Unchanged concentrations of plasma fibronectin in Alzheimer's disease

In Alzheimer's disease the capacity to remove intracellular and intercellular debris is considered to be impaired. It is also claimed that abnormalities attributed to Alzheimer's disease can often be observed in peripheral tissues including skin fibroblasts and the blood. It was therefore thought that an analysis of plasma in relation to repair and maintenance systems might be useful to develop simple tools for the diagnosis of Alzheimer's disease.

In man the concentration of fibronectin in the plasma increases exponentially with age. Such changes in plasma fibronectin concentrations are usually associated with the changes in its rates of synthesis, changed proteolytic breakdown, and inefficient scavenging systems. Some age related diseases, such as diabetes and atherosclerosis, show prematurely increased concentrations of plasma fibronectin. There is, however, no report on the concentrations of fibronectin in the plasma of patients with Alzheimer's disease. We therefore estimated plasma fibronectin concentrations in such patients.

Plasma samples from seven patients with Alzheimer's disease (six women and one man, aged between 55 and 81 years) were kindly provided by Dr J Vijg, TNO Institute for Experimental Gerontology, Rijswijk, The Netherlands. The clinical diagnosis of the disease in these patients was made by Dr P Eikelenboom (Valerius Clinic, Amsterdam) and was found to be in accordance with the diagnostic criteria for "possible senile dementia of Alzheimer's type", as described by McKhann et al. The number of years for which these patients have now been under observation is between seven and 14.

Plasma fibronectin concentration (mg/l) was determined in these samples by a double antibody sandwich ELISA technique, using rabbit anti-human fibronectin antibody as the catching antibody and the same conjugated to horseradish peroxidase as the secondary antibody. For comparison, plasma fibronectin concentrations were also determined in a section of the normal Danish population. For this, venous blood samples from more than 90 apparently healthy volunteers aged between 20 and 82 years were taken in dipotassium edetic acid and using standard methods. Concentrations of fibronectin in plasma were determined as described above. All estimates were made simultaneously in multiple replicates and repeated twice at different times.

In an apparently normal section of the population, an age related exponential increase (from 200 mg/l at 20 years of age to 500 mg/l at 80 years) was observed, which is similar to previous estimates. The increase observed was significant (p < 0:01), and the extent of the increase was slight in men than in women. Patients with Alzheimer's disease had unchanged concentrations of plasma fibronectin compared with their age and sex matched normal counterparts. Many other biochemical processes, such as calcium homeostasis, DNA and protein synthesis, and DNA repair, seem to be changed in Alzheimer's disease, and these changes can be identified in the peripheral tissues. Our studies show, however, that impairment of the scavenging system in the nervous tissue of patients with Alzheimer's disease does not occur generally throughout the body.

References

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