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Review Article

Alzheimer Disease - The Cause and Treatment

Harry Sawyer Goldsmith*

Department of Neurosurgery (Retired), University of California, Davis, CA, USA

***Corresponding Author:** Harry Sawyer Goldsmith, Department of Neurosurgery (Retired), University of California, Davis, CA, USA.

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Abstract

This paper portrays a patient with long-standing and well-diagnosed Alzheimer's disease, who showed notable improvement following omental transposition to his brain. The reason for the improvement may well have resulted from the increased cerebral blood flow due to the omentum on the brain which supported mitochondria and ATP. When there is an increase in blood flow to these critical neurons, Alzheimer's disease symptoms have shown improvement.

Keywords: *Alzheimer's Disease (AD); Cerebral Blood Flow (CBF); Vascular Endothelial Growth Factor (VEGF); Adenosine Triphosphate (ATP); Mitochondria*

Introduction

In 1973, a lifelong search was begun to investigate the possibility that a section of intact omentum could be mobilized subcutaneously up the chest to the brain for neurological problems [1-26]. Over the following years, colleagues tried to convince me that bringing the omentum to the brain was surgically impossible and could never have the ability to improve cognition.

Over time, confidence began to arise that omentum transposition to the brain of patients for a variety of neurological conditions could be effective. It slowly began to be accepted that Alzheimer's disease (AD) possibly resulted from a decrease in energy production by critical intracerebral neurons that are essential for cognition [9]. A surgical operation involving the omentum was found to be able to increase cerebral blood flow (CBF) in sufficient amounts to improve cognition [14]. The operation involved the omentum since it is the most angiogenic structure in the body with the ability to bring new blood vessels and blood to the brain due to vascular endothelial growth factor (VEGF) which is the most vascular material in the body, and its largest volume is in the omentum [27].

It is well known that as one ages, blood flow to the brain decreases, a phenomenon that can slowly result in cognitive decline [8]. There are two critical cerebral neurons that are strongly influenced by the CBF decline in AD patients, namely, mitochondria, the producer of adenosine triphosphate (ATP), the latter being the source of energy necessary to all cells in the brain. Blood flow to mitochondria and ATP must be maintained if AD is to be prevented and treated.

During the period of my slow uncertainty as to whether omental transposition to the brain could be helpful in AD. I came in contact with an AD patient following a telephone call from a person, who I later learned was a very successful businessman in Boston. He called

about his father, whom I later learned was a famous academician at the University of Tehran, in Iran, being professor of both mathematics and physics. His son said his father had severe Alzheimer's disease and he had taken him to the prestigious London Hospital, the Mayo Clinic and the Massachusetts General Hospital in Boston, hopefully for treatment. All three institutions stated his father had Alzheimer disease, and there was no treatment for his condition.

The son of the patient had learned of the work that had been ongoing with omentum placement on the brain for AD and he asked if the operation might be helpful for his father. The son said he and his family were desperate because his father had declined to such a state that he was wiping feces on the wall of his home.

I told his son, that I was aware of a noted neurosurgeon in the Middle East, Dr. Fuad S. Haddad, who was the Chief of Neurosurgery at the American University Hospital in Beirut, Lebanon. I said I would try to contact Dr. Haddad to see if he might be interested in the case. Dr. Haddad was called and he said he would be happy to see the patient but it would be necessary for the patient to travel to Beirut. Dr. Haddad said it might be possible that he and I could operate on the patient at the American University Hospital in Beirut.

The patient was brought to Beirut and was scheduled for surgery. The night before the operation, political powers at the hospital attempted to cancel the case. I had a comparable experience on another occasion in the United States when the night before an omentum operation was to be performed, an unsuccessful attempt was made to block the operation. Because Dr. Haddad was so respected and powerful, the omentum operation in Beirut was allowed to be performed.

The operation went well and on the morning of the third day after the operation, a large number of people entered the patient's room. These included Dr. Haddad, myself, the patient's wife and son, and a large group of nurses and residents. Right at the beginning, the son knelt at his father's bedside. He kissed his father on his father's left cheek and in that position, his father whispered quietly into the son's right ear. The son looked up at the people in the room and said, "This was the first time my father has spoken to me in two years". The patient's wife began to cry, and it was at this moment, all in the room knew they had just experienced a deeply moving emotional event. Because of this heartfelt experience, I became strongly influenced that AD could be treated by adding CBF by the omentum operation for AD.

