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Letter to the Editor

Alzheimer's disease is treatable by increased cerebral blood flow (CBF) from omentum to compensate for a decreased CBF in aging

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Dear Sir,

Alzheimer's disease (AD) is one of the major problems that have confronted the medical community over the years. Millions of patients have suffered from this condition, and yet, despite decades of extensive research efforts, the origin and treatment of AD continue to remain uncertain. It has been suggested that amyloid within the brain might be the basis for the development of the condition, but more recently, a suggestion has been made that natural aging conditions cause AD. This statement is based on personal findings which have been reported.[3-15]

In the presence of AD, there is a loss of energy in the intracerebral neurons, mitochondria, and adenosine triphosphate (ATP) due to a decreasing cerebral blood flow (CBF).[15] When the energy in these critical neurons decreases, a diminished cognitive state can result. There is no evidence that a decrease in amyloid in an AD brain will increase the energy needed by the intracerebral neurons, mitochondria, and ATP,[2] which could lead to improved cognition. What is needed and has not been achieved is a supplemental increase in CBF to compensate for the loss of CBF that routinely occurs in an AD brain as a normal physiological occurrence with aging. The application of the omentum to an AD brain, however, has been shown to improve cognition in patients with AD because of its ability to supply an additional source of CBF to the AD brain.[11] The intracellular energy activity produced by ATP from mitochondria becomes the final energy source of all cells in the body, including the intracerebral neurons.[3] An increase in CBF to compensate for the decreased CBF, which occurs as a function of natural aging, becomes critical for the survival of the intracranial neurons, mitochondria, and ATP.

For decades, a way has been sought to increase cognition in AD patients. This has been attempted by the insertion of various therapeutic agents into the body in the hope that a positive cognitive result could occur. Unfortunately, this goal was never achieved. It was earlier believed that the decreased CBF in an AD brain resulted from a slow deterioration in the energy of the intracerebral neurons, mitochondria, and ATP that no longer required their previous volume of CBF to the brain. But more recently, there has been increasing interest and information showing that it is not the deterioration of the energy of intracerebral neurons resulting in a decreased CBF, but it is actually the decrease in CBF itself that results in the diminished intraneuronal activity that leads to the loss of cognition found in AD.[10]

There has been increasing interest in the omentum's ability to increase CBF to compensate for the decreased CBF that occurs in AD.[12] However, this idea has not been generally accepted. An example is the government's recent approval of anti-amyloid monoclonal antibody studies in the hope that these experiments will decrease amyloid in the brain, resulting in improved cognition. But how can this occur? Where are the physiological actions in the brain that can explain how a low amount of amyloid level in the brain can lead to an increase in CBF and a significant cognitive improvement?

There is now an awareness that the vascularized omentum, when placed on the brain, can markedly increase CBF to cerebrovascular,[8,17] Alzheimer's,[15] and other neurological conditions.[18] An article recently published in *The Journal of Alzheimer's Disease* showed the surgical technique necessary for mobilizing the omentum to the brain, including illustrations of the procedure.[16]

Based on the theory that placing the highly vascular omentum onto an AD brain might improve cognitive results, the omentum was surgically applied to the brain of 25 AD patients. Nine patients had an excellent cognitive result following the operation,[19] but ten patients had only minimal improvement and six patients had no improvement. Because of the known ability of the omentum to supply large amounts of CBF to the brain,[6] all twenty-five AD patients were considered to have had a major increase in CBF following the placement of the omentum onto their brain. It was, therefore, expected that the omental application to the brain would improve cognition in the majority of the patients; however, this did not occur. Why did nine AD patients following surgery have significant cognitive improvement, but sixteen patients in the study had only minimal or no improvement following the operation? An answer to this question was that patients who exhibited no improvement following omental surgery had been expected to have shown an increase in cognition as the result of an expected increase in CBF by the omentum on their brain. No amount of increased CBF could be expected to improve cognitive activity in the presence of non-functioning intracerebral neurons. This is likely the reason why two-thirds of the patients in this study showed little or no cognitive improvement following omental transposition to the brain. The reason nine AD patients did show cognitive improvement following omental transposition to the brain was that apparently the increased CBF from the omentum had a positive effect on viable intraneuronal agents.

