

Introduction

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The increased procoagulant activity is believed to be one of the factors that contribute to the high incidence of premature macro- and microangiopathy and increased morbidity and mortality, attributable to myocardial infarction, nephropathy and retinopathy observed in diabetic patients (Takada et al., 1993).

Type II diabetic patients showed enhanced activation of the blood coagulation system (Gabazza et al., 1996).

Thrombus formation results from disruption of the equilibrium between prethrombotic and antithrombotic factors that control clotting homeostasis. This imbalance may occur due to an ongoing stimulus to thrombogenesis, a defect of the natural anticoagulant or fibrinolytic system. Perturbance of homeostasis has also been implicated in the development of microvascular complications such as nephropathy & retinopathy in diabetic patients (Gabazza et al., 1996).

Hypofibrinolysis is a common finding in patients with diabetes and is a risk factor for the occurrence of micro- and macroangiopathy (Kannel et al., 1990).

It has been reported that the plasma levels of TAFI are increased in diabetic patients, and it may play an important role in the mechanism of hypofibrinolysis observed in these patients (Hori et al., 2002).

The clinical relevance of the fibrinolytic function in the pathogenesis of thrombosis in diabetes is illustrated by the positive relation of hypofibrinolysis with the presence and severity of diabetic neuropathy (Gabazza et al., 1996).