnestic patients in whom TGA was excluded, TGA patients have a substantially lower rate of stroke, myocardial infarction, or vascular death (and also, death from any cause).^{9,11}

Disposition

For cases of classic and otherwise asymptomatic TGA, it is not necessary to transfer a patient to a hospital with neurology services. Neurologic consultation remains at the discretion of the evaluating clinician and hospital protocols, though it may be desirable in prolonged cases or with high-risk patients (see Table 4, page 7). While most TGA syndromes resolve in < 10 hours, TGA patients should be observed until symptoms clear, which, in rare cases, can approach 24 hours.³ Observation can occur either in the inpatient setting or in an ED observation unit, if available. If symptoms do not resolve within 24 hours, differential diagnosis should be revisited and appropriate workup initiated.

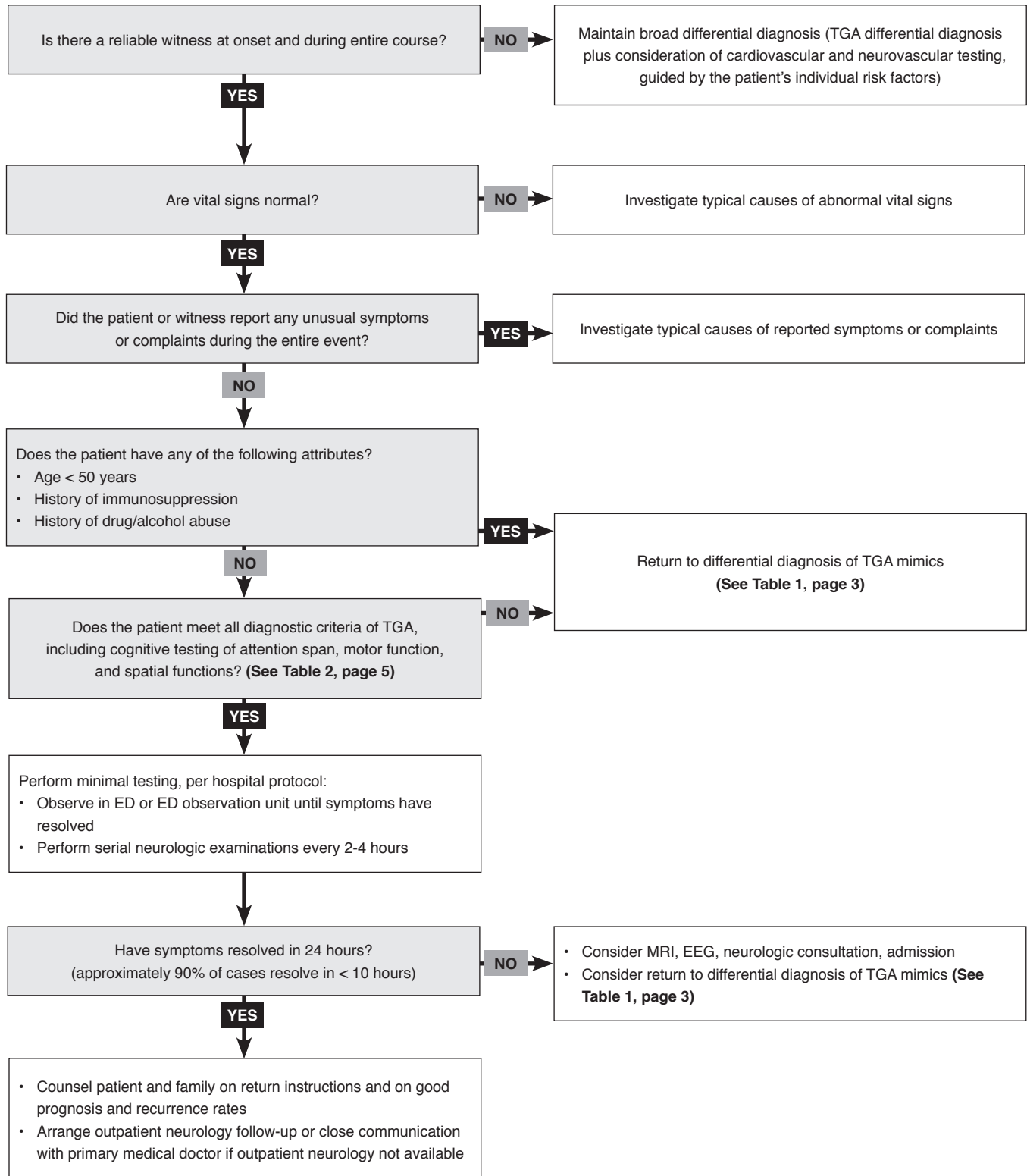
Patients should be considered for discharge when memory impairments resolve and they have returned to their baseline mental status. The expectation is that a resolved TGA patient should be able to form new memories, although they are not expected to recall events that occurred during the attack. Allow time between the beginning of symptom resolution and complete resolution, as many patients regain anterograde memory slowly and may initially wax and wane. Once consistently asymptomatic, patients are clear to return to normal activities, including driving. Follow-up can be with a primary care provider or neurologist within 7 to 10 days.

