Journal of the Neurological Sciences 381 (2017) 7–15

Contents lists available at ScienceDirect

Journal of the Neurological Sciences

journal homepage: www.elsevier.com/locate/jns

Review Article

Neuroimaging and cognitive functions in temporal lobe epilepsy: A review of the literature

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ARTICLE INFO

Article history:
Received 13 February 2017
Received in revised form 2 August 2017
Accepted 7 August 2017
Available online 08 August 2017

Keywords:
Temporal lobe epilepsy
Neuropsychology
Neuroimaging
Memory
Language
Executive functions

ABSTRACT

Temporal lobe epilepsy (TLE) is the most common form of refractory focal epilepsy. Neuroimaging researches have demonstrated structural abnormalities in several cerebral regions. Cognitive impairment has been frequently described as a potential comorbidity of long-term TLE. This review investigated the state of research regarding neuropsychological impairment and neuroimaging studies in TLE patients. Studies were found on PubMed and Web of Sciences databases, 412 publications were selected: only 20 articles met search criteria. Results showed significant alterations in multiple cognitive domains, particularly memory, executive functions and language. The reported findings showed that the involvement of various factors, including neurobiological abnormalities and clinical features, is responsible for the onset of cognitive impairment in epileptic patients.

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1. Introduction

Epilepsy is a chronic disorder affecting people of different ages, races and socioeconomic backgrounds. It has a prevalence of 5 to 10 cases per 1000 habitants [1]. The causes may be genetic or acquired. Young people are more frequently affected by genetic, congenital or developmental factors, while acquired causes, such as brain tumors or strokes, are more prevalent in older people.

Temporal lobe epilepsy (TLE) is the most common form of refractory focal epilepsy, with an incidence of 40% among epileptic patients [2]. The International League Against Epilepsy (ILAE) describes two main types of TLE: mesial temporal lobe epilepsy (mTLE), related to hippocampus, parahippocampal gyrus and amygdala alterations; and lateral temporal lobe epilepsy (lTLE), a less frequent type, where the onset of seizures is localized in temporal neocortex. The ILAE [3], supported by Electroencephalographic (EEG) examinations, classifies three forms of seizures, which originate in TLE: 1) simple partial seizures (SPS), characterized by retention of consciousness, 2) complex partial seizures (CPS), involving a disruption of normal awareness, and 3) secondarily generalized tonic-clonic seizures. SPS or "auras" are frequent in mTLE

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http://dx.doi.org/10.1016/j.jns.2017.08.007
0022-510X/

and they are mainly caused by hippocampal sclerosis (HS) [4]. The most frequent symptoms are: viscerosensory sensations such as olfactory and gustatory hallucinations [5], rising epigastric sensation often described as nausea; "affective auras" including fear, memory flashbacks, déjà vu, dreamy states, auditory and visual illusions and hallucinations and states of altered consciousness [6].

Cognitive impairment has been frequently described as a potential comorbidity of long-term TLE. Seizure frequency and a long disease duration seem to have a negative impact on cognitive impairment [7]. Memory is the most common cognitive domain impaired in TLE patients. However, recent studies [8,9] have reported the involvement of other cognitive abilities, including attention, language, praxis, executive function, judgment, insight and problem solving.

A lot of clinical factors, such as aetiology, age at onset disease, disease duration, seizure frequency and typology of therapy could have an adverse influence on cognition [10]. All these factors could change normal brain structure, metabolism, chemistry, and other dimensions of brain functions. Neuroimaging techniques play an important role in the comprehension of neurobiologic correlates of cognitive impairment in epilepsy. Quantitative MRI studies, in particular, have demonstrated structural abnormalities in hippocampus, amygdala, fornix, entorhinal cortex, parahippocampus, thalamus, basal ganglia, extrahippocampal temporal lobe regions and extratemporal areas [11].

The aim of this review is to explore the correlation between cognitive impairment and neuroimaging findings, in TLE subjects.

2. Materials and methods

This review focused on cognitive functions of TLE patients and their neurobiological correlates. Studies were found on PubMed (from June 1974, year of the first-related published article to November 2016) and Web of Science databases (from 1996 to November 2016). Keywords used were: "temporal lobe epilepsy AND neuropsychological AND neuroimaging" ("epilepsy, temporal lobe"[MeSH Terms] OR ("epilepsy"[All Fields] AND "temporal"[All Fields] AND "lobe"[All Fields]) OR "temporal lobe epilepsy"[All Fields] OR ("temporal"[All Fields] AND "lobe"[All Fields] AND "epilepsy"[All Fields])) AND "neuropsychological"[All Fields] AND ("neuroimaging"[MeSH Terms] OR "neuroimaging"[All Fields]). Only English texts were considered. Articles were selected based on title, abstract and text. They had to meet the following inclusion criteria:

1. Published peer-reviewed research;
2. Studies included patients with TLE and a healthy control (HC) group;
3. Studies which specifically assessed the relationship between cognitive functions and neuroimaging techniques;
4. All studies which used standardized neuropsychological measures;
5. Studies with pediatric population were excluded;
6. Case studies were excluded.

