INTRODUCTION

Transient global amnesia (TGA) is a neurological syndrome that was initially discovered in 1956 by Bender and simultaneously but independently of Courjon and Guyotat. It is defined as a sudden onset anterograde amnesia that lasts up to 24 h. Despite the mild subclinical neuropsychological impairment, it may persist for days after the episode. Transient global amnesia predominantly affects middle-aged or elderly patients. The incidence of TGA is approximately 2.9–10/100,000 worldwide. It is also more common in people living with migraine. Diagnosis of TGA is based on the exclusion of all potential etiologies that may present a similar pattern. The criteria of a TGA diagnosis include the presence of a witness during the attacks, cognitive impairment limited to
anterograde amnesia, preserved conscious state, normal neurologic examination findings; absence of recent head trauma and active epilepsy; and reversibility of the attack in 24 h. 7,8

There is no known treatment for transient global amnesia. The condition is benign and resolves itself within 24 hours. The exclusion of any potential etiologies is the primary goal of clinicians. Multiple pathophysiologic explanations have been suggested, but the cause is still unknown. 9 Here, we present a 60-year-old male patient who presented with a sudden onset of confusion and anterograde memory loss and was diagnosed with TGA.

2 | CASE PRESENTATION

A 60-year-old slightly overweight male patient presented to our neurology department with a chief complaint of confusion and short-term memory loss that started at 7:00 AM while at home. He is married, the father of five children, and well-educated. He was in a state of good health when, according to his son, he suddenly found him confused, disoriented, and asking repetitive questions. The patient started to ask about his whereabouts and what had happened. He remained confused from morning till evening. There was no history of previous similar episodes. He had no recent sleep problems, head trauma, or substance abuse history. The patient did not have a prior history of migraine or associated recent headaches, visual auras, nausea, or vomiting. He had no history of seizure disorder; likewise, he had no complaints of strange smells, tastes, epigastric elevations, or abnormal, inexplicable sensations. There was not any recent seasonal change that could trigger such a condition. He did not have a strenuous physical activity or sexual exertion before the onset of this condition. He had no tingling, numbness, weakness, vertigo, difficulty speaking, blurred vision, loss of consciousness, shortness of breath, or fever.

His past medical and surgical history is significant only for well-controlled asthma. There were no recent changes in the medication dose. His vital signs were normal. A neurological examination revealed that he was awake, alert, and aware of person and place but not of time or situation. He was not sure why he was brought to the hospital. His long-term memory was intact, but his short-term memory was impaired. There were no apparent active delusions or hallucinations. His speech was fluent (no dysarthria or aphasia). There was no cranial nerve deficit seen on the examination. His motor examination showed normal muscle tone and strength (based on the MRC scale), and deep tendon reflexes were normal. There was no gait abnormality seen. Likewise, there was no neck stiffness or meningeal signs. The rest of the examination, including the cardiovascular and respiratory systems, were normal. Laboratory investigations revealed a normal complete blood count, biochemistry profile, and normal thyroid hormone levels. A urine analysis was negative for leukocytes, bacteria, or casts. On the MMSE, the patient could not answer the current date, season, seven serial subtractions, and recall of named objects. He had a score of 18 out of 30.

Brain MRI (including DWI) immediately showed mild gliotic foci but no acute abnormal cerebral pathology (see Figure 1). The EEG did not show any abnormal cerebral activity. Carotid and vertebral arteries duplex ultrasound showed normal findings. The patient was diagnosed with transient global amnesia after the exclusion of the possible etiologies. The patient returned to his baseline normal neurologic state in less than 24 h. A repeated MMSE test later showed a normal score (30/30).

