Case Report

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Epileptic Pseudodementia by Antidepressants

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Abstract

The authors present a case of a patient who previously had depressions and that remains asymptomatic for several years, but after an uncontrolled increase of tricyclic antidepressant clomipramine, an important cognitive alteration appeared that remained even after removing the drug with which it was related. Frontotemporal dementia was diagnosed but we think that it could be a false dementia and postulate empirically that the activation of an irritative frontotemporal focus would be the cause of the cognitive interference. The possibility of a pseudodementia associated with irritative foci or "epileptic pseudodementia" has to be valued in order to alert clinicians given the importance that has the binomial diagnosis-prognosis in dementia. The authors know of no previous records of occurrence of a "dementia" diagnosed as such in neurology, and that really is caused by stimulation of a pre-existing epileptic irritative focus (pseudodementia).

Keywords: Dementia; Pseudodementia; Epilepsy; Irritative focus; Antidepressants; Clomipramine

Introduction

The pseudodementia is a false dementia which usually tends to be a depressive type. The clinical depressive pseudodementia (DPD) occurs with changes in cognition and behavioral alterations that can be confuse the diagnosis of a depressed elderly person with dementia [1].

It is a disorder similar to dementia without being caused by an organic brain disease and can be reversed with a proper treatment [2].

The term of pseudodementia began to be used in the year 1900 by Wernicke in relation to the mental manifestations of some chronic hysterical reactions. Its use began generalized in 1961 when Kiloh considered the depression as the condition of the elderly that can be more easily confused with dementia [3]. It gains since then especially importance the concept of depressive pseudodemencia.

Depressive disorders, especially in the elderly have, as well as alterations of mood and somatic manifestations, multiple cognitive disorders [4], largely determined by the lack of motivation and the bradypsychia (slowness of mental reactions), of sufficient intensity as to be confused with dementia. There is a high incidence of depressive illness on Alzheimer's disease and also it must be taken into account the depressive pseudodementia among the elderly [5,6].

Case Report

Our case is about a woman 66 years old diagnosed of recurrent depressions, who has had two episodes. The first episode occurred when she was 49 years old and the second one with 57 years old.

Since she recovered from her latest depression, she has being taking continuously the antidepressant clomipramine (tricyclic classic, serotonin and norepinephrine reuptake inhibitor [7]) with a maintained dose of 75 mg day and lithium (800 mg/day, with plasma levels within the therapeutic range). She has remained emotionally stable (euthymic) and performing her professional activity with normality, being that this includes an important intellectual activity as a teacher of the University, imparting conferences and preparing masters.

Six years ago (60 years old), still asymptomatic of her depressive illness, she began to suffer confusions and disorientations; she wanted to go to teach her students in the middle of the night, she did not recognize her husband, mistaking him for his cousin, inappropriate responses to questions and difficult to follow conversations, she also did not recognize her usual address. Also suffered from hallucinations in which she spoke with a few people who said were Indians.

Little fluid language, problems in understanding, alteration of executive functions, altered praxis, slowing the speed of processing, global bradykinesia, but no rigidity or tremor had been noticed.

The evolution was progressive with worsening during three months. Then she was neurologically studied and performed the following tests:

PET: Glucidic hypometabolism bilateral frontal cortex and parietal left "suggestive of frontoparietal dementia".

MRI: Bilateral atrophy temporal and parietal.

EEG: Slow waves of high and medium voltage in anterior regions 'Irritative bilateral frontotemporal signs'.

She also has a history of hypertension, hysterectomy and tonsillectomy. Moreover a subclinical hypothyroidism it was detected (who was treated with sodium levothyroxine, 50 micrograms). The diagnosis of the hypertension was 8 years ago and it was treated with ACEi. At the moment of this diagnosis, the patient was euthymic and it did not influence the evolution of her depression. In relation with the hysterectomy (uterine fibroids) and tonsillectomy, the diagnoses and treatments were done over 20 years ago.

It was detected that the cognitive impairment began three months ago when there was a mistake in the taking of clomipramine antidepressant and instead of taking three tablets of 25 mg daily she was taking three tablets of 75 mg daily, having gone from a daily dose of 75 to 225 mg/day.

The anticholinergic toxicity or anticholinergic syndrome (ACS) includes: Confusion, disorientation, loss of short-term memory, inability to concentrate, inability to keep an idea of thought or flight of ideas, incoherent speech, illogical thinking and hallucinations. Acute anticholinergic syndrome is reversible and persists until the supraphysiological levels decrease by excretion of the substance. Thus, the anticholinergic effects of tricyclic antidepressants in elderly patients can cause confusion. The clomipramine has a special affinity for alfa adrenergic and muscarinic acetylcholine receptors [7].

