Memory disturbances and temporal lobe epilepsy simulating Alzheimer’s disease: a case report

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Summary

We report the case of a patient presenting with recurrent transient dyscognitive episodes and memory disturbances, simulating the clinical picture of early Alzheimer’s disease (AD). Neuropsychological examination showed only episodic memory impairment without significant progression over time in the absence of any other cognitive dysfunctions; magnetic resonance imaging failed to show selective temporal atrophy. The suspicion that a nocturnal epileptic seizure had occurred arose on the basis of a clinical report of tongue biting and the findings of sharp waves over the temporal region on standard EEG. Thus 600 mg/day of carbamazepine was added to donezepil therapy. Antiepileptic treatment completely reversed the cognitive disturbances. Our observation stresses the possibility that unrecognized epilepsy may present as early AD.

KEY WORDS: Alzheimer’s disease, epilepsy, memory impairment, neuropsychological evaluation, MRI.

Introduction

Memory impairment is the most frequent symptom of Alzheimer’s disease (AD) in the early stages. On the other hand, memory dysfunction is also widely reported in epilepsy (1,2). The literature contains a few reports of epilepsy patients with memory decline incorrectly diagnosed with AD (3). Here, we report the case of a subject with memory impairment and epileptiform EEG abnormalities who was misdiagnosed with AD, and whose cognitive dysfunctions were completely resolved by anti-epileptic therapy.

Case report

A 67-year-old man had presented disturbances of memory function associated with episodes of spatial disorientation and prosopoagnosia since 1998 (two years before he came to our attention). After one year, these episodes started occurring 2-3 times a month, usually in the evening, each one lasting 1 minute and being followed by unresponsiveness. One night he awoke with a morsus on his tongue. Given the lack of other relevant clinical observations, the occurrence of nocturnal epileptic seizure could not be proved with certainty. In January 2000 his family doctor requested a neurological consultation. General and neurological examinations were normal. Neuropsychological testing was performed in an attempt to establish the presence of cognitive impairment consistent with dementia. An extensive examination, including Mini Mental State (4) and tests exploring different cognitive domains (5,6), was performed. The raw scores, adjusted for age, sex and years of schooling according to Italian standardised parameters (5), were transformed into equivalent scores, ranging from 0 (fully impaired – 2.9% of total population) through 1 (borderline – 10.4% of total population) and 2, 3, and 4 (normal). This evaluation showed moderate impairment of episodic memory, verbal fluency (semantic categories) and mild temporal disorientation (Table I, see over).

A standard electroencephalographic (EEG) recording showed bilateral slow abnormalities in the range of theta and delta frequencies and sporadic sharp waves prevailing over the temporal regions of the right hemisphere. The EEG abnormalities remained unchanged during 3 minutes of hyperventilation and standard intermittent light stimulation. Brain MRI showed moderate dilatation of subarachnoid spaces, minimal cerebellar atrophy and thinning stem associated with considerable ventricular enlargement; no selective atrophy in temporal structures could be detected. Clinical and neuropsychological findings were considered compatible with the early stages of AD. In February 2000 the patient began treatment with donepezil, initially 5 mg/day for three months, then 10 mg/day.

The EEG findings gave rise to the suspicion that the episode experienced by the patient during nocturnal sleep had actually been an epileptic fit. Thus, the patient was given slow-release carbamazepine, up to 600 mg/day. By September 2000, the patient’s episodes of prosopoagnosia and spatial disorientation had disap-
appeared, while his memory difficulties persisted, even though no interference with occupational or social activities was reported. Furthermore, since February 2000, when carbamazepine treatment was started, the patient has reported no further critical nocturnal episodes. The patient came to our attention in October 2000. A second neuropsychological assessment was carried out (Table I); cognitive performance was within the normal range, as shown by the improvement (from 0 to 4) of equivalent scores in the memory domain. In March 2001 the patient reported complete resolution of his memory disturbances. The previous diagnosis of AD seemed to be unlikely and therefore therapy with donepezil was interrupted, while the patient continued to take carbamazepine at the same dosage. A year later, in May 2002, a third neuropsychological assessment was carried out; the results were entirely in line with those of the previous assessment and still normal (Table I). The patient no longer complained of memory disturbances.

Discussion

In accordance with a few previous reports that have appeared in the literature (3), this case demonstrates the possibility of diagnostic misclassification in the initial phase of AD. While memory disturbances characterise AD in the early stages, epilepsy is also frequently accompanied by memory dysfunctions. Equally, EEG abnormalities and critical episodes can also occur during the course of AD. It has been established that the presence of dementia is associated with an at least six-fold increased risk of unprovoked seizures (7). Patients of a younger age at dementia onset have been reported to be particularly susceptible to seizures, but generally epilepsy appears later in the course of the disease (8). These factors undoubtedly increase the risk of misdiagnosis. In our case, the memory impairment was not characterised either by interference with daily and occupational activities, or by the progressive worsening over time that is typically seen in AD. Furthermore, neuroradiological investigation did not give evidence of selective temporal atrophy, which is considered a sign highly suggestive of AD in the absence of other specific markers (9-11). Indeed, Frisoni et al. (11) report that measures of temporal lobe atrophy have a sensitivity of about 80% in the early diagnosis of AD and therefore constitute an accurate marker, that could be used routinely in the clinical setting. After anti-epileptic therapy was initiated no further episode of prosopagnosia or spatial disorientation occurred and neuropsychological testing documented an improvement of memory function. All in all, these findings supported the hypothesis that the patient's dyscognitive episodes were actually temporal lobe epileptic seizures and that their recurrence, together with the memory dysfunction, mimicked early AD. Remission of epileptic amnesic attacks after anti-epileptic treatment has been reported in the literature (12). Unfortunately, neither our observations nor those present in the literature were based on long-term video EEG or 24-hour ambulatory EEG documentation of epileptic seizures or discharges, thus making the hypothesis a deduction based solely on the clinical evidence and interictal EEG findings.

Epilepsy presenting as AD has been reported in the literature (3), although the resolution of the memory impairment was incomplete, and there was a degree of fluctuation in cognitive performance, which nevertheless still contrasts with the steadily progressive deterioration that is typical of AD. The memory improvement observed in our patient cannot be related to the intake of donepezil; in fact, withdrawal of the drug did not cause any worsening of his cognitive status, as documented by the third neuropsychological examination performed several months after

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Table I - Neuropsychological evaluation: raw scores and () equivalent scores
the withdrawal, which remained within the normal range.
We wish to underline the importance of correct application of clinical (13,14) and neuroradiological criteria in order to avoid misclassification in the early phases of AD. In situations like the one described here, characterised by the presence of elements not completely consistent with AD and elements that can generate a suspicion of epileptic seizures, it is, in our view, very important to consider the possibility of differential diagnosis, possibly exploiting findings provided not only by standard EEG but also by long-term EEG investigations.

References