ADDITIONS TO THE DRUG PICTURE OF ALUMINA*

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ABSTRACT: Animals treated with parenterally given aluminium chloride produced disturbances of behaviour similar to human dementia especially in Alzheimer's disease.

In the animal's brains increased concentrations of aluminium could be found as well as a proliferation of neurofibrills.

Because increased aluminium concentrations and neurofibrillar degeneration can be found in several parts of brains of patients suffering from Alzheimer's disease, the aluminium uptake and disturbances in behaviour seen in animals can be used as a model for Alzheimer's disease and dementia.

As Alumina is not a well-proven remedy of our homoeopathic materia medica, the drug picture of Alumina can be completed and corrected by the symptoms and findings seen in Alzheimer's disease as it is explained in extent.

Above all these observations lead to meaningful therapeutic consequences. It is expected that dynamized aluminium will be able to initiate a mobilisation and elimination of incorporated aluminium in Alzheimer's disease and certain forms of dementia and that by that action the degenerative process may be stopped or even reversed.

On these facts possibility of use of potentised alumina for the treatment of dementia particularly Alzheimer's disease is discussed in the present paper.

Aluminium is an abundant constituent of the earth's crust and is used for the construction of aircrafts and other large metal structures, where lightweight and corrosion resistance are required. Especially in the highly industrialized countries, industrial and household equipments, electrical equipments, explosives, paints and deodorizers are made from aluminium. A further source of aluminium is in the form of oral antacid, antidiarrhoeal drugs and protective dermatologic pastes.

Because of the high amount of aluminium in the earth's crust, and also in the lithosphere, hydrosphere and atmosphere, it was not until the early sixties of this century the allopathic medicine began to recognize the harmful effects of chronic exposure of the body to high levels of this metal.

Aluminium was taken up early in the homoeopathic materia medica[1] but belongs, according to Kent,[2] to the comparatively badly proved medicines.

In the following lines the drug picture of aluminium will be enlarged from the results of current researches and new ways of application in the field of neurology and psychiatry will be shown.

OBSERVATIONS IN ANIMALS

Animals treated with injections of aluminium chloride, either intrathecal

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or subcutaneous, soon start to show behavioural abnormalities compared with untreated animals.[3] These behavioural abnormalities were characterized by a gradual impairment of higher and highest brain functions similar to the demential process in human Alzheimer's disease.

It is better to mention here that Alzheimer's disease is a form of presenile dementia usually in persons below fifty years of age associated with Alzheimer's sclerosis which is a type of hyaline degeneration of the medium and smaller blood vessels of brain.

There was a latency period of five to ten days after intracranial aluminium application, during which the animals did not behave strangely; later on an alteration in short-time retention for tricks recently learned appeared. After that the animals were not able to retain a trick longer than five seconds. As the earliest sign of a motoric disturbance, impairment of coordination of front and rear legs could be found. Later on complex sequences of motions could not be exercised. Soon it came to a decrease and total disappearance of motivation and searching behaviour. After fifteen to twenty-one days the animals showed an increase in muscle tonus of the lead-pipe type. Intended motions could not at all be exercised at that point of time.

That phase was followed by generalized cerebral seizures, during which most of the animals died in an epileptic state, inspite of a specific treatment with anticonvulsant drugs and substitution of fluid and electrolytes.

The surviving animals suffered from neurologic deficits in form of a cortical blindness, outbursts of rage after the slightest sensoric stimulation, ataxia and loss of memory. Chemical analysis of the animal's brains by the atomic absorption method, which detects as little as 0.1 μg/g of aluminium showed a mean cortical aluminium level in the aluminium treated animals, who showed behavioural abnormalities ranging from 7.5 μg/g to more than 12 μg/g compared to the 1.5 μg/g in normal animals.

Even more striking were the results of the morphological analysis of brains with high cortical aluminium levels. It could clearly be demonstrated that aluminium (like some other agents) was able to induce a proliferation of the 10nm neurofilaments.[4,5] Aluminium induced proliferation of neurofibrills and that found in Alzheimer's disease can only be distinguished by electronic microscopy.[6]

It could be found that the aluminium concentration is proportional to both the number and distribution of those neurons exhibiting experimentally induced microfibrillar degenerations.

These studies suggest that aluminium may be directly neurotoxic and may promote the development of neurofibrillary degeneration.

