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EDITORIAL

Predictors of prognosis in Alzheimer's disease: The role of cognitive dysfunction, immune abnormalities, and advanced neuroimaging

Hafsa Arshad Raja, Abdulqadir J Nashwan

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Abstract

Alzheimer's disease (AD) is a grave illness that results in cognitive and social issues. A recent study examined the association between neuroimaging results, cognitive dysfunction, atypical cellular immune function, and poor prognostic factors in AD patients who demonstrated poor prognosis. Poor prognosis was associated with abnormal cellular immune function, extrapyramidal symptoms, altered consciousness, abnormal electroencephalogram, modified Rankin scale, increased neutrophil lymphocyte ratio, and severe pneumonia. The impaired cellular immune function characterized by a reduction in the blood T lymphocytes' proportion predicted poor prognosis as an independent risk factor in AD. Early initiation and maintenance of AD medications is associated with better outcomes.

Key Words: Amyloid beta peptide; Alzheimer's disease; Dementia; Cellular immunity; Prognosis; T lymphocytes; Magnetic resonance spectroscopy

Core Tip: Alzheimer's disease (AD) is a serious condition leading to cognitive and social challenges. A recent study found that poor prognosis in AD patients is linked to abnormal cellular immune function, extrapyramidal symptoms, altered consciousness, abnormal electroencephalogram, increased neutrophil-lymphocyte ratio, and severe pneumonia. Impaired cellular immune function, particularly reduced T lymphocytes in the blood, is an independent predictor of poor prognosis. Early and continuous use of AD medications is associated with better outcomes.

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INTRODUCTION

A neurodegenerative disorder called Alzheimer's disease (AD) is typified by severe cognitive impairment. The main clinical signs are memory loss, logic and thought difficulty, and abnormal mental behavior. About 40%–60% of individuals with dementia exhibit it[1]. Tau protein hyperphosphorylation can readily lead to aggregation, which can cause cell death and neuron and nerve synaptic dysfunction, particularly in small glial cell loss. In damaged brain areas, reactive glial cell proliferation frequently results in subsequent cytopathological reactions[2]. The progression of AD is facilitated by the interaction between tau protein and Amyloid β. Typically, aberrant immune function mediates AD's start and progression[3].

Presently, the cognitive function scale and the exclusion of illnesses that are similar serve as the basis for most of the diagnostic criteria used to identify AD. Serum or cerebrospinal fluid (CSF) can include some biochemical indicators associated with dementia, but their specificity and sensitivity are low[4]. Future studies should focus on combining certain biochemical markers in CSF or blood for the early identification of AD; among them, some biochemical markers linked to immunity show excellent research potential. Some researchers have integrated blood biochemical markers with magnetic resonance spectroscopy (MRS) detection results and had successful outcomes[5].

An increasing amount of evidence points to the involvement of persistent immunological inflammation in AD pathophysiology. One significant systemic inflammatory indicator is the blood's neutrophil to lymphocyte-ratio (NLR). The calculation of NLR involves dividing the total number of neutrophils by the total number of lymphocytes. It is increased in certain medical conditions like diabetes, hypertension, and tumors. Flow cytometry can identify T, B, and natural killer cells in the blood, which can be used to determine whether AD patients' immune systems are functioning abnormally[6]. For the early diagnosis and prognosis evaluation of AD patients, cranial MRS has significant therapeutic importance when combined with the identification of pertinent biochemical markers and electroencephalogram (EEG) wave indexes[7].

PREDICTORS OF PROGNOSIS IN ALZHEIMER'S DISEASE

In a retrospective study by Bai *et al*[8], correlative factors of poor prognosis and unusual cellular immune function in AD patients were evaluated. The study analyzed 229 patients with dementia (single or combined), including eighty-seven with vascular dementia, sixty-eight with AD, and fifty-three with other types of dementia. According to the study, the poor prognosis of AD patients was associated with extrapyramidal symptoms, abnormal EEG, MRS, elevated NLR, and complex pneumonia in addition to impaired cellular immune function.

Additionally, it demonstrates that a decline in the blood's T cell proportion following unusual cellular immune function was a risk for indicating a poor prognosis for AD patients. The study concludes that a decline in the percentage of T cells could be a sign of a bad prognosis for AD patients. It is recommended that the cut-off criteria for determining the poor prognosis of AD be set at a proportion of T cells less than 55%. Furthermore, it is noteworthy that MRS, in conjunction with EEG detection, can accurately predict the unfavorable prognosis of AD.

One significant aspect to speculate on is the study's retrospective design. In retrospective investigations, scientists look for patterns and correlations in the data that already exist. But this design inherently limits the capacity to establish causation. More reliable data would come from a randomized controlled trial (RCT), in which patients are randomized into several treatment groups. Because they reduce bias and make it easier to understand how beneficial an intervention is, RCTs are regarded as the gold standard in clinical research.

The total of 229 patients in the study are a suitable sample size for preliminary investigations, although it may not fully reflect the complexity and diversity of the larger community when dealing with the prognosis of AD. These patients' individual circumstances, comorbidities, and overall health may all have an impact on their prognosis. A larger and more varied sample would increase the dependability of the results and give a more accurate depiction of the population.

The study's dependence on subjective measurements is another factor to consider, such as cognitive dysfunction performance characteristics, Mini-Mental State Examination score, modified Rankin scale, drawing clock test, disturbance of consciousness, and extrapyramidal symptoms. These assessments are useful for gathering patient self-reported experiences; however, they are biased and subject to individual standpoints. The immunoglobulin test and the lymphocyte transformation test were not performed, and the components pertaining to the identification of cellular immune function were lacking. In addition to these subjective evaluations, objective measurements could offer a more thorough comprehension of the AD prognostic metric.

Furthermore, a critical consideration is how generalizable the study's discoveries are likely to be. The survey results may not be applicable to other settings or populations because it was performed within a particular patient group and healthcare setting. The results of such interventions can be impacted by parameters such as patient demographics, cultural differences, and healthcare infrastructure. Therefore, without more research, care should be taken when

expanding these outcomes to other groups.

CONCLUSION

The work provides useful insights for anticipating the relationship between impaired cellular immune activity and a bad prognosis in AD patients, highlighting the potential benefits of MRS combined with EEG detection in anticipating the dismal outlook of AD. The data indicate that a drop in T cell percentage may have prognostic value for the adverse outcome of AD. The threshold criteria for determining poor prognosis in patients with AD is recommended to be set at a proportion of T cells less than 55%.

FOOTNOTES

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