The patient did well for a day or two and then developed pseudomonas pneumonia. As with all my patients whom I operated on overseas, I never left them until they were discharged from the hospital. This required that I stay in Beirut for three to four weeks until the patient was in a position to travel back to Tehran.

Upon my return to the United States, I kept in close contact with the patient's wife and son over the next several weeks. I was informed of the tremendous improvement present in the patient. I was also told that he appeared almost cognitively normal and that his colleagues from the university were now visiting him and were having scientific discussions. One day I called his wife and she told me how well he was progressing. She mentioned that his doctor was presently at their home, and would I like to speak with him. This was the perfect time to learn from the patient's doctor of his true condition. The doctor told me how well the patient was doing and that he and others were amazed at his recovery. I asked the doctor if there was any clinical problem that remained an issue. The doctor said that their only concern was the loss of weight resulting from the patient's recent operation. I asked the doctor how were they planning to increase his weight and he informed me that they were giving him large amounts of Ensure. I asked him where he could get Ensure?? He replied, "in the grocery store". On the night following the telephone call with the doctor, the patient suddenly died from a heart attack.

The cause of Alzheimer's disease

Following the sad death of the patient, it seemed imperative to carefully consider the basis of AD and if omental transposition to the brain of Alzheimer patients could lead to cognitive improvement.

For many years, it was felt that the decline in CBF that occurs in Alzheimer disease was due to the aging of cerebral neurons that no longer required a continuing need for CBF. Years earlier this situation was simply called senile dementia. A new concept is now developing that it is not the aging of cerebral neurons in AD that leads to decreased CBF, but it is actually the decrease in CBF itself that leads to the failing of critical cerebral neurons [19]. This decrease in CBF occurs as a result of a normal aging phenomenon. If there is to be a method to successfully treat patients with AD, adequate CBF must be maintained to the brain [18].

The treatment of Alzheimer's disease

The critical feature in AD is the slow but continuing decrease in the energy of an AD patient's cerebral cells resulting from a decrease in CBF. Energy in the brain is created by mitochondria and ATP. These neurons always require an adequate CBF supply if AD is to be treatable. If CBF to these cerebral neurons is not maintained, AD symptoms develop which include forgetfulness, memory loss, and the person's inability to do everyday tasks [25].

Alzheimer disease treatment might be standardized if there was a method to show that an increase in CBF to the AD brain could be cognitively successful. This method would have to show that this increase in CBF could be confirmed for an indefinite period of time [7]. Pharmaceutical applications and research efforts have not been able to accomplish this [29], but studies have now been shown that a significant amount of CBF can be introduced into a brain by way of a surgical operation that places a piece of viable omentum directly on the brain which allows large amounts of CBF to enter the brain [17]. This operation, omental transposition to the brain, presently appears to be the only way to introduce large amounts of CBF to the brain of patients with AD [15]. The surgical technique for performing the operation has been well-documented [18,26] and has been reported to be successful [28].

In order to find a successful method to treat AD, research must continue to be performed. Recent enthusiasm by some who believe that a lower amyloid concentration in the AD brain by way of anti-amyloid monoclonal antibodies will be important in the future. An example of this idea is seen in a recent article "Anti-Amyloid Monoclonal Antibodies are Transformative Treatments that Redefine Alzheimer's Disease Therapeutics" [30].

One may ask how lowering the level of amyloid in the AD brain can increase CBF to mitochondria and ATP, the energy source in the brain, since amyloid has no connection to CBF. Even if there was a relationship between amyloid and CBF, where could one find a new and large source of blood needed by the mitochondria and ATP in the AD brain in spite of a low amyloid level?

Studies on the role that anti-amyloid monoclonal antibodies might have on AD could take years before the results of the study are known. As opposed to this, clinical results following omental transposition to the AD brain could be rapidly obtained. This could be accomplished by a small study on the omentum to the AD brain which could be small, economical, and basically simple. The study would require only a small number of patients, ten being sufficient. The patients in the study should be no older than 65 - 75 in age, have had AD for no more than one year, and most importantly have a mini-mental state examination (MMSE) score of 14 or above. The study would not require a great deal of financial support as usually occurs in present-day experiments that frequently cost millions of dollars. The results of the study could be known within weeks of the operation.

Conclusion

If the operation of omental transposition to the brain of AD patients can demonstrate a significant post-operative cognitive improvement within a short period after surgery, the importance of omental transposition in AD may be recognized, and may become the basis for the treatment of AD. It is hoped that clinicians, institutions, and foundations will respond to this request for a small study of omental transposition to the AD brain because of its future importance to the many people throughout the world who presently have or will suffer from Alzheimer disease.

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