Alzheimer's Disease: A Case Study

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A 59 year old university teacher with symptoms of Alzheimer's disease visited our clinic and was treated for five weeks, from November 9 to December 15, 1995. When she was admitted to our clinic she was complaining of progressive memory loss. She had been forgetting professional terms and concepts, which forced her to resign from her position at the university. In day-to-day matters she was forgetting to switch off electronic devices and kitchen appliances, and she continuously became lost, even in her own neighborhood.

She had been ill for 13 months and had suffered an acute onset of the disease. Her history showed that at the time the amnesia first appeared she had been restless and disoriented. The first bouts of amnesia lasted three to four hours, before she recovered. Since that time, an increase in dementia and memory loss was registered.

The patient suffered also from chronic gastroduodenitis and pancreatitis. Her blood pressure rose to 150/90 mm.

One fact of importance was that her brother had suffered from similar memory loss and died at age 57 with dementia symptoms. Her mother suffered from dementia, beginning at age 52 and died from ischemic cerebral stroke at age 56. We concluded that this predisposition was based on genetics, not gender.

Objective

Routine examination of internal organs revealed no pathology. In routine neurological tests no local pathology was observed and there were no axial signs.

Mental state

The patient was disoriented and without any signs of delirium. Critical behavior was preserved. We noted acute memory loss concerning life events such as childhood, and she could not recall her maiden name. The patient had difficulty with recall and with the execution of the most simple arithmetic operations. Her ability to conduct abstract-logical thinking was diminished.

Diagnostic

CT scan showed atrophy of the cortex, widening of subarachnoidal spaces and brain ventricles. Roentgenoscopy revealed some reduction of blood flow in all areas. Doppler ultrasonography did not reveal significant reduction of blood flow in the vessels of the head or neck. EEG examination showed a slowing of the main cortex rhythm. Blood tests showed slightly elevated cholesterol levels.

Therapy

The patient was treated with **Cerebrum compositum**, one ampule injected three times a week. **Cerebrum compositum** is a complex homeopathic formula produced in ampules of 2.2 ml. Each ampule contains in its formula, as active ingredients:

Aesculus hippocastanum 4X

Cinchona officinalis 4X

Cocculus indicus 4X

Conium maculatum 4X

Gelsemium sempervirens 4X

Ruta graveolens 4X

Aconitum napellus 6X

Anacardium orientale 6X

Hyoscyamus niger 6X

Kali phosphoricum 6X

Thuja occidentalis 6X

Cerebrum suis 8X

Ignatia amara 8X

Kali bichromicum 8X

Manganum phosphoricum 8X

Ambra grisea 10X

Bothrops lanceolatus 10X

Embryo totalis suis 10X

Hepar suis 10X

Magnesia phosphorica 10X

Phosphoricum acidum 10X

Placenta suis 10X

Selenium metallicum 10X

Sulphur 10X

Medorrhinum 13X

Syphillinum 13X

Arnica montana, radix 28X: 22 mcl each

Results

1st week: no change

2nd week: no change

3rd week: The patient mentioned some improvement and she became more physically active, but felt no changes in her state of memory.

4th week: The patient's ability to perform routine tasks improved. She was able to retell articles and her

In 1907, Alois Alzheimer described the non-atherosclerotic form of senile dementia that Kraeplin later named after him. The disease is essentially a cortical dementia which follows a characteristic, progressive course. Disturbances in both short- and long-term memory are present and are typically accompanied by disturbances in speech and spatial orientation, a decrease in creative ideas, and difficulty in drawing. The patient is often irritable and poorly motivated.

Memory continues to deteriorate in all areas as the disease progresses. The terminal stage is reached after a period of 8-15 years. Death is usually caused by concomitant illnesses.

Alzheimer's disease is the most common cause of intellectual decline in old age. Because differential diagnosis is difficult and can only be definitively confirmed by histological examination after death, the estimated number of unconfirmed cases is high. It is still not fully clear whether the prevalence of the disease is increasing or whether diagnosis is simply becoming more exact. Of course the increase in average life expectancy has also led to an increase in this age-related illness.

The senile plaques found primarily in the cerebral cortex of Alzheimer's patients consist of a small protein molecule (beta-amyloid) and the degenerative neurites. It is not known whether these deposits cause the illness or whether they are simply the visible consequences of the disease process.

Similar amyloid deposits are also found to a much more limited extent in healthy older people. Genetic factors seem to play a role in this. The APO-4 gene on chromosome 21, which produces amyloid precursor protein (APP), is strongly associated. A defect in this gene seems to prevent APP from being chemically split in the right way. This results in insoluble beta-amyloid, which forms the typical masses in the brain. We see similar consequences in Down's syndrome, which is also due to a chromosome-21 defect.

At present there is no curative treatment for Alzheimer's. Circulatory disorders play a minor role and seem to have a negative effect only on metabolic functioning. Deficiencies of neurotransmitters, especially acetylcholinetransferase, have been documented in Alzheimer's patients, but their significance has not yet been adequately explained.

Immune-system disorders such as those that can occur as a result of psychological stress seem to influence the course of the disease, and slow viral infections probably also contribute. The extent of the influence of environmental stress factors (free radicals) still needs clarification, but administering antioxidants certainly cannot do any harm.

Adjuvant therapy of Alzheimer's syndrome can also be conducted on a naturopathic basis. The patients are treated in exactly the same way as other patients with age-related dementias. Regular, intensive memory training is as important as the consistent attention of trusted caregivers.

Antihomotoxic medicine offers an interesting therapeutic option. Its detoxification procedures and metabolism-activating catalyst preparations may at least reduce the typical burdens of old age on the body. Homeopathic combination remedies such as Cerebrum compositum, Coenzyme compositum, and Ubichinon compositum can be used, as can various oxygen therapies and phytotherapy with Ginkgo biloba to promote circulation.

Klaus Küstermann

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ability to perform arithmetic tasks improved.

5th week: The patient noted improvement in her abilities and her mood, including abstract-logical thinking.

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Note from the Medical Editor:

Fifty-five percent of caregivers report that they have tried at least one alternative therapy to improve the patient's memory. Another twenty percent have tried three or more unproven therapies. Most frequently used were vitamins (84%), health foods (27%) 'smart pills' (9%), and home remedies (7%) though with these therapies, caregivers did not notice significant improvement. [1]

Cooperation is needed between complementary practioners and conventional practitioners to evaluate unproven therapies for Alzheimer's patients.

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