FINDINGS IN ALZHEIMER'S DISEASE

Since neurofibrillary degeneration is seen in the human dementia and especially in the Alzheimer's disease, a number of investigators[7] have used the animal studies to form an experimental model of Alzheimer's disease and dementia. Although there are important histochemical and histological

differences between the human and animal experimental findings the major similarities between the aluminium induced encephalopathy of animals and that seen in Alzheimer's disease, suggest that the pathophysiology and pathogenesis of the two states may be related.

In postmortem cases of Alzheimer's disease, the aluminium concentration ranged from 0.4 to 107 μg/g of dry weight, whereas the neurologically normal patients showed an aluminium content of 1.9 ± 0.7 μg/g of dry weight. When regional brain samples were studied it could be found that areas free of neurofibrillary degeneration had aluminium levels of less than 4μ/g and that the density of fibrillary lesions was approximately proportional to the concentration of over 4μg/g.[8,9]

The same concentration could be found in cerebral biopsies of patients with suspected Alzheimer's disease. So it could be proved that the aluminium accumulation occurs early and not as a terminal event with breakdown of the blood-brain barrier.

DIALYTIC ENCEPHALOPATHY

In addition to dementia, which is comparatively a slowly progressing disease, a more acute encephalopathic syndrome has been described in relation to aluminium toxicity.[10,11,12,13] Uraemic patients on haemodialysis receiving aluminium containing gels to control phosphate levels during dialysis developed a dialytic encephalopathy, which involved a mixed dysarthria, apraxia of speech, asterixis, myoclonus confusion, focal seizures, and an abnormal EEG. It has been reported that dialytic encephalopathy is a leading cause of death in long-term haemodialysis patients.

The direct relation between the accumulation of aluminium in the brain and the neurofibrillary degeneration has led to many theories about the role of aluminium in metabolism. These are mentioned below:

(1) Aluminium retards the movement of molecules necessary for membrane receptor activity.

(2) Aluminium acts as an inhibitor of the dihydrobiopterinreductase, which catalyses the regeneration of tetra hydrobiopterin, thyrosin and other neurotransmitters.

(3) It forms complexes with DNA.

(4) It forms complexes with ATP and noradrenaline, which are deposited in the grey substance of the brain. All theories suggest the partial or total reversibility of the process. In allopathic medicine no substance has been introduced yet, which is able to eliminate aluminium from brain tissues.

SYMPTOMATOLOGY

In the following text, the process of senile dementia Alzheimer's disease as a special form of a demential process is explained and the possibilities of application of dynamized aluminium is shown.

Dementia is such a common disease in Western Europe and the USA that it is not regarded as a real disease by many psychiatrists and neuro-

logists, but only as a quantitative variation of the physiological decrease of physical and intellectual ability, that are commonly found in aging people. But there are so many examples especially given by illustrious personalities, who were fully capable of their supreme intellectual abilities in old age (e.g. Samuel Hahnemann) that in the author's opinion any symptom and finding of dementia should be evalnated with the utmost care and the patient treated by Homoeopathy as early as possible because as shown above dementia is reversible when treated efficiently at an early stage of the disease.

By definition of the American Psychiatric Association organic brain syndromes (i.e. dementia) are manifested by the following clinical features:[11]

"Impairment of orientation, impairment of memory, impairment of all intellectual functions such as comprehension, calculation, knowledge and learning, impairment of judgment and lability and chilliness of affect".

These are the criteria by which an unequivocal diagnosis may be established, but they, by no means, dominate each segment of the clinical cause and above all a homoeopathical repertorisation based only on these criteria, would not be successful.

The course of a demential process will be explained and all findings and symptoms that can be found in Kent's *Repertory* (often altered or wrongly formulated), are also included and given below:

The initial symptoms of dementia in most of all cases is a lack of memory. Difficulties arise beeause impairment is usually minimized by the patients with significant dementia. It is emphasized only by the family aud other observers who are likely to perceive the dysfunction with much greater clarity than does the patient.

In the incipient phase of cerebral degeneration the individual experiences diminished energy and enthusiasm. He has less interest and concern for vocational, family and social activity. Lability of affect is common with considerable increase in the overall anxiety level particularly as the individual becomes aware of his failing powers. He has less interest in goals and achievements, diminished creativity, lesser pertinacity to stick to a task, trouble in concentrating and difficulty in avoiding disturbing environmental stimuli. Failures, frustrations, changes of the environment, delays and troublesome decisions, produce more annoyance due to internal political or social discussion which are usual and it is harder to regain mental equilibrium after such disturbances.

The characteristic psychic defence mechanisms every individual owns are utilized more frequently with less than normal effectiveness.

