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Short Communication

Recent Insights into Alzheimer's Disease: A Progressive Neurologic Disorder

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DESCRIPTION

Alzheimer's disease is a neuropsychiatric disorder affecting elderly people first characterized by Alois Alzheimer in 1906. It is a progressive mental deterioration manifested by memory loss, inability to calculate, visual spatial disturbances, confusion and disorientation. Alzheimer's disease causes 50% of dementia seen in clinics and hospitals. Age is the main risk factors of Alzheimer's disease its prevalence increasing dramatically between 65 and 85 years of age. As life spans and consequently the number of aged people are increasing, Alzheimer's disease may become a major health problem in the coming decades. It ranks fourth among all causes of death worldwide. According to one projection a total of 16 million individuals will be affected by 2050. It is now thought to be affecting 2% of population in the industrialized countries and it is the third leading cause of death in these countries. Thus Alzheimer's disease is a socio-economic problem that requires better diagnostic tools, management and effective therapies to combat this disease effectively. Despite extensive studies the etiology of Alzheimer's disease is not yet clearly understood. But studies have shown that oxidative reactions play a significant role in the pathogenesis of Alzheimer's disease and oxidative stress is primarily responsible for the production of neurofibrillary tangles, the histo-pathological hall mark of the disease which is also generally accompanied by the formation of senile plaques precipitated in the brain [1].

Neuronal loss and impaired synaptic function are the other manifestations. The oxidative stress may be caused by some trace elements. As with many proteins, the mis-metabolism of any metal ion within the body will result in creating levels outside the normal physiological range and can result in biological damage. It is also well known that metals play a crucial role in the functioning of the central nervous system. Toxicological metals such as mercury, cadmium, lead and aluminium have no known biological function and are believed to be detrimental to any organism when absorbed. Interestingly brain appears to be the primary target organ for such metals. While biochemically functional metals are tightly homeostatically regulated, non-essential toxicological metals may be able to freely gain access to an organism by virtue of the fact that they share properties such as ionic charge or other characteristic with their biochemically functional counterparts. Senile plaques are closely related to the aggregation of β-amyloid (Aβ) within the neocortex [2].

There is evidence showing that Aβ precipitation and toxicity leading to Alzheimer's disease is caused by abnormal interactions with neocortical metal ions, such as Cu, Fe and Zn. The fact that the homeostasis of these metals is perturbed in Alzheimer's disease and that they concentrate in senile plaques, neurofibrillary tangles and cerebrospinal fluid is well supported. Hence it is hypothesized that the accumulation of these metals might be the key to the damage caused in Alzheimer's disease and perhaps to its treatment. Metal ions and complexes play key roles in a broad range of processes essential for the function of the brain. Lithium is used to manage bipolar disorder, although its mechanism of action is not understood in molecular detail. Zinc is a major regulator of synaptic transmission and neuronal processes and appears in the synaptic cleft at concentrations greater than 1mM [3].

Iron is involved in respiration, in the synthesis of DNA and neuro transmitters; similarly copper has an important Introduction 6 role in brain metabolism as it is essential for the well-known enzymes Cu-Zn superoxide dismitase, ceruloplasmin, cyto-chrome c-oxidase, tyrosynase and dopamine beta-hydroxylase. Many trace elements like aluminum, manganese, iron, copper, zinc, cadmium, mercury and lead are implicated in Alzheimer's disease. Most of the earlier studies on the imbalance of these trace elements were done on post mortem tissue samples of the brain. Subsequently studies measuring certain trace elements of interest in body fluids of Alzheimer's disease patients like whole blood, serum and Cerebrospinal fluid were undertaken. Like in the case of Parkinson's disease, diagnosis of Alzheimer's disease is also entirely clinical based on symptoms. As on today, there is no specific "blood test" or imaging test that is used for the diagnosis of Alzheimer's disease.

