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Memory Disorders Revealing Transient Epileptic Amnesia Of Temporal Lobe Epilepsy

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Abstract:
Background*: Temporal lobe epilepsy is the best known of the partial epilepsies. Temporal lobe epilepsy can cause prolonged post-ictal memory disorders which must be distinguished from disorders of consciousness. Temporal lobe epilepsy can cause cognitive disorders of undetermined etiology; temporal lobe epilepsy should be considered.*
Clinical case report: 67-year—old *patient presents to the consultation for cognitive disorders evolving paroxysmally .Critical EEG: (Theta discharge and burst of spikes in the left temporal region.) Clinical (seizures and memory disorders) and electrical improvement after taking the antiepileptic treatment Carbamazepine 600 mg as monotherapy in progressive stages.*
Conclusion: *Faced with paroxysmal memory disorders, consider temporal lobe epilepsy with transient epileptic amnesia.*
Key Words*: Memory disorders, Temporal lobe epilepsy, Transient epileptic amnesia*

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I. Introduction

Wieser divided the temporal lobe into 5 regions [1]: temporobasal, temporopolar, neocortical, opercular and cingulate frontobasal. ILAE recognizes two syndromes: mesial and neocortical (lateral). You should know that mesial temoral epilepsy is more common and the best known [2] [3]

Mesial temporal lobe epilepsy is the most common form of focal epilepsy in adolescents and adults. It represents 40% of all epilepsies (adolescents and adults) [4]

In the personal history of patients we can note: infection of the central nervous system, perinatal trauma, head trauma, febrile seizures. A family history of epilepsy can be found[5] . The majority of patients report an aura [6] accompanied by experiential and viscerosensory symptoms. The psychological phenomenon includes anxiety, déjà vu, never seen before and fear. The typical aura is an epigastric sensation [7]. followed by fixed gazes and oral or gestural automatisms, accompanied by vegetative phenomena such as pupillary dilation, hyperventilation, and tachycardia. Contralateral dystonic postures with ipsilateral automatisms during the attack are reliable signs of lateralization [3]. Prolonged seizures, secondary generalization and status epilepticus are rare. [4]

Neocortical temporal epilepsy presents a clinical picture different from that of mesial epilepsy. History of CNS infection, perinatal complications or head trauma, febrile seizures, are less common than in subjects with mesial temporal lobe epilepsy[9]. Seizures appear in patients 5 to 10 years later than in mesial temporal epilepsy [2]. Approximately 60% of seizures are preceded by an aura, such as auditory phenomena, psychic experiences or already seenand never seen, visual distortions and dizziness symptoms [10,11]. Fixed gaze and lack of response are the first objective clinical signs, often followed by early contralateral clonic movements with secondary generalization [12].

II. Clinical Case

Patient right-handed, foreign language teacher. 67-year—old patient presents to the consultation for cognitive disorders evolving paroxysmally since adolescence. Clinic: History of undetermined febrile convulsion, feeling of déjà vu, brief loss of contact, manual automatism (rubbing the forehead with the hands), phasic disorders lasting a few seconds. Critical EEG: (Theta discharge and burst of spikes in the left temporal region.) Brain CT: normal. Biology: normal. Brain MRI: left hippocampal atrophy. Functional imaging (SPECT

ECD-TC 99m): Left temporal hypoperfusion. MMSE outside of seizures and outside of postictal periods 30/30. Clinical (seizures and memory disorders) and electrical improvement after taking the antiepileptic treatment Carbamazepine 600 mg as monotherapy in progressive stages.

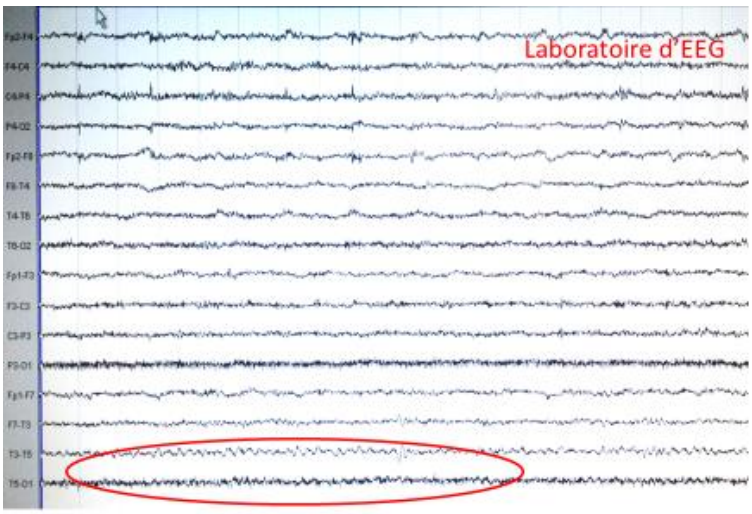

Figure 1. Critical EEG, theta discharge and burst of spikes in the left temporal region.