As the disease progresses, the achievement of personal goals and fulfilments becomes less important. The patient becomes more and more absorbed with himself and his own problems and less concerned with the feelings and reactions of other people. His anxiety increases and marked irritability with outbursts of anger may occur. Depression, when present before, may intensify as the patient becomes increasingly aware of his diminished abilities. It is during this early phase of illness accompanied by depression that the

patient often complains about his loss of memory. Mostly the patient complains of loss of memory for current events remarking that memory for past events remains crystal clear. For the physician-in-charge a thorough memory testing is important because in dementia short term memory is less but long term memory is crystal clear. But in other diseases of the central nervous system long term memory is hampered but short term memory remains normal.

During this phase the patient feels difficulties in making plans, dealing with new situations and starting activities. Choices and decisions are avoided. Slowed speech and understanding, unreliability in calculation and impairment of judgment may be very troublesome. This decrease in mental abilities can be demonstrated by psychometric means in a decrease of the I. Q. of 10 to 12 points per year in the Hamburg-Wechasler Intelligence Test.[15] With worsening of the condition, drives and feelings diminish. The patient may even lose interest in other's opinions on him. Time and space orientation become faulty. The patient is easily lost, understanding is troublesome. Some patients are restless and overactive and others lethargic without energy. It is usually in this phase of dementia that the motoric and sensoric neurological signs of brain dysfunction begin to appear. As damage proceeds, the patient becomes apathetic. The human substance of personality is lost. No feeling for danger, pain even threatening death may rouse only the feeblest reaction. The patient is now completely disoriented to place and time, the whole memory is defective and the patient is indifferent or unaware of people and situation.

At the end, there is paralysis, dumbness, incontinence, stupor and coma.

THERAPEUTIC CONSEQUENCES

As quoted above, Alumina does not belong to the well-proven remedies of our materia medica. The drug picture needs a completion and a correction by the results seen in aluminium induced encephalopathy in men and animals and by the clinical observation of dementia and in Alzheimer's disease.

Kent's *Repertory*, too, needs completion and a correction, because some findings and symptoms of the drug picture of Alumina cannot be found in Kent's *Repertory*. Others are wrongly interpreted or found under a wrong heading.

The close connection between increased aluminium concentrations in brains of patients with Alzheimer's disease and the psychopathological findings similar to Alzheimer's disease or in dementia after application of high doses of aluminium lead to the conclusion that dynamized aluminium might be a specific in Alzheimer's disease and certain forms of dementia. It might be thought of an elimination of incorporated aluminium from brain cells of the diseased person after application of aluminium in dynamized form similar to the experiments of Professor Bier[16] with Sulphur. The proof of an increased aluminium excretion under the treatment with dynamized aluminium

might be extremely difficult because of the extremely low coneentrations of alumina in the human body. Psychometric tests seem to fit better to prove a standstill or regression of the disease. If the completion of the drug picture of Alumina is taken in view, one will more often find Alumina as a similimum in patients suffering from a demential process and Alumina will be able to stop dementia or make the process reversible and save the patients and his relatives from endless sufferings.

Case report: The patient was an old barber, 64 years of age. He was married to an immensely vital and merry wife from the south-western part of Germany which added much complication to the case because he could not adjust himself with his lively wife.

The patient reported about two operations on the stomach in 1944 and 1951 respectively and an attack of muscle rheumatism in 1972. Soon after the operations of the stomach he began to suffer from formications in the feet and the fingertips and a polyneuropathia and an anaemia due to lack of iron was diagnosed.

In this case the taking up of the patient's history was of eminent importance because dementia accompanied by paraesthesia in hand and feet can be found also in cases of Vitamin B-12 deficiency, e.g. in funicular myelosis, specially after stomach operation. This could be ruled out by laboratory findings. The patient reported about feelings of anxiety, lack of memory (long and short-term), internal restlessness, inability to make up decisions and a depressed mood. He was shy, listless and without joy. The appetite was poor as well as thirst. Stools were light coloured and soft and passed involuntarily with micturition which was delayed.

The examination showed loss of weight (4.1 kg in 4 months). The tongue was white coloured, the liver was 2½ fingers enlarged but indolent. Reid's sign was positive (tremoling of the head when turning the eyes aside).

The patient was treated with Alumina 12x, an I.V. injection of 2 ml twice a week, later once a week.

By this treatment a dramatic change took place within four months. The gait became erect, the patient was able to dress appropriately by himself and visited my office without company. He was delighted. To prove his rejuvenated memory he confidently told me a joke which he heard on the radio five years ago.

The above case is a proof of therapeutic effect of dynamized Alumina in dementia.

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