

INTRODUCTION

Diabetic men and women developed more cerebral, coronary and peripheral vascular disease than non diabetic individuals *Garcia et al (1974)*. Also, *Esko and Pikko(1978)* suggested that diabetic patients have more coronary atherosclerosis and a higher incidence of clinical coronary heart disease than the non-diabetic population of similar age.

The washed platelets, but not platelets rich plasma, from the diabetics show greater sensitivity to aggregation in response to thrombin, collagen and arachidonic acid than controls. Also, there was no significant difference in platelets aggregation angiopathy (*Chen et al., 1990*). On the other hand, *Trip et al. (1990)* Concluded that spontaneous platelets aggregation in vitro is a useful biologic marker for the prediction of coronary events and mortality in survivors of myocardial infarction.

Diabetics had higher levels of fibrinogen, triglycrides but lower HDL-cholesterol values. Fibrinogen values rise throughout the range of blood sugar levels (*Kannel et al, 1990*). Also, diabetics had a higher incidence of obesity and hypertenstion.

Furthermore, high plasma fibrinogen levels are strongly correlated with the frequency of two major thrombotic complications of atherosclerosis, stroke and myocardial infarction (*DiMinno and Mancini, 1990*).

Moreover, *Ceriello et al (1988)*, recorded that induced hyperglycaemia was able to increase factor VII levels in both diabetic patients (type 1) than normal control subjects while, when euglycaemia was achieved in diabetic patients, factor VII Values returned to normal range.

On the other hand, induced hyperglycaemia doesn't change factor X and antithrombin III concentration but in vivo hyperglycaemia produces a decrease of factor X activation but at the same time increases fibrinopeptide A formation due to a greater decrease of antithrombin III, anti-Xa activity (*Ceriello et al, 1990b*).

Four naturally occurring thrombin inhibitors exist in normal plasma. The most important is antithrombin III which contributes approximately 75% of the antithrombin activity. Antithrombin III can also inhibit the activities of factor IXa, Xa, XIa, and XIIa (*Harfenist and Murray, 1993*).

Schernthaner et al, (1989) found that total protein S values were significantly increased and protein C values were decrease in type I diabetes when compared to control group whereas levels of factor VII and free protein S were near normal. Also, there was negative correlation of protein C to glucose.

Total protein S, free protein S, C4 binding protein and protein C were all decrease in liver disease, elevated in diabetes mellitus and normal in collagen disease. (*Takahashi et al, 1989*).

Raised level of plasma fibronectin, an alpha-2 glycoprotein produced by vascular endothelium have been described in diabetic patients with retinopathy and overt nephropathy (*De Giorgio et al, 1988*). Also, *Dyck et al (1990)* recorded that alteration in plasma fibronectins levels may provide insight into the systemic response to acute myocardial infarction.