Infiltrative Brain Mass Due To Progressive Alzheimer’s Disease

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Abstract

Background: Magnetic resonance (MR) imaging in patients with Alzheimer’s disease can present challenging diagnostic dilemmas.

Methods: A seventy-five year old female presented with a slow history of memory loss over five years. An MR demonstrated development of a large area of T2 signal abnormality involving the medial left temporal lobe with extension into the insular cortex and hippocampal formation. The patient underwent a stereotactic brain biopsy.

Results: Pathology revealed no evidence of tumor, but there were extensive neurodegenerative changes with gliosis and was tau and beta amyloid positive.

Conclusions: An unusual radiographic presentation of progressive Alzheimer’s disease is discussed including possible immuno-inflammatory mechanisms.

Introduction

Magnetic resonance (MR) imaging in patients with Alzheimer’s disease can present challenging diagnostic dilemmas. The importance of pathological confirmation of the intracranial process is essential to determining appropriate treatment and prognostic implications for the patient. This case illustrates the need for tissue diagnosis and postulates possible mechanism for the clinicopathological findings.

Case Report(s)

A seventy-five year old female presented with a slow history of memory loss over five years. Initial evaluation at onset of symptoms revealed a non focal neurologic exam and normal magnetic resonance (MR) imaging of the brain. She was started on Aricept ®. Her past medical history was significant for rectal carcinoma in remission. Follow-up neuropsychometric testing demonstrated significant decline in memory processes and personality changes. Electroencephalography revealed persistent slowing in the left mid to anterior temporal region of both theta and delta frequencies without epileptiform activity. An MR demonstrated development of a large area of T2 signal abnormality involving the medial left temporal lobe with extension into the insular cortex and hippocampal formation (Figure 1). There was mild mass effect and no abnormal enhancement.

Results

The patient underwent a stereotactic brain biopsy. Five sequential biopsies were performed through the course of the mass. Pathology revealed no evidence of tumor, but there were extensive neurodegenerative changes consistent with gliosis. The significant abnormal findings on the MR could be consistent with an inflammatory response in the absence of any evidence of tumor. The tissue was beta amyloid and tau positive (Figures 2 and 3). The patient was maintained on Aricept and vitamin E. Follow-up MR five months after the biopsy and subsequently one year later demonstrated no change in the lesion. The patient declined further biopsy and remained neurologically stable over the course of the next year. Antibody measurement to aggregated Abeta amyloid was performed and compared to ten control patients of similar age and were found to be substantially higher than the controls. She was started on prednisone for to three weeks without improvement. Follow-up MR two years after the biopsy demonstrated slight enlargement of the mass with continued gradual neurocognitive decline. Further biopsy was declined. The patient expired four years after the pathologic diagnosis from progressive Alzheimer’s disease.

Discussion

An unusual radiographic presentation of progressive Alzheimer’s disease is discussed. While the possibility that this lesion represented sampling error of a low grade glioma, the pathologic findings and stain results would make that less likely. These findings prompted further investigation into possible etiology of the mass and possible mechanism of development. There has been report of a similar mass having developed in a patient who received the Alzheimer’s disease vaccine [1]. In that case, the mass was studied at autopsy and attributed to macrophage infiltration and edema.
secondary to the immunotherapy treatment the patient received. In addition, a phase II drug trial involving an experimental vaccine against Alzheimer’s disease was suspended when 15 patients developed clinical features consistent with aseptic meningitis [2]. This may have represented a cell-mediated immune response to the vaccine. Abeta antibodies can reduce Abeta levels by microglial activation [3]. It is possible this represents an autoimmune inflammatory response and T-lymphocyte activation. Amyloid deposition requires phagocytosis by reactive microglia and can activate an inflammatory response.

It remains unclear if the antibodies have an effect on the progression of Alzheimer’s disease. Plaques are believed to be formed by the accumulation of amyloid peptide A and phagocytized via the Fc-mediated process by microglial cells. The formation of antigen-antibody complexes peripherally can aid in preventing amyloid from accumulating in plaques. It has been demonstrated that B-amyloid infusions in rats can generate activation of brain astrocytes and microglia as well as increased IgG immunostaining in the cerebrovascular structures and brain parenchyma. This can lead to a further inflammatory response.

Conclusion

This patient demonstrated a significantly higher level of antibodies to beta amyloid than controls. Sampling error always remains a possibility in such cases where no evidence of neoplasm is identified. However, this patient underwent several biopsies throughout the abnormal area identified on the MR scan. While tau and beta amyloid depositions are not uncommon findings in brains of elderly patients, combination of findings in this patient raise suspicion of another process. It is a possible mechanism that an exaggerated immunological response to beta amyloid may have played a role in the development of the tumor-like mass identified on MR imaging.

References

Illustrations

Illustration 1

T2 axial MR demonstrating extensive signal involving the temporal lobe, insula and hippocampus

Illustration 2

High-power view of beta amyloid stain and presence of plaques with absence of amyloid angiopathy
Illustration 3

Tau protein positive stain in neurofibrillary tangles (arrow)
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