SUMMARY & CONCLUSION

This study included 60 subjects, 45 patients and 15 normal healthy controls, aged from 41 to 60 years and of both sex, selected from diabetic and coronary care units of Benha and Ain shams university hospitals. They were classified into 4 groups.

1) 15, healthy normal controls.
2) 15, non-insulin-dependent uncontrolled diabetic patients.
3) 15, patients with recent myocardial infarction without diabetes mellitus.
4) 15,patients with both uncontrolled diabetes mellitus and acute myocardial infarction.

From all groups, blood samples were taken (fasting and postprandial) and divided into three parts. The first Part were left to clot and the sera separtated were used for determination of glucose, cholesterol, CPK, LDH, GOT, GPT, albumin, urea and creatinine as a routine investigations.

The second part of the blood samples were taken on EDTA powder and the plasma separated were used for determination of fibrinogen, ATIII, and fibronectin.

The third part of the blood samples were taken on tri-sodium citrate solution (3.2%) and the plasma separated were used for determination of factor VII & X activities and total protein S.

The aim of this work is to study some coagulation parameters (platelet aggregation, fibrinogen, factor VII and X activities), some anticoagulation parameters (AT III and total protein S) and plasma fibronectin in patients with AMI and in diabetics with and without infarction.

A) Diabetic group:

Our findings demonstrated that; there were a significant increase of platelet aggregation, plasma fibrinogen, factor VII activity and total protein S when compared with the normal control group.

The significant increase of platelet aggregation in our findings may be due to increase sensitivity of the platelet to the circulating ADP which was increased in diabetes mellitus due to inhibition of Kreb's cycle. Also, it may be due to imbalance between prostacyclin (PGI_2) and thromboxane A_2 (TXA_2) owing to vascular complication of diabetes mellitus.

Also, the significant increase of plasma fibrinogen could be explained by the increase of fibrinogen as an acute phase reactant protein, increase demand for fibrinogen secondary to repeated formation of microvascular thrombi and /or haemoconcentration secondary to glucosuria.

In addition, the significant increase of factor VII activity may be also due to hypercholesterolaemia which causes atheromatous plaques, rupture of the surface of these plaques leading to the release of tissue factor which activates clotting factor VII.

Moreover, the significant increase of plasma total protein S may be due to increase in the synthetic function of the liver due to repeated formation of small microvascular thrombi.

On the other hand, there was a significant decrease of plasma AT III compared with normal control group. This is may be due to decrease synthesis by the liver and / or increase consumption due to the formation of thrombin-antithrombin III complex.

Also, our findings demonstrated that; there were a non-significant increase of plasma factor X activity and fibronectin compared with normal control group.

Our results showed a significant positive correlation between fasting and postprandial blood glucose with plasma fibrinogen, factor VII & X activities, while there were non-significant positive correlation with platelet aggregation, plasma total protein S and plasma fibronectin. On the other hand, there was non-significant negative correlation with plasma antithrombin III.

Also, our results showed a significant positive correlation between serum total cholesterol with platelet aggregation, plasma fibrinogen, factor VII & X activities, while there were non- significant positive correlation with plasma total protein S and fibronectin. Although, there was non-significant negative correlation with plasma antithrombin III.

Moreover, our results showed a significant positive correlation between plasma fibrinogen and platelet aggregation, while there were non-significant positive correlation with plasma factor VII & X activities, total

protein S and fibronectin. Although, there was non-significant negative correlation with plasma antithrombin III.

B) Acute myocardial infarction group:

Our findings demonstrated that; there were a significant increase of platelet aggregation, plasma fibrinogen, factor VII activity and fibronectin as compared with normal control group.

The significant increase of platelet aggregation in this work may be due to increase sensitivity of the platelet to the ADP, circulating thrombin and /or imbalance between prostacyclin and thromboxane A2 owing to endothelial damage secondary to hypercholesterolaemia.

Also, the significant increase of plasma fibrinogen may be due to increase demand for fibrinogen secondary to thrombo-embolic complication.

In addition, the significant increase of plasma factor VII activity may be due to vascular complications associated with AMI perhaps due to atheroma, in the presence of tissue factor as would occur with the rupture of an atheromatous plaque, the coagulation activity of factor VII increased.

Moreover, the significant increase of plasma fibronectin may be due to increase demand for the healing process of myocardial injury and /or may be due to increase its incorporation into blood which becomes cross linked to fibrin by the factor XIII transglutaminase.

Also, our results demonstrated a non-significant increase of plasma factor X activity compared with normal control group. On the other hand,

there were a significant decrease of plasma AT III and a total protein S compared with the normal control group.

The significant decrease of plasma AT III may be due to decrease synthesis by the liver and/or increase consumption by thrombotic process.