As stated previously, patients with active memory impairment do not have decisional capacity and therefore must not be permitted to be discharged against medical advice while symptoms are present. During the amnestic event, patients with TGA should be identified as elopement risks and proper precautions taken, according to hospital protocols.

Summary

Patients with amnestic syndromes are most likely to present to the ED in the acute setting. While emergency clinicians must be aware of the specific criteria

Clinical Pathway For Sudden And Persistent Amnesia (< 24 Hours)



Abbreviations: ED, emergency department; EEG, electroencephalogram; MRI, magnetic resonance imaging; TGA, transient global amnesia.

for making the diagnosis of TGA, the emergency clinician's first priority in assessing a patient with acute memory loss is to consider and rule out other dangerous conditions, mainly by addressing any concurrent symptoms or risks. However, in normal-risk, otherwise asymptomatic witnessed TGA, no diagnostic tests (laboratory and imaging) are necessary for diagnosis of TGA, once all criteria have been met and are confirmed by the full, spontaneous, and consistent resolution of symptoms in < 24 hours. We recommend first verifying hemodynamic stability and completing a detailed history and physical examination. Establishing the type of memory loss the patient is experiencing is the linchpin of the evaluation. If it is determined that the patient is experiencing any deficit other than profound anterograde amnesia (though a component of modest retrograde amnesia is permitted), TGA is excluded.

Generally, we recommend minimizing low-yield tests. This can be accomplished primarily by avoiding inappropriately widening the differential diagnosis to infectious, cardiac, or other more dangerous neurologic etiologies in the absence of signs or symptoms that might suggest such rare and otherwise occult causes. However, special attention must be paid to high-risk individuals (see Table 4, page 7) as well as any patient with abnormal vital signs or unusual concurrent symptoms (both neurologic or otherwise), as these features appear, almost without exception, in rare TGA mimics. Emergent neurologic consultation in the ED is not always obligated for presumed TGA unless other dangerous conditions cannot be fully excluded, suspicion for other etiologies is elevated, or admission is warranted.

No evidence in the literature supports the use of any pharmacologic interventions for shortening the clinical course of TGA or preventing future episodes. Patients should be observed in the ED, an observation unit, or inpatient floor in the hospital until symptoms completely resolve. This often takes up to 12 hours, if the definition of TGA is met and the syndrome lasts < 24 hours. If symptoms have not resolved by 24 hours or if new symptoms appear, the diagnosis of TGA cannot be made. Once resolved, there are no activity restrictions at discharge.

Time- And Cost-Effective Strategies

- Given the relatively low prevalence of TGA in the ED, cost-effective management may be less of an issue at the local level. However, testing should be strictly directed towards the patient's precise clinical presentation. A less-thoughtful approach to the TGA workup could cost, by one estimate, \$6000 per hour (or, \$42,000 per 7-hour case).¹⁰⁵ Replicated in each of the 15,000 to 30,000 TGA cases per year, such an approach could needlessly cost the healthcare system up

to \$1.26 billion per year.