It was considered that the antidepressant could, by this cholinergic effects, have been the cause of such an important cognitive impairment and so it was deleted; only being prescribed lorazepam 3 mg/day and haloperidol 5 mg daily.

The clinical evaluation for the next 6 months found that visual hallucinations had disappeared but remained the cognitive impairment, the difficulty of carrying out executive functions, orientation and recognition of family members. The mini-mental state examination (MMSE) provided a value of gravity very important, having a score of 9 (cutoff point 23 of a possible 30).

Then the neurological diagnosis was made as frontotemporal dementia and it was prescribed the acetylcholinesterase inhibitor rivastigmine once-daily transdermal patch 9.5 mg/24.

Six months after treatment with cholinesterase inhibitor there was no significant improvement. She was not able to write, with no coordination using the PC, and when she tried to write by hand she could not understand what she was writing, the bradykinesia remained, she could not follow a conversation and although she recognized her husband she still was disoriented at home.

During our visit we made at that time, we raised the empirical hypothesis that instead of a dementia it was a "false dementia" (pseudodementia) and that all cognitive alteration was caused by activation of the irritative frontotemporal focus that would lead to the cognitive interference. We added carbamazepine to the treatment and programmed a gradual increase dose until she got within a month a dose of 600 mg per day.

Two months after she was taking the full dose of carbamazepine (600 mg/day) we found a full recovery of her cognitive abilities, being able to read, write, prepare lessons and perform all of her intellectual normal activity, although it appeared insomnia and mild depressive symptoms. Because of this we added to the treatment venlafaxine 75 mg at breakfast and 50 mg of trazodone at night, and two months later she was with normal mood (euthymic) and without any cognitive impairment. Four years later she continues with the same medication (venlafaxine, rivastigmine and trazodone) and only in 2016 (four year euthymic) she suffered a mild depressive oscillation for 3 months, that corrected with a temporary increase of 37.5 mg of venlafaxine, following her emotional and intellectual normal life until now in 2017.

Discussion

The difficulty of concluding a diagnosis as neurocognitive disorder (NCD) due to another medical condition as is shown by the DSM 5 [8], is that certainly we have the temporal relationship between the onset of the medical condition (increase of the dose of antidepressant clomipramine) and the development of the cognitive deficit. However in the NCD the diagnostic certainty is increased when the cognitive deficits improve in the context of the treatment of the medical condition, in our case the suppression of clomipramine. And the latter did not take place.

According to the consensus of Lund-Manchester [9] on the clinical criteria of frontotemporal dementia our patient suffers; a behavior disorder, early loss of insight and personal and social consciousness, mental rigidity, tendency to distraction, emotional blunting, apathy, facial amimia (reduction in facial expressiveness) and reduction of expressive speech, brain imaging (structure or functional) abnormality and beginning before age 65.

The diagnosis of frontotemporal dementia was supported by the lack of pseudodemential characteristics, [10] the clinical data, the lack of improvement when it was eliminated the possible cause of cognitive disturbance and by the neurological exam.

Epilepsy is a clinical entity can be associated with convulsive ways. Only by having an irritative focus we cannot diagnose of epilepsy. It is very important to know, if they exist, the interictal epileptiform discharges (IED), the irritative zone [11]. In our case we can understand that this dysrhythmic cerebral activation has been the reason for the neurocognitive alterations which has given rise to confusion with dementia. Especially because the irritative focus was found at the frontotemporal lobes.

We believe that the empirical hypothesis of an interference of the frontotemporal irritative focus is the more plausible since the dementia symptoms have disappeared after specific antiepileptic treatment.

The definitive diagnosis must often be postponed until follow-up, after the recovery of a medical illness [11].

We especially consider the importance of rating all alternative possibilities before a diagnosis of dementia disease that has an important handicap in the future prognosis.

There is an extensive bibliography on the cognitive, emotional and confusional effects of different drugs and in the same way there is a very extensive bibliography on psychiatric alterations in relation with different medical pathologies [12-14]. In the first case, when the substance that causes the cognitive alteration disappears, the disease also disappears. In the second case, it is sometimes necessary to treat the psychiatric disease regardless of the overall medical illness.

Likewise, with regard to epilepsy have been published numerous articles that relate to mood alterations and changes in consciousness [15]. It has been associated the treatment with clomipramine with few unusual side effects as yawning-orgasm [16] but we have not found any bibliography that relates a continued state of “dementia” with the stimulation of an irritative focus.

Conclusion

The limitation of our empirical findings is that we should have suppressed the treatment with carbamazepine to check if the cognitive disorders returned or if we should cause stimulation with clomipramine (or with some activating substance of the irritative focus). But in any case none of these options was ethically viable.

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