

Effect of inherited thrombophilia on the long-term outcome of percutaneous transluminal angioplasty in patients with diabetes

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Background: Immediate effect of percutaneous transluminal angioplasty (PTA) is influenced mainly by number of patent arteries, whereas the long-term clinical outcome can be modified by possible later re-stenosis (microthrombosis or intimal proliferation). There have been reported multiple alterations in coagulation in diabetic patients - they may be predisposed to thrombosis mostly because of metabolic changes and acquired or inherited coagulation disorders. The aim of our study was to analyse several inherited thrombophilia factors that influence the long-term outcome of PTA and to compare them with diabetic patients without peripheral arterial disease (PAD). **Methods:** Diabetic patients with chronic critical limb ischemia (defined by transcutaneous oxygen pressure [TcPO₂] before treatment < 30 mm Hg) treated by PTA in our centre between January 2008 and December 2011 were included into the study. Patients were divided into unsuccessful PTA group (75 patients) and successful PTA group (58 patients). Diabetic patients without PAD formed control group (65 patients). The clinical success of PTA was defined by necessity of re-PTA or by-pass or TcPO₂ above 40 mm Hg after one year from revascularization. Inherited thrombophilia was assessed by: single point mutations in the factor V gene (Leiden), factor II (prothrombin), genes for methylenetetrahydrofolate reductase - MTHFR (positions of nucleotide C677T and A1298C) and by other coagulation parameters (protein C and S, antithrombin III, fibrinogen, D-dimers, homocystein). All samples were taken from peripheral blood before the procedure and homozygote and heterozygote form of each mutation was analysed. **Results:** Patients in unsuccessful PTA group revealed significantly more frequently heterozygote Leiden mutation (13.3 vs. 1.7 vs. 1.5 %, both $p < 0.01$) and heterozygote prothrombin mutation (12 vs. 3.4 vs. 3.1 %, both $p < 0.05$) compared to successful PTA and control groups. Mutations in genes for MTHFR 677 and 1298 were not significantly different among all groups. There was no significant difference in homozygote mutations and other coagulation parameters among the study groups. **Conclusion:** Our study showed significantly higher frequency of heterozygote form of Leiden and prothrombin gene mutations in diabetic patients with unsuccessful outcome of PTA during first year in comparison with patients after successful PTA and diabetic patients without PAD; these results may support next research of adequate antithrombotic therapy after PTA.

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