- Widening the differential diagnosis to include, for example, anginal equivalents or infectious etiologies is appropriate only in patients who clearly exhibit relevant symptoms (during or leading up to the event) or who have conspicuous risks and historical features that impart an especially high clinical suspicion for these alternative diagnoses. While some testing may prove unavoidable, eliminating some low-yield cardiac testing and advanced imaging can save thousands of dollars per ED visit and minimize exposing otherwise well patients to unnecessary medical testing.¹⁰⁵
- The use of ED observation units in place of admission for non-high-risk patients with witnessed, isolated amnesia may be appropriate and may reduce the need for hospital admission, provided that serial neurologic, cognitive, and memory testing can occur on a regular basis.

Case Conclusions

You admitted the first patient to the ED observation unit for 24 hours. After 8 hours, the patient's symptoms began to resolve. When you entered the room, he said, "We've met, right?" but still couldn't remember who you were. Ten hours into his ED course, the patient no longer displayed signs of memory loss. He was able to recognize you as his doctor and remembered you when you left and returned an hour later. He was finally able to pass with ease the 3-item memory test at 5 minutes. While he couldn't remember how he got to the hospital or any of the events from the first 8 hours of his ED stay, he was glad that his symptoms resolved and asked when he could go home. You informed him that it would be best for him to remain in observation for at least an hour or two longer, so that you could recheck and make sure his symptoms did not return. After another 2 hours, the patient remained symptom-free, and you subsequently discharged him home. He asked if he could drive himself, and you instructed him that he could do so safely.

Although your second patient initially appeared to be experiencing an episode of TGA, later symptoms changed the diagnosis. Despite the absence of other medically concerning findings early in the course, she could not initially be discharged against medical advice because amnesic patients failing to encode memories are unable to correctly assimilate new information and, therefore, lack decisional capacity. However, once that ability returned, the presence of concerning findings (in this case, new ataxia that removed TGA as a primary consideration because the main concern was no longer amnesia alone) did not render her without capacity. Even though the late new findings were alarming, this patient demonstrated a consistent and reliable ability to weigh the risks and benefits. She was therefore permitted to be discharged against medical advice, with strict return precautions.

1. **"She has a history of repeated memory loss that began 3 hours ago, and did not report any neurologic deficits. I assumed this episode was TGA. I performed my usual neurologic examination because I felt comfortable, based on history, that the patient had TGA."**
In cases of suspected TGA, an especially thorough neurologic examination is essential, as the presence of even mild neurologic deficits may be the only indication of a rare stroke syndrome presenting with amnesia as the most conspicuous feature. In cases in which rare stroke syndromes have been mistaken for TGA, the vast majority of patients exhibited subtle neurologic deficits that a cursory neurologic examination might have missed.
2. **"I gave my patient 100 mg of intravenous thiamine because I read that it is a treatment for suspected TGA. My patient improved, confirming the diagnosis of TGA."**
Given the poorly understood pathophysiologic pathways of TGA, using the response to any empiric treatment to rule in or rule out this syndrome should be avoided. There are currently no studies suggesting that thiamine, or any other pharmacologic agent, is an effective treatment for TGA in either hastening symptom resolution or preventing future episodes.
3. **"I thought the patient had less than 6 hours of memory recall abnormalities and no fevers or headaches, which made me suspect TGA. I forgot to ask about encephalitis risk factors. I made the mistake of early diagnostic closure."**
While some red flags of infectious encephalitis—such as fever and focal neurological deficits—are apparent on examination, it is the emergency clinician's job to take a detailed history and physical examination, which should include immunosuppression status, as those patients have a higher predisposition for specific central nervous system infections (most notably herpes simplex virus encephalitis) that can, rarely, present as isolated anterograde amnesia.
4. **"Part of my routine workup for a new neurologic complaint includes a noncontrast CT of the brain and sometimes a lumbar puncture, especially if the patient has altered mental status, as it captures many dangerous etiologies such as bleeding, strokes, and infection."**
In amnesic patients, the differential diagnosis can be inappropriately broad if the emergency clinician incorrectly conflates anterograde amnesia with altered mental status. Neuroimaging (such as CT) is only indicated for patients who have concurrent signs or risk factors that would normally lead an emergency clinician to acquire imaging in the absence of memory impairment. Such signs would include any signs or symptoms of an acute stroke syndrome, a history of head trauma, or historical and physical findings consistent with aneurysms. A lumbar puncture is indicated only in the presence of symptoms that would normally lead the clinician to suspect a central nervous system infection or high-risk intracranial hemorrhage.
5. **"The patient began to recall some events and otherwise looked well. Her family wanted her to go home and follow up with her primary care doctor the next day, so I discharged the patient in spite of the fact that she was still having occasional amnesic symptoms."**
This is one of the great pitfalls of TGA, as patients appear largely well and retain executive functions, such as the ability to get dressed without assistance. As TGA begins to resolve, amnesic features often wax and wane. It is important to observe patients until they are consistently asymptomatic in order to rule out abnormally long courses (worrisome for rare alternate diagnoses) and for patient safety, as described previously. TGA patients can be frustrating in their repetitive requests to leave, as they may not remember that you have asked them to stay until symptoms have completely resolved. They often will not remember the discussed care plan or have any sense of how much time has elapsed since your last evaluation. All efforts should be made to maintain the patient's safety and not discharge the patient prior to complete and consistent symptom resolution. TGA patients should be considered elopement risks until all symptoms have resolved, as the diagnosis of TGA cannot actually be made until symptoms resolve.

