Does structural MRI correlate with a stage of Alzheimer's disease – a case report

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Abstract

Background: Cortical atrophy is discussed as a topographical marker of the Alzheimer's disease (AD). Structural MRI has been shown to be sensitive in detecting brain changes associated with AD. However, data are conflicting regarding the correlation of MRI findings with the clinical stage of AD.
Case presentation: Here, we report the case of a 74-year-old white woman presented initially with predominantly cognitive complains. AD in a stage between mild and moderate was suspected. In contrast, MRI showed bilateral severe atrophy of entorhinal cortex and hippocampus. Within following seven months disease progressed with development of psychosis. We suggest that clinical presentation and neuroimaging data were dissociated at the first presentation in the neurological setting.
Conclusions: This case represents an interesting presentation which brings arguments into hypothesis of predictive value of neuroimaging data for diagnosis of AD. **(TCM-GMJ August 2025; 10 (2): P32-P35)**

Keywords: dementia, structural MRI, psychosis, disease stage, Alzheimer's disease

Introduction

Dementia is a group of predominantly progressive neurodegenerative syndromes that affect the brain and clinically manifested by a decline in memory and other cognitive skills. The prevalence of all-type of dementia is increasing with age and global dementia cases expected to triple by 2050 due to the aging population (1, 2). The most prevalent type of dementia is Alzheimer's disease (AD) commonly affecting older adults. AD has a severe impact on both the patient and his family and places a great burden on the healthcare system. Global economic cost of AD comparable with the costs of such dangerous diseases as cancer, coronary heart disease and stroke. Researchers projected that this economic burden would rise substantially by 2050-60 for dementia (3, 4). In particular, in the USA, formal care cost will increase from $196 billion in 2020 to $1.4 trillion dollars in 2060 (5).

Definitive diagnosis of AD requires post mortem neuropathologic brain examination to detect specific changes (6, 7). Therefore, a probabilistic diagnosis of dementia ('probable AD') was widely introduced, which, along with the obligatory clinical manifestation, was based on the results of neuropsychological scales and structural neuroimaging (computed tomography or magnetic resonance imaging (MRI) and electrophysiological (EEG) studies (8). In recent decades, the situation has changed and it is possible to make clinicobiological diagnosis of dementia during the patient's lifetime (in vivo). Pathophysiological biomarkers obtained from cerebrospinal fluid and/or peripheral blood and amyloid positron emission tomography (9, 10, 11, 12) are used for this purpose. The anatomical pattern (topographical biomarker) of AD neurodegeneration is brain atrophy, particularly in the temporal and parietal regions, which is greater than that observed with normal aging. Subtypes of AD with cortical (associational cortex) or limbic (entorhinal) atrophy have also been identified (13). However, there is still no consensus on whether the degree of atrophy (volume loss) correlates with the clinical manifestation of the AD (disease severity). The case presented below may be of interest as an analysis of this association.

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Case report

A 74-year-old white woman was brought by her husband to the office for neurologic evaluation due to the following complaints: the patient could not recognize her husband, forgot the names of close relatives, was unable to fully fulfill her duties as a housewife (cooking food), and had dramatically changed her behavior. During this period, a magnetic resonance imaging examination of the brain was performed. A diagnosis of Alzheimer disease (AD) was suspected by a neurologist on the basis of an MRI data and clinical assessment. The patient was prescribed rivastigmine and memantine. Unfortunately, we do not have medical documentation from this period for a more detailed information.

The patient underwent a colectomy with colostomy due to a colorectal tumor 18 years ago. Otherwise, her past medical history was not remarkable. She has a higher education and worked in a financial department in a factory for a long time.

After three and a half months, she came to our department for a second opinion. At this point, the following were noted: repeated stereotypical aberrant behavior, neglect of bowel hygiene with the bowel exposed to the abdominal surface, sometimes wearing clothes backwards, disinhibited excessive talking, laughing for no reason (euphoria), confusion of orientation and location, inability to recognize relatives and spouse. She periodically showed agitation and resisted her spouse, in particular, questioning the need for treatment, although he still managed to convince her and take medication. Mini-Mental State Examination (MMSE) score was 16 points, Katz Modified Daily Activity Index (ADL) - 4 points, Montreal Cognitive Assessment (MOCA) score was 11/30, Clinical Dementia Rating Scale (CDR) 1 point. The condition was assessed as mild AD, although its extreme spectrum. The decision was made based on clinical assessment and semi-structured interview with the patient and her husband performed by experienced neurologist. Additional treatment with sertraline and hydroxyzine was prescribed.

