



Forgotten hours, Lingering Spikes: Distinguishing TGA from Its Epileptic Twin

Dr. Shamisha Khade, Dr. Vibhor Pardasani

Dept of Neurology, Bombay Hospital and Medical Research Center, Mumbai

INTRODUCTION

- Transient Global Amnesia (TGA) involves sudden onset of anterograde amnesia lasting less than 24 hours, without other neurological deficits.
- EEG abnormalities, especially temporal spikes, may lead to diagnostic confusion with Transient Epileptic Amnesia (TEA), a form of temporal lobe epilepsy.
- Differentiating between the two has critical therapeutic implications.

AIM

- To Highlight Diagnostic challenges and provide clinical markers to distinguish TGA from TEA in the presence of EEG abnormalities

METHODS

- We retrospectively analyzed patients presenting with transient episodes of sudden memory loss.
- Each patient underwent detailed neurological examination, Brain MRI and EEG
- Clinical follow-up was done over a period of 6 months
- The clinical presentation was compared against established criteria for both TGA and TEA.

Zeman Diagnostic criteria for TEA:

- (1) Recurrent witnessed episodes of transient amnesia
- (2) Cognitive functions other than memory, intact during episodes
- (3) Evidence for diagnosis of epilepsy on the basis of ≥ 1 of following:
 - (a) EEG: Epileptiform abnormalities
 - (b) Concurrent onset of other clinical features of epilepsy (e.g., lip-smacking, olfactory hallucinations)
 - (c) Clear-cut response to anti-seizure medications. Attacks occur several times a week at most & < 1 / year, often upon awakening, and lasting 30-60 min.

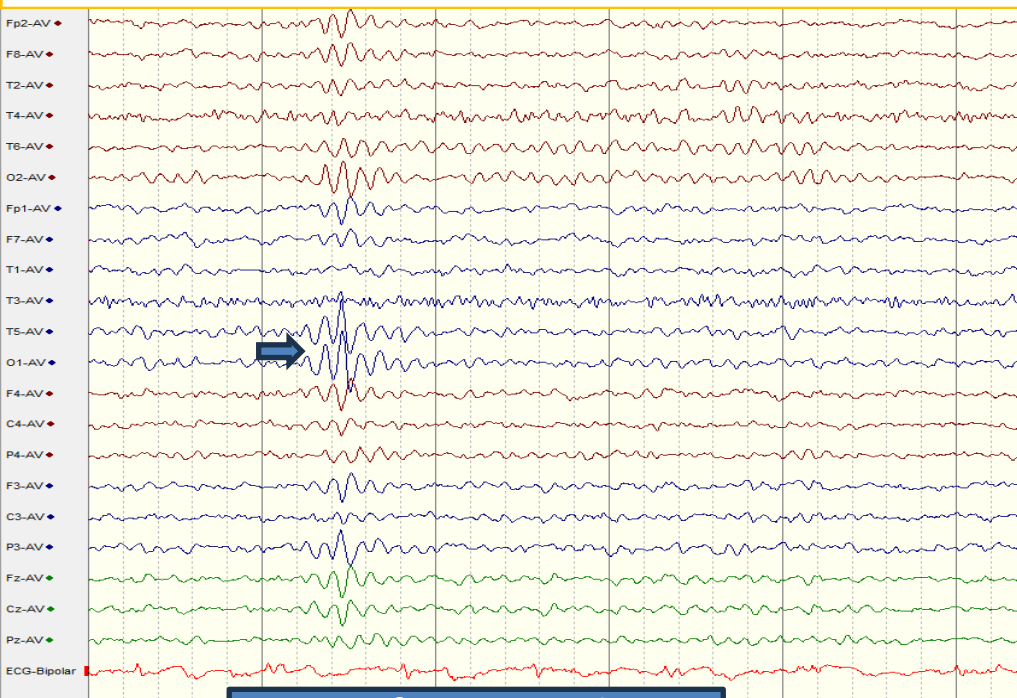
Hodges Diagnostic criteria TGA:

- (a) Attacks witnessed by a capable observer who was present for most of the attack
- (b) Clear-cut anterograde amnesia during the attack
- (c) Clouding of consciousness & loss of personal identity must be absent; Cognitive impairment limited to amnesia
- (d) There should be no accompanying focal neurological symptoms during the attack and no signs afterward
- (e) Epileptic features must be absent
- (f) Attacks must resolve < 24 hr
- (g) Patients with recent head injury / active epilepsy (remaining on medication or 1 Sz in past 2 years) excluded

