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Diabetes mellitus and Alzheimer's disease

Dear Sir,
Recently in a letter in *Diabetologia* [1] it was suggested that "the occurrence of AGE (advanced glycation end products) in both diabetes and Alzheimer's disease might lead one to predict a higher-than-expected incidence of concurrent diabetes and Alzheimer's disease. However, predictions of diabetes and concurrent Alzheimer's disease unfortunately must await epidemiological analyses. . .." "Conversely, life-long glycation may result in compensations, leading instead to discordance of Alzheimer's disease and diabetes".

The Umeå Dementia Research Group reported a population-based study of 839 patients with various dementia diagnoses of whom 457 were classified as multi-infarct dementia (MID), 317 as dementia of Alzheimer type (SDAT) and 65 as confusional states [2]. Sixty patients were found to have diabetes, none in the SDAT group, but 55 in the MID group and 5 in the group of confusional states. Oral glucose tolerance tests were performed in patients not known to have diabetes and compared in patients with SDAT, MID, cerebrovascular diseases, hospitalized control patients and healthy elderly patients. Fasting blood glucose levels were lower and the areas under the oral glucose tolerance curves smaller in the SDAT group compared with the cerebrovascular and the hospitalized control groups. SDAT patients had higher insulin levels than the healthy elderly group. Our findings may be in accordance with the second suggestion mentioned above. However, initially low concentrations of brain transmitters are further negatively influenced by the lack of an important source of energy or perhaps by the lack of a metabolite necessary for the production of transmitters.

Yours sincerely,
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Elevated circulating adhesion molecules in NIDDM – potential mediators in diabetic macroangiopathy

Dear Sir,
Increased expression of endothelial adhesion molecules (AMs) may have a potential role in accelerated diabetic macroangiopathy [1], possibly stimulated by oxidized low-density lipoproteins (LDL) and free radicals derived from glucose auto-oxidation [2], since "classical" risk factors including hyperlipidaemia, hypertension and obesity do not completely account for the increased incidence of atherosclerosis in diabetes [3]. Such adhesion molecules are involved in chemotaxis of circulating monocytes and binding of leukocytes and platelets to the endothelium. This leukocyte recruitment is considered an initial step in development of atherosclerotic lesions [4]. Raised plasma concentrations of soluble circulating adhesion molecules occurring after proteolytic cleavage of the extracellular domain may indicate endothelial activation and increased interaction of leukocytes and platelets with the endothelium as recently shown for a small number of non-insulin-dependent diabetic (NIDDM) patients ($n = 13$) [5] and diabetic patients not requiring insulin treatment [6].

In a cross-sectional study, we therefore analysed serum concentrations of circulating intercellular adhesion molecule-1 (cICAM-1), vascular cell adhesion molecule-1 (cVCAM-1), and endothelial leukocyte adhesion molecule-1 (cELAM-1 = E-selectin) in a large group of metabolically stable NIDDM patients ($n = 159$) and age-matched healthy subjects ($n = 71$) with no clinically manifest infections and who were not receiving any anti-inflammatory medication. NIDDM patients were treated either by diet alone, with additional sulphonylurea or biguanides ($n = 80$), or by conventional insulin therapy ($n = 79$) with two daily subcutaneous insulin injections (clinical data are given in Table 1).

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