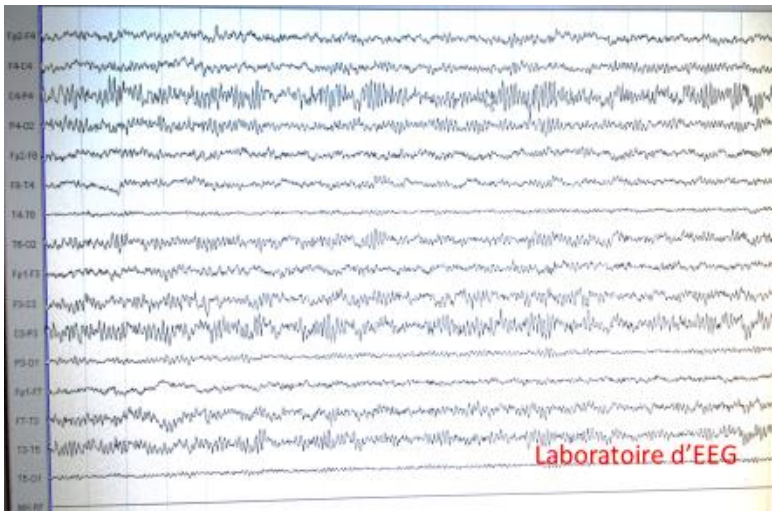

Figure 2. Inter-critical EEG, disappearance of epileptic discharges.

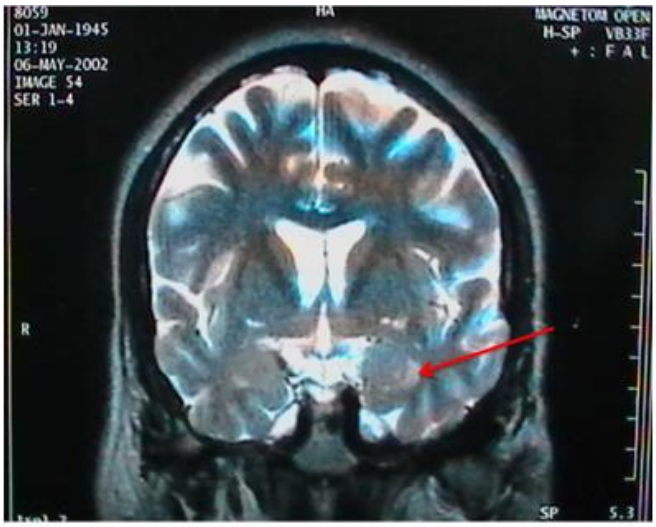

Figure 3. Brain MRI, left hippocampal atrophy.

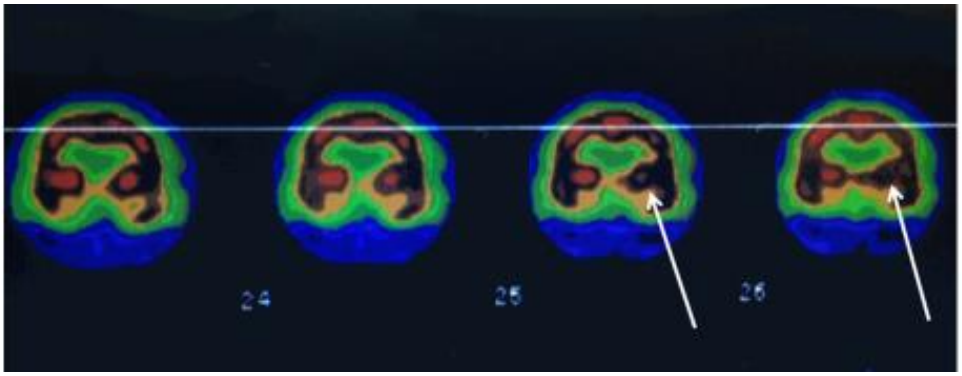

Figure 4. Functional imaging (SPECT ECD-TC 99m), left temporal hypoperfusion.

III. Discussion

Anamnestic analysis showed the early occurrence of temporal seizures in the form of a dream state with loss of contact and manual automatism before the appearance of memory disorders. The critical EEG revealed the presence of a left temporal paroxysmal focus. Favorable response to antiepileptic treatment (Carbamazepine 600 mg/day). This is a patient with temporal lobe epilepsy presenting arguments in favor of Transient Epileptic Amnesia.

Transient Epileptic Amnesia (TEA) is a clinical epilepsy syndrome of late onset ,characterized by the frequent repetition of episodes of paroxysmal amnesia, associated with the presence of inter-critical memory disturbances [13].The etiology and pathophysiological processes underlying TEA are not yet known. All of the clinical and imaging data already described involve temporal structures. internal organs, particularly the hippocampus[14]. A bilateral deep ictal discharge at the level of the hippocampus and temporal lobe, and/or post-ictal changes are possibly involved in the pathophysiological process. [14] [15] Critical episodes are typically episodes of transient amnesia, of variable duration but generally less than 60 minutes (sometimes longer due to the length of the postictal phase), during which the patient is unable to remember recent events and/or retain new information. During the paroxysmal amnestic episode, the patient may maintain appropriate behavior or present other symptoms of an epileptic nature (loss of contact, automation).

IV. Conclusion

Faced with paroxysmal memory disorders, consider temporal lobe epilepsy with transient epileptic amnesia.

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