3. Results

Of the 412 articles identified, only 20 articles met the inclusion criteria (Fig. 1). All studies investigated the relationship among TLE and cognitive functions and their neurobiological correlates (Table 1–4).

For the purpose of this review, we divided the different studies according to the cognitive impairment:

1. Multiple cognitive impairments;
2. Memory deficits;
3. Executive and attentional deficits;
4. Language deficits.

3.1. Multiple cognitive impairments

Seven of the 20 selected articles, reported an impairment of multiple cognitive domains in TLE. In particular, they focused on intelligence, memory, language and executive functions (Table 1).

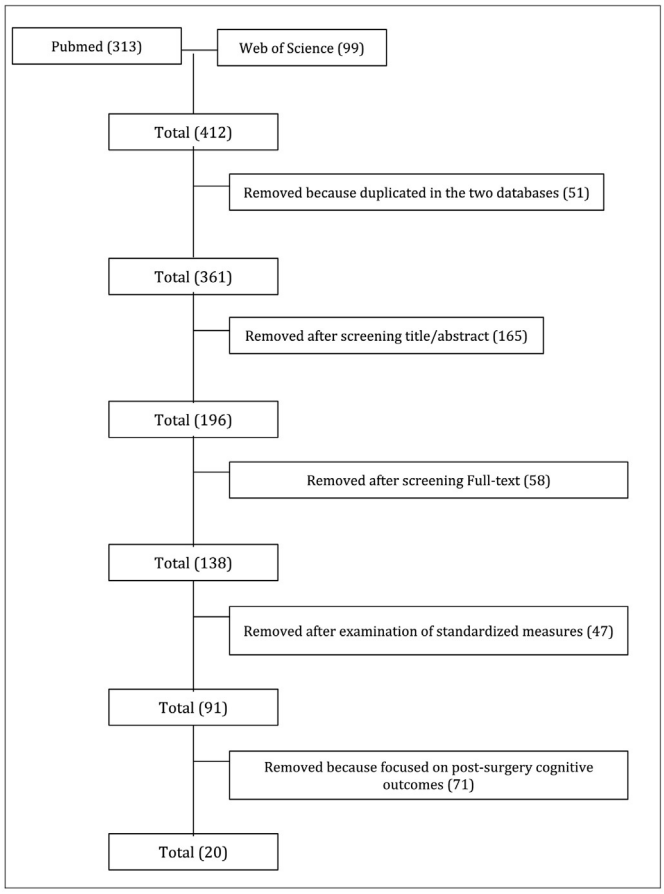

Fig. 1. Procedure of selection of eligible articles.

Oyegbile et al. [12] investigated the relationship among cognition, neuroimaging and clinical seizure features. A sample of 96 patients with TLE and CPS and 82 HC underwent Magnetic Resonance Imaging (MRI) and neuropsychological evaluation to define how the clinical seizure features and volumetric abnormalities could affect cognitive domains. MRI results showed an increase of total cerebrospinal fluid (CSF) and a significantly decrease of total white and grey matter volume if compared to HC. The disease duration could be an important predictive marker of cognitive impairment and it may be related to volumetric abnormalities in grey matter volume. These findings indicate that cortical brain volume is strictly correlated to neuropsychological functions.

In another study, the same author [13] considered the possible link between cortical surface features and cognitive performances. The analysis of gyral and sulcal curvature and total cortical surfaces area in 46 TLE patients and 48 HC, showed significant differences in cortical morphology between two groups, with abnormalities in gyrification and surface cerebral spinal fluid in TLE patients. These results were associated with lower performance in all cognitive domains.

Riley et al. [14] studied the effects of white matter tract abnormalities and clinical factors on cognitive functions. Twelve patients with TLE and 10 HC underwent Diffusion Tensor Imaging (DTI) and a neuropsychological evaluation. DTI results showed a reduced white matter fractional anisotropy (FA) in four specific regions: anterior temporal lobe, posterior mesial temporal lobe, cerebellum (in the epileptogenic cerebral hemisphere); and frontoparietal lobe (in the contralateral cerebral hemisphere). These white matter tracts alterations are associated with significant cognitive and clinical impairments. In particular, the mesial temporal lobe FA, including fornix and posterior inferior longitudinal fasciculus, are involved in immediate memory performances. The anterior temporal lobe FA, that includes uncinate fasciculus and anterior aspects of inferior longitudinal fasciculus, is associated with delayed