v. 1, n. 1, 2021

International Journal of Health Science

EARLY ALZHEIMER'S DISEASE

Laura Cristina Ribeiro Cangue
University Center of Belo Horizonte
Belo Horizonte - Minas Gerais
https://orcid.org/0000-0002-5652-030X

Ludmila Rodriguez Augusto
University Center of Belo Horizonte
Belo Horizonte - Minas Gerais
http://lattes.cnpq.br/0544831568910024

Maria Teresa Hosken dos Santos
University Center of Belo Horizonte
Belo Horizonte - Minas Gerais
https://orcid.org/0000-0001-7160-8257

Carolina Pimentel Orsini
University Center of Belo Horizonte
Belo Horizonte - Minas Gerais
http://lattes.cnpq.br/4484706317314910

Negilson Kaizer Correia Cosme
Federal University of Minas Gerais
Belo Horizonte - Minas Gerais
https://orcid.org/0000-0003-0948-6601

Sergio Randt Barbosa
University Center of Belo Horizonte
Belo Horizonte - Minas Gerais
http://lattes.cnpq.br/0574324663645706

Alice Ramalho Tavares
University Center of Belo Horizonte
Belo Horizonte - Minas Gerais
https://orcid.org/0000-0003-4448-9103

Anne Lara Moreira Gomes
University Center of Belo Horizonte
Belo Horizonte - Minas Gerais
http://lattes.cnpq.br/1197882835974488

Eduarda Luiza Loschi de Araújo
University Center of Belo Horizonte
Belo Horizonte - Minas Gerais
https://orcid.org/0000-0003-0612-8729

Gabriela Raposo Tavares
University Center of Belo Horizonte
Belo Horizonte - Minas Gerais
http://lattes.cnpq.br/1796269366563551

Larissa Gobbi Colares
University Center of Belo Horizonte
Belo Horizonte - Minas Gerais
https://orcid.org/0000-0001-9162-0106

Abstract: Alzheimer's disease (AD) is a neurodegenerative pathology, clinically defined by slow and progressive decline of cognitive functions, being divided into 2 subgroups according to its onset time. Early AD is characterized when the diagnosis is made before the age of 65 and has a rapid cognitive decline. These are rare cases, which correspond to 5% of the total and a family involvement is observed in successive generations. The diagnosis of AD is based on the clinical picture and can be complemented by imaging. This report aims to expose the history of a 64-year-old female patient diagnosed with early AD at the age of 59, the evolution of the clinical picture, the possible treatments and the impact of the disease on the life of the patient and family members. For this, a literary review was performed in the Scielo and PubMed databases, in addition to the analysis of medical records, neuropsychological evaluation and imaging of the patient.
Keywords: Alzheimer's disease, presenile, early dementia.

INTRODUCTION

Alzheimer's disease (AD) is a progressive neurodegenerative disorder that develops with cognitive deterioration, impaired daily activities, and neuropsychiatric symptoms. It can be classified as early onset, when there is rapid decline in cognitive functions before 65 years of age, are rare cases, which correspond to 5% of the total (FALK, 2018). There are no biological markers for early AD, diagnosis is made by clinical evaluation and exclusion of other dementias. Due to the intensity of the presenile impact of AD, including social and occupational repercussions, it is essential that the diagnosis is made early, because therapeutic interventions have beneficial effects on the patient's quality of life and delay the progression of the disease (TRUZZI, 2005).

CASE REPORT

S.F.B, 64 years old, born in Belo Horizonte, with 2 higher education courses, diagnosed in 2015 with early AD at the age of 59. In his initial neuropsychological evaluation, severe short- and long-term episodic memory difficulty was evidenced with impact on executive functions, temporal disorientation and language alteration in semantic and phonemic verbal fluency aspects, being classified as mild dementia by the Clinical Dementia Rating (CDR 1) scale. In addition, magnetic resonance imaging (MRI) was observed in brain volumetric reduction, ventriculomegaly and focus suggesting partial loss of myelin by microangiopathy. Single photon emission computed tomography (SPECT) showed mild cerebral perfusion heterogeneity without a specific scintigraphy pattern.

The main findings that contributed to the diagnosis were: insidious neurocognitive decline, with recent initial memory loss, speech alterations, progressive dysfunction of basic activities of life and temporal disorientation, associated with volumetric reduction of the encephalic parenchyma with ventriculomegaly, suggestive of partial loss of myelin by microangiopathy and bilateral hippocampal reduction on MRI.

In 2016 there was a significant worsening in the neuropsychological evaluation, with loss of 3 points in the mini mental state examination (MMSE) and 10 points in the functionality scale, in addition to significant change in behavior, and donepezil 5 mg was started. However, the patient did not tolerate the medication, presented nausea, vomiting, abdominal pain and vertigo. The drug was replaced by adhesive rivastigmine 10 9.5 mg/24 hours, with good tolerance, which favored treatment adepitis and the stability of cognitive decline in subsequent years.

In 2018, the patient began to present sleep fragmentation with psychomotor agitation, a condition suggestive of sunset syndrome, and the use of escitalopram was started. In the following year, there was behavioral worsening and maintenance of fragmented sleep, being introduced memantine, indicated Mediterranean diet and neurobehavioral therapy, with stabilization of the condition until the present moment.