After 4 months, the clinical picture showed acute psychotic symptoms: delusions (she thinks that there are uninvited guests in the house, strangers are talking to her on TV and she enters into communication with them) and hallucinations: auditory, olfactory and visual. At the last examination, the MMSE score was 10, the ADL score was 1, and the MOCA score was 6/30. CDR score was 2. The disease progressed to a clearly defined moderate severity stage.

At the initial visit, magnetic resonance imaging of the brain was performed using T1se, T2se, T2tse_dark_fluid, DWI sequences with sagittal, coronal and axial slices acquired. There was a symmetrical decrease in the volume of the temporal lobe, a moderately dilated ventricular system, and a reduced hippocampus in volume. Overall, there was moderate atrophy, temporal type, with a global cortical atrophy scale (GCA) score of 2 (14) and medial temporal lobe atrophy (MTA) score of 3 (Fig.1, Fig.2).

Discussion

Cortical atrophy has long been considered an important diagnostic tool for AD (10, 15). Many studies consistently highlighted distinct neurodegenerative patterns across AD phenotypes (16, 17). Starting from preclinical AD disease progression over time leads to accelerated atrophy rates of distinct medial temporal lobe subregions (entorhinal cortex and amygdala) (18, 19). This typical pattern of disease progression opened possibilities for the early diagnosis of AD by investigating these brain parts with imaging techniques.

Studies have shown that changes on MRI can be detected in the early or even preclinical stages of dementia, when only mild cognitive impairment is present (19, 20) and therefore be a predictor of the development of AD (21). However, a Cochrane systematic review did not confirm the diagnostic value of MRI in the early diagnosis of dementia in individuals with mild cognitive impairment (22). Structural magnetic resonance imaging is only one neuroimaging tool for dementia and is not a universal biomarker, like any imaging study taken alone. It has its own limitations, since MRI lacks the molecular specificity to directly determine the neural source of volume or thickness loss. Cerebral atrophy, hippocampal atrophy, or ventricular dilation are often present to some degree in normal aging and other diseases, which may be caused by factors other than neurodegeneration (23).

It is noteworthy that our clinical case represents a kind of dissociation between neuroimaging data and the disease clinical stage. The neuroimage scans taken immediately after first visit to a neurology setting show gross symmetrical cortical atrophy with a significant decrease in hippocampal volume, while the severity of dementia ranged from mild to moderate according to the clinical and neuropsychological assessment performed several months later. Over the next 4 months, the patient's condition deteriorated significantly, and psychotic symptoms appeared in the clinical picture. In our opinion, this clinical picture is more consistent with the neuroimaging findings obtained seven months ago.

Over half of people with AD will experience psychotic symptoms during their illness. Studies confirm that psychoses often occur in patients with moderate severity of AD (24). The delusions, auditory and visual hallucinations described in our clinical case precisely represent the typical misidentification subtype of psychosis in patients with Alzheimer's disease (25, 26). The coincidence of phenomenology and its late onset during the course of AD confirm that psychosis is associated with AD and not independent mental phenomenon. Psychosis in AD patients is associated with a pronounced decrease in the volume of certain areas of the brain's gray matter, in contrast to AD patients who did not have psychosis. These areas are involved in cognitive control, memory processing and perception, which may be the basis for the manifestation of psychosis (27, 28). Therefore, it was expected that our patient would have pronounced atrophic patterns, which would contribute to the manifestation of psychosis during the course of the disease. Some studies have found lateralized reduction of grey matter volume, especially temporal gyri in AD patients with delusions of misidentifica-