Alzheimer's disease is diagnosed when:

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- A person has sufficient cognitive decline to meet criteria for dementia;
- The clinical course is consistent with that of Alzheimer's disease;
- No other brain diseases or other processes are better explanations for the dementia.

Trace element imbalance is surfacing as potentially instrumental in explaining neuro-degeneration. The trace elements constitute a minute part of the living they are of great importance for growth and development. They are involved in all major metabolic pathways and are thus of fundamental importance in human physiology. They are undoubtedly important for the vital processes of life, but even more important is the fact that their functional forms and their characteristic concentrations must be maintained within narrow limits. Exposures to excessive amounts or deficiencies of trace elements induces a range of clinical and biochemical end-points [4].

Most of the trace elements are essential to life, but they can have deleterious effects when present in excess. Further, it is also possible for some essential elements present in excess to displace other essential elements from their actual function and thus produce toxic symptoms. For instance, zinc is replaced by some elements in zinc finger proteins, thereby affecting their function. Discoveries of both essential and non-essential trace elements can markedly influence several key biological events like cell regulation, processes involved in replication, immune-competence, anti-oxidative effects and other factors have strengthened that variations of trace elemental concentrations in both the extracellular and intracellular environment may markedly influence neuro-degeneration.

In view of the immense importance of trace elements in human physiology and vital role they play in the development and degeneration of neural tissues, considerable amount of research has been carried out to determine the concentrations of various elements in cerebral tissues and to elucidate the effect of variation of these elements in the pathology of various neurodegenerative diseases. Even though extensive work has been carried out to find an association between trace elements and neurodegenerative diseases, and to understand the mechanisms involved in degeneration, no definite conclusions are drawn so far [5].

It is not yet clear whether the excess or deficient levels of these elements in the tissues of brain or other nervous system related structures involving both the central and peripheral nervous systems or in the fluids supporting their functioning are the cause or the consequence of neuro-degeneration process. In order to correctly assess the role played by trace elements in the initiation, promotion or inhibition of degeneration of neurons and to correlate concentration levels of certain trace elements of diseased individuals with that of healthy individuals, there is a need for acquisition of data by trace elemental analysis of serum samples of patients afflicted with these diseases from different regions.

CONCLUSION

Multi elemental analysis of brain and the other nervous tissue could become a powerful diagnostic and prognostic tool and moreover, subsequent interruption or alteration of the trace element supply to the neural tissue could be developed into an effective treatment strategy. Once it is established that excess or deficient levels of particular trace element are responsible for the initiation, promotion and progression of degeneration of neurons, suitable measures can be adopted to achieve optimal concentrations of that element. The interaction among elements in various biological processes is well known. The level of one element influences the activity of another. In other words we can say that the action of an element can either be potentiated or reduced by the presence of another.

The interactions between elements can be synergistic or antagonistic. The deficient levels of a particular element may not necessarily mean that the element is deficient, but it is also possible that high levels of another element are depressing levels of this element by interfering with its absorption. Further, it is also possible for some essential elements present in excess to displace other essential elements from their actual function and thus produce toxic symptoms. Proper chelating agents and antagonistic elements can be supplemented to reduce excess accumulated levels of potentially neurodegenerative elements and prevent their further absorption, provided no adverse effects are produced. Single element studies may be misleading. A subject may be suffering from deficiency of an element, even though the normal amount is consumed.

Therefore, the requirement and hence the nutritional adequacy of a particular element depends on other elements already present in the body chemistry. Moreover, in some single element studies it has been shown that the abnormalities and specific biochemical changes, induced by the deficiency or excess of a certain trace element, can be prevented or cured on restoring the normal levels of that element. This restoration is brought about by making use of elements or compounds which interact with this element. This approach would have been effective had that single element been responsible for the abnormality. If, on the other hand more than one element plays a role in the development of the diseased state then multi elemental analysis rather than single element analysis would be more appropriate. It is plausible to think that better understanding of the mechanisms of trace element interactions might be used in slowing the progression of various neurodegenerative diseases as well as in improving their recovery rate.

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