Also, the significant decrease of plasma total protein S may be due to consumption of free protein S during thrombo-embolic attacks which leads to the decrease of total protein S.

Comparative study of AMI versus the diabetic group showed a significant increase of plasma fibrinogen and factor VII activity. while, there were non-significant increase of platelet aggregation, plasma factor X activity & fibronectin. On the other hand, there was a significant decrease of plasma total protein S while, there was a non-significant decrease of plasma AT III concentration in AMI group compared with the diabetic group.

Our results showed a significant positive correlation between fasting and postprandial blood glucose with plasma fibrinogen while there were non-significant positive correlation with platelet aggregation, plasma factor VII & X activities, total protein S and fibronectin. Although, there was non-significant negative correlation with plasma AT III concentration.

Also, our results showed a significant positive correlation between serum total cholesterol with platelet aggregation, plasma fibrinogen, factor VII & X activities while there was non-significant positive correlation with plasma ATIII concentration. Although, there were non-significant negative correlation with plasma total protein S and fibronectin.

Moreover, there was a significant positive correlation between plasma fibrinogen and platelet aggregation while there were non-significant positive correlation with plasma factor VII and plasma fibronectin. Although there were non-significant negative correlation with plasma factor X activity, AT III and total protein S.

c) Diabetics with acute myocardial infarction group:

Our findings demonstrated that; there were a significant increase of platelet aggregation, plasma fibrinogen, factor VII & X activities, and fibronectin as compared with normal control group.

On the other hand, there were a significant decrease of plasma ATIII and total protein S compared with the normal control group.

Comparative study of diabetic with AMI versus the diabetic group showed a significant increase of plasma fibrinogen, factor VII and X activities while, there were a non-significant increase of platelet aggregation and plasma fibronectin. On the other hand, there was a significant decrease of plasma total protein S while there was a non-significant decrease of plasma ATIII concentration.

Moreover, comparative study of diabetics with AMI versus the AMI group showed a significant increase of plasma factor VII activity while, there were a non-significant increase of platelet aggregation, plasma fibrinogen, factor X activity and fibronectin. On the other hand, there were a non-significant decrease of both plasma ATIII concentration & total protein S .

Also, our results showed a significant positive correlation between fasting and postprandial blood glucose with plasma fibrinogen, factor VII & X activities while, there were non-significant positive correlation with platelet aggregation, plasma total protein S and plasma fibronectin. Although, there was non-significant negative correlation with plasma AT III concentration.

In addition, there were a significant positive correlation between serum total cholesterol with platelet aggregation, plasma fibrinogen, factor VII & X activities while there were a non-significant positive correlation with plasma total protein S and fibronectin. Although, there was a non-significant negative correlation with plasma AT III concentration.

Moreover, there were a significant positive correlation between plasma fibrinogen with platelet aggregation and plasma factor VII activity while, there were non-significant positive correlation with plasma factor X activity and total protein S. Although, there were non-significant negative correlation with plasma AT III concentration and fibronectin.

CONCLUSION

Hyperglycaemia and hyperlipidaemia were risk factors for cardiovascular disease. Disturbances of some coagulation and anticoagulation parameters in diabetics before treatment with anticoagulant therapy were a prominent features in the development of acute myocardial infarction. Diabetes mellitus is associated with a "hypercoagulable state". This hypercoagulability could influence the onset of AMI in different ways:

Firstly, fibrinogen exerts its influence through an effect on blood viscosity and fibrin released may be involved in the development of microthrombi.

Secondly, thrombin produced by the coagulation cascade during the occurrence of micro vascular thrombi may cause platelet aggregation. This would likely be effective in the presence of low AT III level.

Thirdly, fibrin is a main constituent of most occlusive thrombi, adding size and stability to platelet aggregates.

Finally, the imbalance between the coagulation and anticoagulation parameters found in our diabetic patients may play a role in the development of acute myocardial infarction (AMI) later on. Whether this imbalance was a cause or effect of AMI is unclear.

RECOMMENDATIONS

Although the use of clotting factors in clinical diagnosis is still growing, they may be of help as a potential risk factors in follow up.

So, we recommend to follow up the diabetic patients by evaluation of some blood coagulation and some anticoagulant prameters as a routine investigation to interfere as early as possible with anticoagulation therapy as a prophylactic treatment, before appearance of complication of coagulopathy.

This may be of clinical value in the correction of imbalance between coagulation and anticoagulation parameters in diabetic patients which could be effective in prevention of thrombo-vascular complications in diabetics before development of ischaemic heart disease.

Also, further study of blood coagulation and anticoagulation parameters in all patients with ischemic heart disease will be recommended.