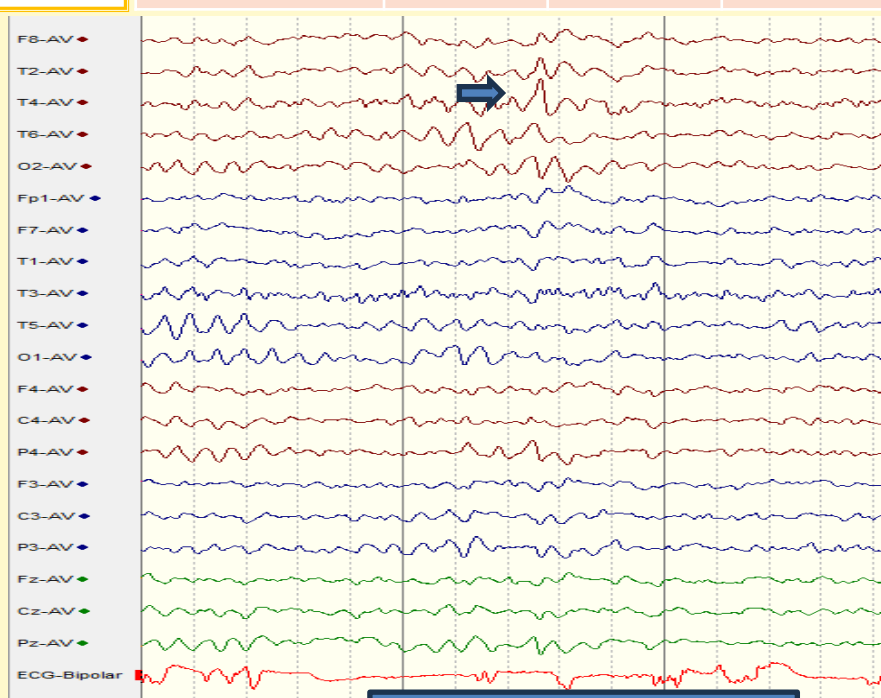
RESULTS

- ❖ All five patients experienced: **TGA phenotype** :
 - Sudden-onset amnesia lasting between 4–12 hours
 - Preservation of personal identity and consciousness
 - No automatisms, olfactory hallucinations, or focal deficits
 - No prior history of seizures or similar episodes
- EEG in all cases revealed Interictal Temporal Sharps.
- MRI findings were either normal.
- None of the patients were started on antiepileptic medications. Patients symptomatically better.
- No recurrence of amnestic episodes or seizure activity was noted during follow-up.

EEG Abnormalities	Left temporal sharps	Right temporal sharps	Bilateral sharps
Sharps	4(80%)	0	1(20%)
Slowing	2(40%)	0	0
Persistent abnormalities after 3 months	1(20%)	0	0



Left temporal



Right temporal

DISCUSSION

- **Fisher and Adams (1964)** used the term TGA for the first time
- **Jacome (1989)** demonstrated: EEG abnormalities were present in **36% of TGA** patients, often non-specific, with only 10.6% showing epileptiform discharges
- **Kwon et al. (2014)** found: EEG abnormalities in **22.9% patients** with TGA, with a strong **left-sided predominance**, suggesting possibly lateralized vulnerability of temporal networks

Clinical presentations

- **TEA** : Recurrence, shorter episode duration, additional “TGA-plus” symptoms (confusion, automatisms, language impairment, olfactory hallucinations)
- **TGA** in contrast : isolated, self-limiting

Pathophysiology

- **TGA** is linked to **Transient Hippocampal Dysfunction** (ischemia, venous congestion, migraine)
- **TEA** represents **Temporal Lobe Epilepsy**

Therapeutics

- TGA is generally Benign, with low recurrence & no long-term sequelae
- TEA requires early recognition to avoid mis-attribution to TGA & hasten treatment with anti-seizure medications
- Misclassification can delay therapy, exposing patients to preventable recurrent amnestic episodes
- Management of a properly diagnosed TGA is reassurance that in most patients this will not recur and does not cause long term cognitive difficulties

CONCLUSIONS

- **Temporal spikes on EEG should not prompt a diagnosis of TEA in patients with otherwise typical TGA features**
- **Clinical context remains paramount in differentiating TGA from TEA**
- **In the absence of recurrence, ictal signs, or behavioral arrest, a conservative approach without immediate anti-seizure therapy seems appropriate**

REFERENCES

1. Fisher CM, Adams RD. Transient global amnesia. Trans Am Neurol Assoc. 1958;83:143–146
2. Kwon YS, et al. Left dominance of EEG abnormalities in patients with TGA. Seizure. 2014;23(10):825–9.
3. Lanzzone J, et al. Transient epileptic and global amnesia: Real-life differential diagnosis. Epilepsy Behav. 2018;88:205–11.
4. Jacome DE. EEG features in transient global amnesia. Clin Electroencephalogr. 1989;20(3):183–91.