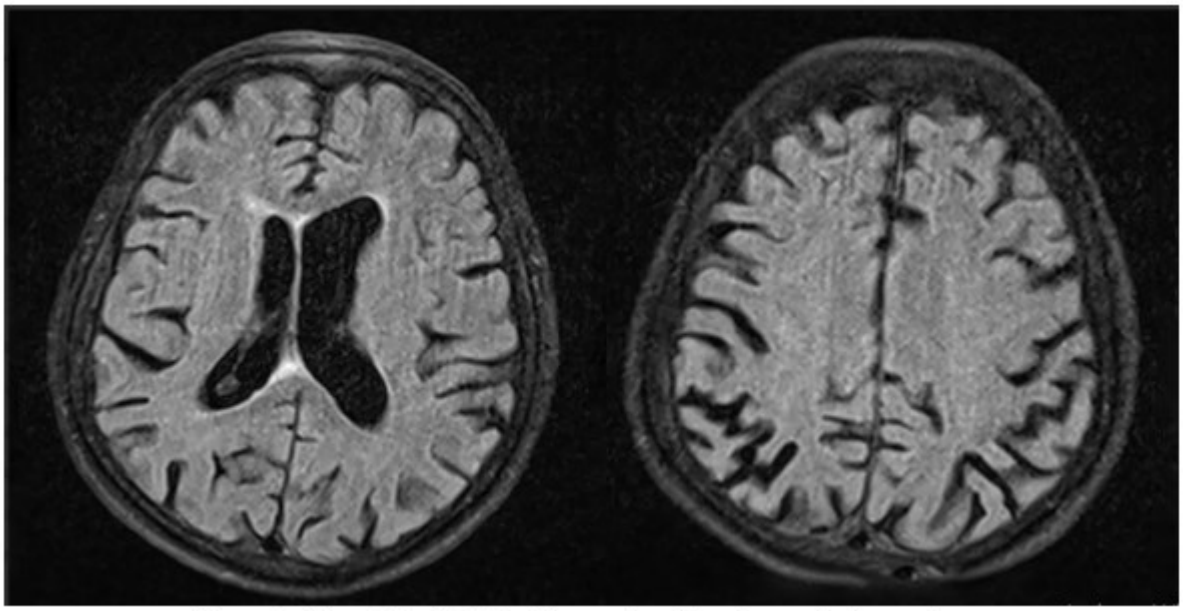

Figure 1. T1 weighted MRI of the patient showing cortical atrophy

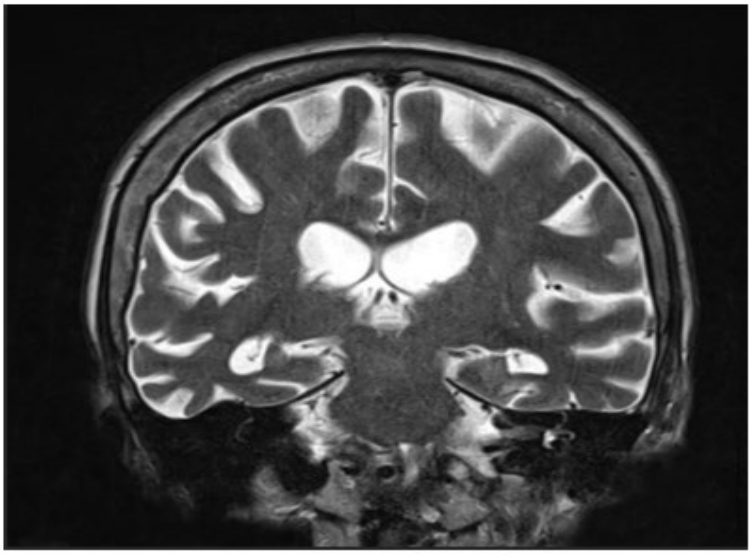

Figure 2. Coronal T2 weighted image showing medial temporal atrophy

tion (28, 29, 30) pointing of possible disruptions to object identification neural network. In our case no lateralization was shown on neuroimaging, entorhinal cortex (BA 35, 36) was compromised bilaterally.

We have described the case in which structural neuroimaging changes preceded clinical manifestations of AD. Although it remains speculative to suggest that neuroimaging findings may serve as a marker for early diagnosis, our case supports this hypothesis.

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