6. **"I try to provide a specific diagnosis for every neurologic complaint of my patients at the time of disposition."**

In the case of suspected TGA, it is difficult to assign a specific diagnosis in the emergency setting since the diagnosis relies on resolution of symptoms within 24 hours after a well-documented, witnessed time of onset. Further, an incorrect diagnosis can mislabel a patient as having a benign process and might lead to anchoring bias by future physicians who will reassess the neurologic examination. This could potentially lead to failure to detect dangerous alternative etiologies such as seizures or developing central nervous system infections. Until symptoms resolve, the diagnosis of "amnesia" is appropriate, but not TGA.

7. **"I often feel pressure to 'do something' and intervene on behalf of my patients. My patient's family kept asking me to do something to help her."**

It is difficult to alleviate the concerns of family members of patients experiencing an acute TGA event. TGA events are often understandably frightening for patients, their families, and their friends witnessing the event. We recommend talking with patients and their families and setting appropriate expectations. Directing expectations through careful guidance and reassurance of your comfort managing this syndrome can help. Counseling patients and family so that they fully understand the expected time course of the syndrome and its expected benign outcome can reduce anxiety. Avoiding the pitfall of overtesting and treating can be difficult, but reassurance and the provision of a safe environment are arguably the most critical interventions an emergency clinician can offer in classic TGA cases.

8. **"My patient's family was very concerned about dementia, given that the patient had sudden memory loss at the age of 67."**

The mean age of patients with TGA attacks (65 years) makes it difficult to counsel patients and their families through an event with full reassurance, as elevated age is itself the primary risk factor for dementia. However, dementia presents with a gradual onset, and unless the patient has exhibited symptoms of memory loss prior to an acute attack, sudden anterograde amnesia is not consistent with the initial presentation of dementia, nor does it predict the development of dementia in the future.

9. **"My patient had a past medical history of seizures and a sudden episode of amnesia, without any other symptoms. Since she has not required antiepileptic medication in over 1 year, TGA was my primary diagnosis."**

Patients with a known seizure should cause the emergency clinician to maintain a broader differential diagnosis, including transient epileptic amnesia and, in cases without resolution in an appropriate timeframe, nonconvulsive status or even a rare stroke syndrome. These may require neurologic consultation, evaluation, and potentially neuroimaging, a neurologic consultation to discuss the role of an EEG, and consideration of anticonvulsant medication.

