

Transient epileptic amnesia: a challenging diagnosis, pitfalls and key features.

M. Tombini¹, G. Assenza¹, C. Campana¹, F. Assenza¹, V. Di Lazzaro¹

1. Unit of Neurology, Neurophysiology, Neurobiology, Department of Medicine – Campus Bio-Medico University – Via Alvaro del Portillo, 21, 00128, Rome

BACKGROUND AND OBJECTIVES. The differential diagnosis of transient amnesia is challenging; in particular, differentiation between transient global amnesia (TGA) and transient epileptic amnesia (TEA) may be difficult on the basis of the only clinical features of the episodes¹⁻³. Our aim was to describe clinical and electroencephalographic features of patients affected by TEA compared to patients with TGA, in order to highlight major pitfalls and key features.

MATERIALS AND METHODS. Among 67 patients admitted to our department because of one or more episodes of transient amnesia from 2008 to 2016, 14 patients (20,9%) were diagnosed as affected TEA. We assessed clinical features and performed bv neurophysiological and neuroimaging assessment in all patients.

Pt	Age	Sex	Diagnosis	MRI	Focus	Duration	Recurrence	Onset	Symptoms PLUS	Therapy (dose/day)	Outcome
1	41	М	TLE	normal	T bil	2 h	-	morning	initial staring	-	SF
2	69	F	TLE	normal	T bil	2,5 h	-	morning	-	CBZ 400 mg	SF
3	70	М	TLE	normal	T right	10 min- 3 h	5	morning (awakening)	-	LVT 3000 mg	SF
4	75	М	TLE	normal	T left	12 h	3	morning (awakening)	_	LVT 1000 mg	SF
5	63	F	TLE	normal	T left	6 h	-	morning	headache	LTG 200 mg	SF
6	74	F	SFE	Hydrocephalus	T right	16 h	-	afternoon	tremor, ataxia	LVT 1000 mg	SF
7	60	М	TLE	normal	T left	15 min	3	afternoon	-	LVT 1500 mg	SF
8	64	F	TLE	normal	T right	1 h	-	morning	-	LTG 150 mg	SF
9	67	F	TLE	normal	T left	1 h	5	morning	language disorder	LVT 1500 mg	less than 1/y
10	59	F	TLE	normal	T left	5 h	4	morning (awakening)	confusion and headache	CBZ 800 mg	less than 1/y
11	61	F	SFE	Thalamic cavernoma	T left	2 h	2	morning	spatial disorientation	LVT 1000 mg	-
12	68	F	TLE	normal	T right	24 h	5	morning	temporo-spatial disorientation	ZNS 300 mg	-
13	74	F	TLE	normal	T right	1 h	3	morning (awakening)	spatial disorientation	LVT 1000 mg	SF
14	66	F	TLE	normal	T left	3 h	2	evening (awakening)	-	LVT 1250 mg	SF

Table 1

RESULTS. Twelve out of 14 patient with TEA had temporal epilepsy without mesial sclerosis or structural abnormalities, and the remaining two patients showed symptomatic focal epilepsy (Table 1). Patients with TEA and TGA did not differ for age (64,3 vs 61,8 years, respectively) and sex, and surprisingly for duration of amnesic episodes (5,3 vs 5,6 hours). The two groups differed significantly for the recurrence of episodes (higher in patients with TEA) and presence of other symptoms than pure amnestic ones (Table 2). Trigger factors were suggestive of TGA (25/53 cases), while the occurrence of amnestic episodes on awaking was more frequent in TEA (6/14 cases, Fig 1). The routine EEG recording showed epileptiform abnormalities only in 10 (71,4%) out of 14 patients, whereas the 24-hour ambulatory EEG revealed these abnormalities in all patients studied (mostly during sleep, Fig 2). In all patients the response to antiepileptic drugs (AEDs) was good.



EEG



DISCUSSION. TEA is a clinical condition that typically affects older people and mostly represented by cryptogenic temporal epilepsy. The transient epileptic amnesic episodes are usually frequent, may have additional ictal manifestations, tend to occur on awaking and respond well to AEDs. The long duration of the episodes observed in some patients could be due to postictal phenomena. Interictal abnormalities may be absent in routine EEG studies, but have always been detected during EEG sleep recordings (Fig 2).

	TGA	TEA	Р
N°	53	14	
Age (v)	61.8 ± 9.4	64.3 ± 8.6	n.s.
Sex	29 F/24 M	10 F/ 4 M	n.s.
Duration (hour)	5,6 ± 5,2	5,3 ± 7,1	n.s
Times of recurrence (%)	1 ± 1,4	2,3 ± 2	.035
Trigger factors (%)	25 (47,2)	1 (7,1)	.010
Awakening (%)	4 (7,5)	6 (42,9)	.001
TGA_plus (%)	7 (13,2)	8 (57,1)	<.001
Post-amnesic symptoms (%)	9 (17)	2 (14,3)	n.s.
EEG st (%)	5/51 (9,8)	10/14 (71,4)	<.001
EEG holter (%)	7/18 (38,9)	10/10 (100)	.002
Migraine (%)	10 (18,9)	2 (14,3)	n.s.
OSAS (%)	4 (7,5)	0 (0)	n.s.
PFO (%)	2 (3,8)	0 (0)	n.s.
Psychiatric comorbidity (%)	13 (24,5)	2 (14,3)	n.s.

CONCLUSIONS. The differential diagnosis of TEA from TGA may be difficult in the individual patient. Efforts should be made to obtain in these patients an EEG sleep recording that demonstrated a higher diagnostic sensitivity in our case series.

References

1. Butler CR, Graham KS, Hodges JR, Kapur N, Wardlaw JM, Zeman AZ. The syndrome of transient epileptic amnesia. Ann Neurol. 2007 Jun;61(6):587-98.

Bilo L, Meo R, Ruosi P, de Leva MF, Striano S. Transient epileptic amnesia: an emerging late-onset epileptic syndrome. Epilepsia. 2009 May; 50 Suppl 5:58-61.

Table 2

Bartsch T, Deuschl G. Transient global amnesia: functional anatomy and

4. clinical implications. Lancet Neurol. 2010 Feb;9(2):205-14.