There has been a great effort over the years to introduce therapeutic agents into the body that were considered to have the potential to improve cognition in AD patients, but these efforts were unsuccessful.[16] Only two of 2700 research attempts from 2004 to the present were successful. The cognitive improvement failed in these numerous experiments apparently because of the inability to obtain a supplemental source of CBF to the AD brain at the time the AD brain was undergoing a decreased CBF. The 2700 experiments had no way to obtain a supplemental CBF to compensate for the decreased CBF, which was occurring as a normal aging phenomenon in AD patients. The omentum, with its extensive vascular potential, can deliver the supplemental blood supply that is needed in the AD brain, which can lead to improved cognition.

Functional intracerebral neurons are apparently the key to cognition. When there is a decrease in CBF as a function of aging, the energy source of intraneuronal elements decreases, and an additional new source of CBF must be attained. The reason for the many failures in past experiments was the apparent inability to introduce an increased CBF to compensate for the decrease in CBF that occurs in the AD brain.[10]

A new vascular theory has slowly begun to arise, as seen in a recent article in the *Journal of* AD titled "Alzheimer's disease: a decreased CBF to critical neuronal elements is the cause."[15] This may become a new concept in the cause and treatment of AD. The reason that previous failures to implicate various agents as the basis and treatment of AD was the apparent inability to increase CBF; at the same time there was a decreased CBF to the critical intraneuronal neurons, mitochondria, and ATP.

There is strong enthusiasm by some that monoclonal antibodies will improve cognition in AD patients in the future,[1] but there is little evidence at present that monoclonal antibodies will have the ability to lower levels of amyloid in the AD brain and at the same time obtain a supplemental source of CBF required by mitochondria and ATP in AD.

Based on the clinical findings of the AD patients who showed no improvement following omental surgery, the pre-operative condition of each patient was critically evaluated, and significant information was obtained, which could explain the negative results of their operation. The patients who showed no improvement were over the age of 80, had AD for a number of years, and had a low mini-mental score (MMSE), which measures a person's cognitive state. A maximum MMSE is 30, and a score of 25 or more is considered normal. Any score below 25 is considered abnormal, and if this number is markedly lower, the number would indicate an expected, unfavorable result on the outcome of an omental operation on the brain. Anyone considering applying the omentum to an AD brain should not operate on a patient with an MMSE score below 14. This is critical.

It now appears to be the time to critically study cognitive improvement following omental placement on the AD brain. This can be done in a small group of ten AD patients. All patients must be in excellent physical condition and have a MMSE score of at least 14 and above. They should be between the ages of 65 and 75 and have had AD for no longer than 1 year. It will be highly desirable if the patients will incur no financial expense for the operation. With the National Institutes of Health (NIH) spending 3.8 billion dollars each

year on Alzheimer's projects, such a small operation should present no financial problem for the NIH.

It would be hoped, but probably unlikely, that this small project could be performed in the prestigious NIH clinical research hospital located in Bethesda, Maryland. The reason for suggesting this NIH hospital is that this outstanding clinical research institution can authorize the procedure without the need for a time-consuming grant application and the need for an institutional review board for permission to perform the procedure. Assuming that such a small project can be successful, it would be of enormous importance if the NIH were involved. Most important, it would be of benefit to millions of patients throughout the world who have, or will have, AD in the future. We will await the decision of the NIH since if the operation being proposed is approved and successful, it will be of significant medical importance.

Ethical approval

Institutional Review Board approval is not required.

Declaration of patient consent

Patient's consent is not required as there are no patients in this study.

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Conflicts of interest

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Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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