10. **"My patient with TGA wanted to sign out against medical advice and go home. He seemed reliable and lived next to the hospital. To protect myself, I allowed him to sign out against medical advice."**

Despite appearances, patients with acute amnesia do not have decisional capacity. It is the healthcare provider's responsibility to verify that a patient has capacity to sign out against medical advice. Patients must be able to understand the information they are given and make logical decisions based on that information. This relies on the patient's ability to understand his current medical status or condition over time, not just "in the moment." Patients with TGA suffer from short-term memory impairment and may not remember their current medical condition or follow-up instructions. They should be appropriately marked as a high elopement risk, according to local protocol at your institution. While TGA itself is not medically dangerous, the inability to form memories could pose genuine hazards to the patient in other environments. For example, a TGA patient could turn on a kitchen stove and forget he did this. He might repeatedly take his normal medications, leading to unintended overdose. For the patients' own safety, they should not be discharged against medical advice or otherwise until they are consistently asymptomatic.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study is included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, are noted by an asterisk (*) next to the number of the reference.

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1. **How long do most TGA events last?**
 - a. 5 to 30 minutes
 - b. 30 to 60 minutes
 - c. 1 to 10 hours
 - d. 11 to 24 hours
2. **Which of the following features DECREASES the likelihood of a TGA diagnosis?**
 - a. The patient has no history of seizures.
 - b. The patient's age is 25.
 - c. The symptoms initially resolved after 4 hours but then returned for 3 more hours before disappearing completely.
 - d. The patient has no memory of the event, even after regaining the ability to form new memories.
3. **Which statement best explains the pathophysiology of TGA?**
 - a. It is poorly understood, but ultimately benign.
 - b. It is a stroke syndrome with a better prognosis than other strokes.
 - c. It is a form of epilepsy.
 - d. It is psychogenic.
4. **Which of the following has not been reported as a possible inciting factor for TGA events?**
 - a. Sexual activity
 - b. Mild traumatic brain injury (concussion)
 - c. Recreational substance use
 - d. Invasive medical procedures
5. **A patient presents to the ED with a family member and reports complete resolution of anterograde memory loss that lasted for 8 hours. The family member witnessed the event and corroborates this timeline. The patient is currently exhibiting no symptoms. What is the next step in management?**
 - a. Reassure and discharge home with instructions for TGA.
 - b. Order MRI brain imaging.
 - c. Discuss the case with the neurology consult service.
 - d. Obtain a more detailed history and physical examination.
6. **What is the most likely risk factor for TGA?**
 - a. Alcoholism
 - b. Epilepsy
 - c. Prior TGA event
 - d. Stroke
7. **A patient with TGA would exhibit which of the following cognitive features?**
 - a. Inability to draw the face of a clock
 - b. Transient loss of personal identity
 - c. Inability to perform immediate recall (ie, cannot repeat the names of 3 items directly back to the clinician)
 - d. Ability to perform immediate recall but cannot remember the items 5 minutes later
8. **Which neuroimaging procedure is required for the diagnosis of TGA?**
 - a. Contrast-enhanced CT of the brain
 - b. Contrast-enhanced diffusion-weighted MRI
 - c. Non-contrast-enhanced CT of the brain
 - d. No imaging is required to make the diagnosis of TGA
9. **Your patient, aged 39 years, presents to the ED after not taking her seizure medication for the past 3 months since her prescription ran out. During the past year, she has been asymptomatic and without any seizure events. She has been under increasing stress due to getting fired from her job. On examination, she is disoriented to time and place and keeps repeating questions. Her adult niece is there to clarify that the event began approximately 2 hours prior to ED arrival. The patient does know her name. On examination, she displays no seizure-like activity, and the niece reports the same prior to her arrival in the ED. Per the niece, this is not similar to her history of past seizures. Which of the following is the most appropriate next management step?**
 - a. Explain to the niece that this is most likely a benign process such as TGA, but further testing will be necessary.
 - b. Perform a full neurologic examination. Consider neurology consult.
 - c. Take the patient to radiology for a head CT scan.
 - d. Take the patient to radiology for diffusion-weighted MRI
10. **You are discharging a patient from the ED observation unit after his symptoms have completely resolved. Which of the following discharge instruction is safe?**
 - a. No driving for 12 hours
 - b. No driving for 24 hours
 - c. No driving until cleared by a neurologist
 - d. No restrictions on driving

