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Hippocampus and epilepsy

Transient epileptic amnesia: Update on a slowly emerging epileptic syndrome

L'amnésie épileptique transitoire : un syndrome épileptique émergent

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INFO ARTICLE

Article history:
Received 26 September 2014
Accepted 11 November 2014
Available online xxx

Keywords:
Temporal lobe epilepsy
Episodic memory
Autobiographical memory
Amnesia
Hippocampus

Mots clés :
Épilepsie temporale
Mémoire épisodique
Mémoire autobiographique
Amnésie
Hippocampe

ABSTRACT

Transient epileptic amnesia (TEA) is a recently individualized, late-onset, pharmaco-sensitive form of mesial temporal lobe epilepsy with recurrent episodes of acute memory loss, but also interictal memory disturbances characterized by autobiographical and topographical memory impairment and a long-term consolidation deficit. In this article, we review the main clinical and electrophysiological characteristics of TEA, discuss its putative neuroanatomical substrate and mechanisms, common features and how it differs from related concepts, with the overall aim to defend the idea that TEA deserves to be recognized as a distinct epilepsy syndrome. While the pathophysiological basis remains largely unknown, emotional and/or dysimmune factors may have a potential influence. Most importantly, the concept of TEA is highly relevant to tertiary epilepsy and memory clinics, but also to routine neurology practice, leading to an adequate diagnosis and management of epilepsy-related, acute and long-standing memory deficits.

RÉSUMÉ

L'amnésie transitoire épileptique (ATE) est une forme d'épilepsie mésio-temporale d'individualisation récente, pharmaco-sensible, de début tardif, caractérisée par la survenue d'épisodes récurrents d'amnésie aiguë ainsi que des perturbations mnésiques interictales,

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http://dx.doi.org/10.1016/j.neurol.2014.11.010

Please cite this article in press as: Felician O, et al. Transient epileptic amnesia: Update on a slowly emerging epileptic syndrome. Revue neurologique (2015), http://dx.doi.org/10.1016/j.neurol.2014.11.010

comprenant des troubles de la mémoire autobiographique et topographique et de la consolidation à long terme. Cet article précise les principales caractéristiques cliniques et électrophysiologiques de l'ATE, discute de ses bases neuro-anatomiques et mécanismes physiopathologiques potentiels, ainsi que des points communs et des différences avec les concepts et entités proches, dans la perspective de défendre l'idée selon laquelle l'ATE nécessite d'être identifiée comme un syndrome épileptique distinct. Bien que ses bases physiopathologiques soient méconnues, des facteurs d'ordre émotionnel ou dysimmunitaire pourraient avoir un rôle potentiel. Au-delà de la discussion syndromique, le concept d'ATE s'avère extrêmement utile en pratique clinique, aussi bien en soin neurologique courant que dans les consultations mémoires ou les centres épileptiques spécialisés, permettant le diagnostic et la prise en charge adaptée de certaines perturbations mnésiques, aiguës ou chroniques, d'origine épileptique.

1. Introduction

That mesial temporal lobe seizures can manifest as recurrent episodes of isolated memory loss has been known for more than a century [1]. Since, several terminologies have been proposed to qualify these episodes, including pure amnestic seizures [2], epileptic amnestic attacks [3,4] and, more recently, transient epileptic amnesia (TEA). The term of TEA, coined in the early 1990's by Kapur [5], was later refined to designate an epileptic syndrome with ictal and interictal memory disturbances as core clinical manifestations [6–8].

Diagnostic criteria for TEA include:

- a history of recurrent witnessed episodes of transient amnesia;
- cognitive functions other than memory are intact during typical episodes as observed by a reliable witness;
- evidence for a diagnosis of epilepsy provided by epileptiform abnormalities on electroencephalography (EEG), concurrent onset of other clinical features of epilepsy (e.g., lip-smacking, olfactory hallucinations), and/or a clear-cut response to anticonvulsant therapy – or by a combination of these three factors [6].

Although the concept to TEA is becoming increasingly recognized, its individualization as a full-blown epileptic syndrome remains of debate. This is partly due to the rarity of the disorder in a clinical setting and, consequently, limited available data from large well-investigated cohorts. To date, only three large series have been reported, respectively from Italy (30 cases), the UK (50 cases) and France (30 cases) [7–9]. That transient amnesia episodes could be related to a non-epileptic cause has also been advocated. Finally, and perhaps most importantly, the clinical relevance of delineating a new epileptic entity within an already dense epilepsy landscape has been questioned [10]. In this article, we will review the main clinical and electrophysiological characteristics of TEA, discuss its neuroanatomical substrate and putative pathophysiological mechanisms, with the overall aim to defend the idea that TEA deserves individualization as a distinct epilepsy syndrome.

2. Demographical data

TEA is usually viewed as a late-onset form of temporal lobe epilepsy [6,8]. The prevalence of TEA is unknown. TEA typically begins in the late middle-age period, with a mean age around 60 years [7–9], close to the age of onset of transient global amnesia (TGA) [11]. A slight predominance in male was found in two reports [7,9]. The median delay before diagnosis of TEA was 12 months in the English series [7]. In our experience, it was on average 3 years, but considerable variation was observed, from several weeks to more than 10 years [9].

3. Amnestic seizures

3.1. *Amnestic seizures*

Although amnestic seizures have been known for a long time [1], they have been poorly defined in the literature, probably because they are often confused with the more widely known syndrome of transient global amnesia (TGA). Pure amnestic seizures are not specific of TEA and can be observed in several forms of temporal lobe epilepsies (TLE), as demonstrated by video-EEG monitoring [2,12]. Depth electrode recordings suggest that the memory impairment is related to ictal discharges affecting the medial temporal lobe regions bilaterally. Yet, following electrical stimulation of the left amygdala in a TLE patient, a typical amnestic seizure was induced, that became clinically apparent when the epileptic activity spread to the right hippocampus, probably via the dorsal hippocampal commissure [2].

Features of amnestic seizures are often described in comparison with TGA. Anterograde amnesia occurs during a state of normal consciousness, with no or mild behavioral changes (perplexity, excessive quietness), and amnesia of the episode. Repetitive questioning, long considered as a highly suggestive feature of TGA [5], can however be found in 30 to 50% of TEA patients [7,9]. In contrast to TGA, partial recollection of the ictal event is occasionally reported (30% of cases in [9]).

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