

Hyperhomocysteinemia and homozygous *MTHFR* 677C>T mutation finding in an Alzheimer's disease case with metabolic syndrome diagnosis

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Dear Sir,

We read the manuscript entitled "Association between genetic and environmental factors and the risk of Alzheimer's disease (AD)" written by Styczynska M, Strosznajder JB, Religa D, et al. in your journal [4]. In this manuscript, the reported genetic factors (apolipoprotein E-Apo E, endothelial nitric oxide synthase-NOS3, methylenetetrahydrofolate reductase-*"MTHFR"*) were analyzed for the correlation with the environmental factors (homocysteine, vitamin B₁₂, cholesterol) in the etiology of AD. The authors empha-

size that the haplotype profiles of APOE epsilon 4 and NOS3 G alleles were not correlated with the homocysteine, vitamin B₁₂, cholesterol status in the AD patients [4]. On the other hand we have an AD patient with homozygous type *MTHFR* 677C>T mutation with elevated homocystein level, vitamin B₁₂ deficiency and hypercholesterolemia findings (Fig. 1). Additionally our patient had obesity, hypertension, diabetes mellitus type 2, deep vein thrombosis, thrombotic thrombocytopenia findings. Obesity, hypertension, hypercholesterolemia and diabetes

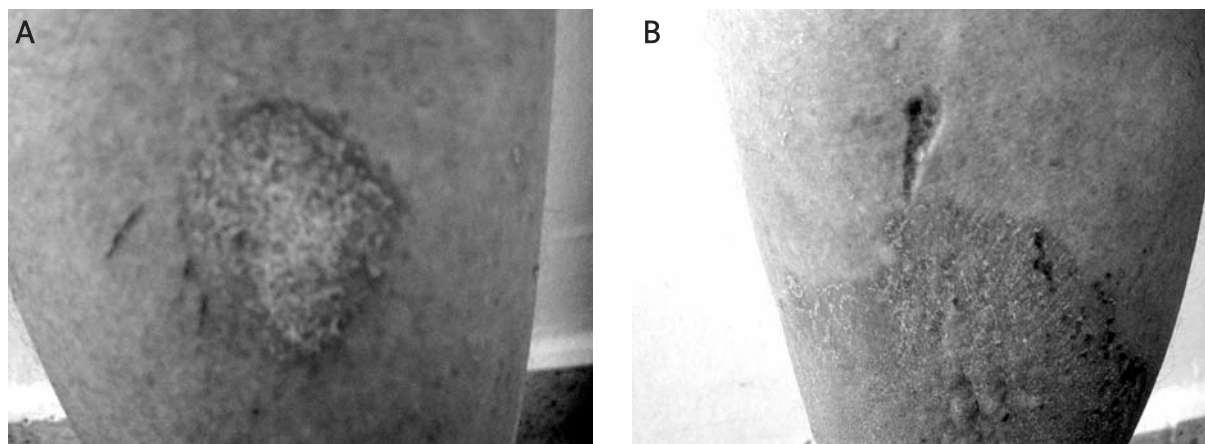


Fig. 1. The ulcerative lesions, observed on the patient's (A) right, (B) left legs. These lesions were probably due to diabetes mellitus and deep vein thrombosis.

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mellitus were noticed in his family history. Due to these findings he had metabolic syndrome diagnosis [1]. In the patient's physical examination, ulcerative lesions were observed on his legs probably due to diabetes mellitus and deep vein thrombosis (Fig. 2) [5]. He had also alcohol consumption history. *MTHFR 677C>T*, *FV R506-Q506*, *FII prothrombin G20210A* mutation analyses for the atherosclerosis findings, cytogenetic analyses in bone marrow samples for thrombotic thrombocytopenia findings were applied [6]. *FV R506-Q506*, *FII prothrombin G20210A* mutation analyses revealed no mutation and bone marrow cytogenetic analyses represented normal karyotype. Homozygous type *MTHFR 677C>T* mutation was observed which supports hyperhomocysteinemia findings. He had long term medication for his complains including B₁₂ vitamin drugs.

As a life-style disorder, no role of the hyperhomocysteinemia with *MTHFR 677C>T* mutation in metabolic syndrome etiology was known. Hitherto, in our case homozygous type *MTHFR 677C>T* mutation with hyperhomocysteinemia may stimulate the metabolic syndrome findings. Also, atherosclerosis is a risk factor for vascular dementia and AD disease [2–4]. Metabolic syndrome and hyperhomocysteinemia with *MTHFR 677C>T* mutation findings cause the generalize atherosclerosis and these findings may be related to the AD in our case which emphasize Styczynska M, Strosznajder JB, Religa D, et al.'s manuscript findings [4].

References

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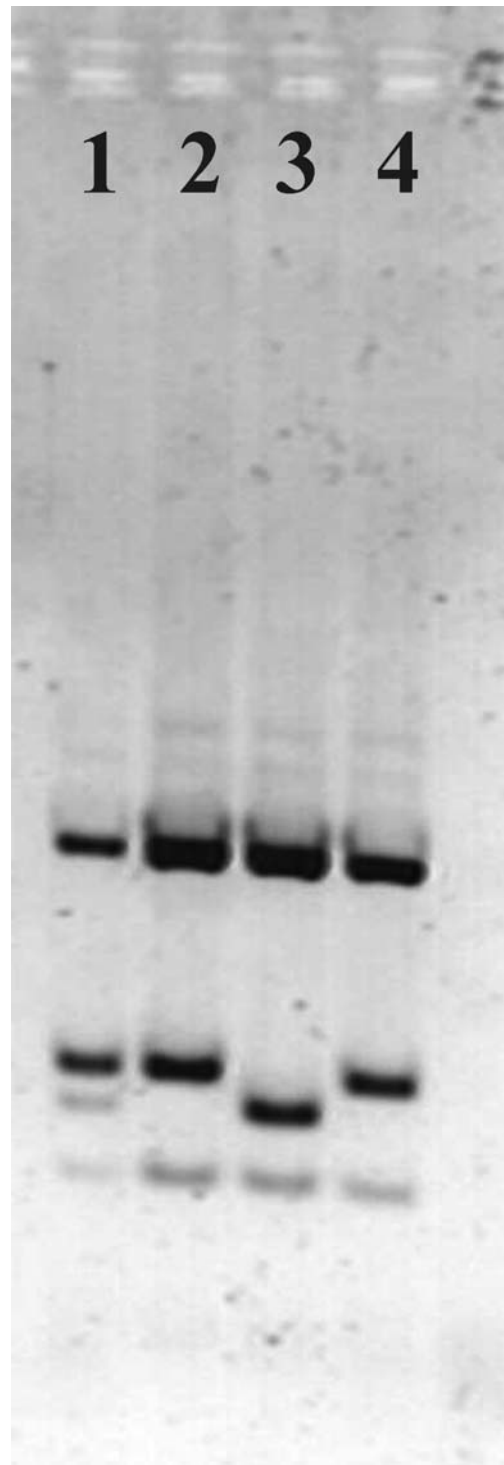


Fig. 2. The patient's DNA revealed homozygous type *MTHFR C677T* mutation. Line 1: Control DNA (heterozygous *MTHFR C677T*). Line 2: Control DNA (Normal), line 3: Patient's DNA (homozygous type *MTHFR C677T*), Line 4: Control DNA (Normal).