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Analysis of oxidative DNA damage and its repair in Polish patients with diabetes mellitus type 2: Role in pathogenesis of diabetic nephropathy

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Distal symmetric polyneuropathy (DSPN) is common chronic complication of type 2 diabetes (T2DM). Its pathogenesis is complex and not entirely known. One of the factors is oxidative stress which develops neuronal apoptosis. In this work we investigated the role of free radicals which may be connected with polymorphisms of DNA repair genes. We also analyzed oxidative induced DNA damage and capacity of their repair. Materials constitute the peripheral blood of patients with T2DM with and without coexisting DSPN and subjects without disturbance of the carbohydrate fraction as the reference group. The study of gene polymorphisms which products taking part in base excision repair pathway: 726 Val/Ala ADPRT, 324 His/Glu MUTYH and 148 Asp/Glu APE was carried out on a group of 209 T2DM patients and on 146 healthy subjects using PCR-RFLP method. The study of DNA damage induced with hydrogen peroxide and also with combination with Fpg endonuclease and the efficiency of their repair in correlation with polymorphisms were carried out on patients in each group using the alkaline version of the comet assay. It was found that in group of patients together with T2DM and T2DM/DSPN 726 Ala ADPRT allele was significantly susceptible to increased risk for T2DM (OR = 1.59; 95% CI: 1.08–2.36). Investigate of DNA damage and repair revealed that T2DM patients have decrease ability to DNA repair. In patients with T2DM/DSPN, it is even less than in people with diabetes alone. ADPRT and APE polymorphisms were significantly associated with higher DNA damages ($P<0.05$) in heterozygous and mutant homozygous in correlation to homozygous wild type, but for MUTYH polymorphism it was not confirmed. These findings suggest that pathogenesis of diabetes mellitus and development of their complication may be related to oxidative stress connected with BER gene polymorphisms.

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