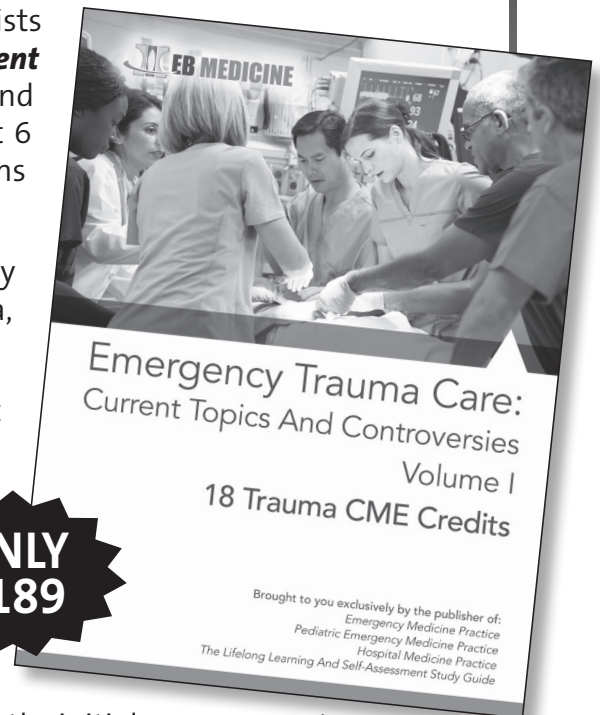
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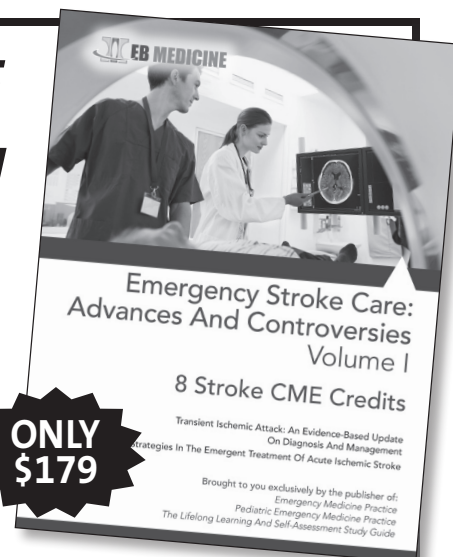
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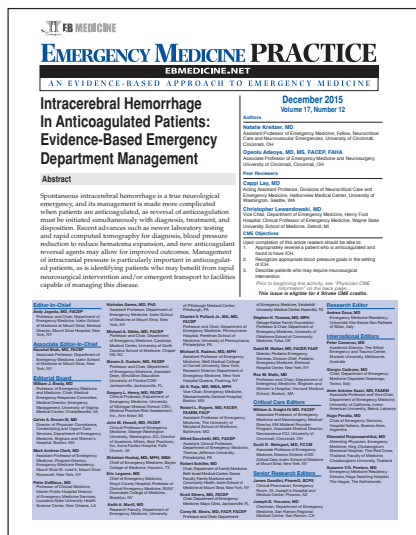
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Editor-in-Chief Andy Jagoda, MD presented **Emergency Medicine Practice's** first "Issue of the Year" award to Natalie Kreitzer, MD and Opeolu Adeoye, MD for their December 2015 issue, "Intracerebral Hemorrhage In Anticoagulated Patients: Evidence-Based Emergency Department Management." Dr. Jagoda's recommendation, *EMP* Editorial Board votes, and subscriber comments were factored into the choice of this issue for the prestigious award. To view the press release, go to www.ebmedicine.net/IOYaward.

Our congratulations go to Dr. Kreitzer and Dr. Adeoye.



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