DISCUSSION

(AD) is a progressive and fatal neurodegenerative disorder manifested by cognitive deterioration, progressive impairment of activities of daily living and a variety of neuropsychiatric and behavioral symptoms, with high genetic complexity (TRUZZI, 2005). AD when diagnosed before 65 years is considered early or family, represents 5% of all cases, has a pattern of autosomal dominant inheritance, especially in the APP, PSENI, PSENII genes, with involvement of numerous individuals in each generation, different from late-onset AD that has no predominance of family aggregation and is linked to polymorphisms in the APOE gene (FALK, 2018).

AD generally settles insidiously and develops slowly and continuously for several years, but early-onset AD has a faster evolution, with more acute involvement (TRUZZI, 2005). Cardiovascular disease, traumatic brain injury, psychiatric illness, alcohol abuse and estrogen-related factors have been identified as potential risk factors for early AD and there is a relationship with cumulative severity of exposure (RYMAN, 2014).

Neuropathological and biochemical changes in AD can be divided into two areas: structural changes and changes in neurotransmitters (FALK, 2018). Structural changes include the complicated neurofibrillary, neuritic plaques and changes in amyloid metabolism, as well as sinaptic

losses and neuronal death, and the apoptotic process in neuronal and glial cells represents a significant aspect in the pathology of early AD. Some neurotransmitters are significantly affected indicating a pattern of system degeneration (BARBER, 2017).

Currently, the diagnosis of AD is clinical and is linked to neuropathological changes in the disease that trigger symptoms of cognitive impairment (FALK, 2018). The onset of the disease can be observed by the damage to instrumental activities of daily living such as paying bills, remembering to take medications, forgetting the stove lit. The progression of the disease can further impair the activities of daily living, including difficulty eating, bathing, dressing, going to the bathroom, walking. Although physical examination findings are generally normal in patients with dementia, they can help identify potentially reversible causes of cognitive decline, including hypothyroidism, vitamin deficiencies, neurosyphilis, intracranial tumors, normal hydrocephalus pressure, depression, and hypoperfusion of heart insufficiency (BRASIL, 2013).

Some tests are performed to assess the degree of cognitive impairment, with MMSE being the most commonly used cognitive assessment tool, with a sensitivity of 89% and specificity of 81% for dementia detection (FALK, 2018). Patients who have confirmed cognitive impairment should be examined for depression, should receive laboratory tests for other common disorders that may cause cognitive impairment, and should undergo brain imaging (BRASIL, 2013).

The preferred imaging is MRI, but it is not performed to make the diagnosis, but to exclude other abnormalities such as stroke, subdural hematoma, hydrocephalus or some tumor. In patients with rapidly progressive symptoms, CSF analysis should be considered to exclude infectious processes (FALK, 2018). The role of CSF biomarker tests for Alzheimer's disease in clinical practice has not yet been established, it is often used to analyze treatment progression and prognosis (BARBER, 2017).

The treatment of AD should be multidisciplinary, involving the various signs and symptoms of the disease and its peculiarities. Drug treatment aims to stabilize cognitive impairment (TRUZZI, 2005). Acetylcholinesterase inhibitors, cholinergic drugs donepezil, galantamine and rivastigmine are considered first-line, all of which are recommended for the treatment of mild to moderate AD. The patient should be monitored every 6 months to estimate the benefit and need for continuity of treatment through clinical evaluation and performance of the MMSE and the CDR tests (BRASIL, 2013).

The early onset of AD can be confused with psychiatric conditions, especially when there is no family history of the disease. Due to the great impact on the life of the patient and the family, it is of paramount importance that the diagnosis be made early so that the treatment is instituted in the early stages of the disease, in order to improve the quality of life of the patient and reduce the burden of family members and caregivers (CATIONS, 2016).

CONCLUSION

Due to the intensity of the presenile impact of AD, both for the patient and his/her family, including social and occupational repercussions, it is essential that clinicians and geriatrics diagnose this comorbidity early, because the therapeutic interventions performed in a timely manner have beneficial effects on the patient's quality of life and delay the progression of the disease.

REFERENCES

1. BARBER, Imelda S. *et al*. **Mutation analysis of sporadic early-onset Alzheimer's disease using the NeuroX array.** Neurobiology of Aging,v. 49, p. 215. e1-215. e8, 2017.

2nd CATIONS, Monica *et al*. **What is the role of modifiable environmental and lifestyle risk factors in young onset dementia?** European Journal of Epidemiology,v. 31, n. 2, p. 107-124, 2016.

3. Falk N, Cole A, Meredith TJ. **Evaluation of Suspected Dementia.** American Family Physician. v. 97, n. 6, p. 398-405, 2018.

4. Ministry of Health. Ordinance no. 1,298, of November 21, 2013. **Protocolo Clínico e Diretrizes Terapêuticas da Doença de Alzheimer. Diário Oficial da União 2013.**

5. RYMAN, Davis C. *et al*. **Symptom onset in autosomal dominant Alzheimer disease: a systematic review and meta-analysis.** Neurology, v. 83, n. 3, p. 253-260, 2014.

6. TRUZZI, Annibal; Laks, Jerson. **Enfermedad de Alzheimer esporádica de inicio precoz**. Archives of Clinical Psychiatry, v. 32, n. 1, p. 43-46, 2005.