INTRODUCTION

Diabetes develops due to a diminished production of insulin (in type I) or resistance to its effects (in type II and gestational), both lead to hyperglycemia ***(Alberti and Zimmet 1998)***.

The aetiology of atherosclerosis, including lower extremity arterial disease (LEAD), is multifactorial. Major risk factors are hyperglycemia, smoking and hypertension, but age, duration of diabetes, obesity, and dyslipidemia also contributes to the risk ***(Dieter et al., 2002)***.

Diabetes increases the risk of developing LEAD 2- to 5-fold ***(Raffetto et al., 2005)***.

The fibrinolytic system is primarily an interaction between tissue plasminogen activators (tPA), and inhibitors (PAI-1) and one response to vascular injury is an activation of tPA. Increased tPA-activity may therefore be a potential indicator of an early ongoing vascular damage, Both tPA and PAI-1 mass levels have been suggested as indicators of vascular damage and studies have indicated an association between the development of LEAD and impaired fibrinolytic potential ***(Tzoulaki et al., 2006)***.

In diabetic patients, elevated plasma levels of t-PA and PAI-1 accompany impaired fibrinolysis ***(Saigo et al., 2004)***.

AIM OF THE WORK

The purpose of this work is to study the relation between plasma level of tissue plasminogen activator (t-PA) and lower extremity arterial disease (LEAD) in diabetic patients.