3 | DISCUSSION

Transient global amnesia (TGA) is a clinical syndrome characterized by the abrupt onset of anterograde amnesia (the inability to encode new memories), accompanied by repetitive questioning that occasionally has a retrograde component. TGA could last up to 24 h without impairing other neurological functions. 10

Most TGA cases are seen in middle-aged and older people. The incidence of TGA among those less than 50 years old is estimated at 23–32 per 100,000 each year. 11 There is no known etiology for TGA. Known risk factors for stroke, such as hypertension, diabetes mellitus, smoking, and hypercholesterolemia, are not associated with TGA, while migraines have been found to be highly associated with TGA. 12,13 The present case presented with sudden onset confusion and anterograde amnesia that lasted for 20 h. He was in a state of good health and had no previous similar episodes. He did not have a recent history of sleep problems, head trauma, or substance abuse. There was no previous history of migraine or seizure disorder. He had no history of diabetes mellitus, hypertension, hypercholesterolemia, known cardiac disease, or thromboembolism. Likewise, there were no other associated neurologic deficits.

Pathogenesis of TGA remains unclear; however, theories suggest that it may be caused by venous and arterial factors and conditions like migraines, epilepsy, and psychogenic diseases. 12 Transient ischemic attacks caused by arterial thromboembolism and TGA share some characteristics, including occurrence in older patients and a shorter duration of less than 24 h; hence, the arterial ischemic hypothesis has been proposed. It is worth noting that TGA usually lasts longer and patients with TGA tend to
have lower cerebrovascular risk factors such as hypertension, diabetes, hypercholesterolemia, smoking, and underlying cardiac pathologies.\textsuperscript{14,15}

Diagnosis of TGA is based on the exclusion of all other potential causes of acute memory loss. The following criteria by Hodge and Warlow\textsuperscript{8} in 1990 is used for the diagnosis of TGA: (a) information from a witness that was present at the beginning of the event to rule out possibilities of head trauma, conscious impairment; (b) A complete neurological examination should be normal except for anterograde amnesia; (c) Anterograde memory should return to normal within 24 h; and (d) Seizure event or active epilepsy must be excluded. Anoxia, hypoglycemia, alcohol intoxication, drug withdrawal, encephalitis, metabolic disturbances, and Wernicke’s encephalopathy are other conditions that must be differentiated from TGA.\textsuperscript{16}

The present case was witnessed by his son. There was no incident of head injury, seizure attack, conscious impairment, or loss of personal identity. Neurologic examination was normal except for anterograde amnesia; (c) Anterograde memory should return to normal within 24 h; and (d) Seizure event or active epilepsy must be excluded. Anoxia, hypoglycemia, alcohol intoxication, drug withdrawal, encephalitis, metabolic disturbances, and Wernicke’s encephalopathy are other conditions that must be differentiated from TGA.\textsuperscript{16}

The present case was witnessed by his son. There was no incident of head injury, seizure attack, conscious impairment, or loss of personal identity. Neurologic examination was normal except for anterograde amnesia with preserved other cognitive domains. There was no history of alcohol intoxication, drug withdrawal, hypoglycemia, or other metabolic derangements. Brain MRI excluded acute cerebral pathology, including cerebrovascular accident and encephalitis. An electroencephalogram showed normal brain activity.

Thus, the most useful management for TGA is to monitor the patient until the amnesia resolves. The condition does not usually recur; however, according to previous studies, the annual recurrence rate ranges from 2.5 to slightly over 5 percent, and individuals who experience TGA do not have a greater risk of mortality, epilepsy, or stroke.

4 CONCLUSION

Transient global amnesia is a benign self-resolving condition characterized by anterograde amnesia that resolves in less than 24 h with no associated neurologic deficit. Although less common, it should be considered after excluding all possible etiologies for a patient who presents with sudden onset anterograde amnesia that complies with the diagnostic criteria of TGA.

AUTHOR CONTRIBUTIONS

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.
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CONFLICT OF INTEREST
The authors declare that they have no competing interests.

DATA AVAILABILITY STATEMENT
The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

ETHICAL APPROVAL
No ethical conflicts to disclose.

CONSENT
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

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