Original Research Article

Study of plasma fibrinogen in diabetes mellitus with complications

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Abstract

Diabetes mellitus is a complex syndrome catheterized by hyperglycemia, leading to vascular complications such as retinopathy, nephropathy and macrovascular diseases like atherosclerosis. Diabetes patients with and without complications are taken for the study. Fibrinogen levels have been reported to correlate with degree of diabetic control [coller 1978].studies was carried out by[konner 1987] in diabetics it has been reported that plasma fibrinogen levels are increased in diabetics. Thrombi produced from plasma of diabetics with blood glucose 250mg/dl were larger than those produced from plasma of control. This may possibly reflects higher fibrinogen concentration available for clot formation.[ferguson et al 1975].

Key Word: plasma fibrinogen, diabetes mellitus.

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INTRODUCTION

Diabetes mellitus is a group of metabolic diseases charecterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both. The chronic hyperglycemia of diabetes is associated with long term damage, dysfunction, and failure of various organs, especially the eyes, kidneys, nerves, heart and blood vessels.

The cause of diabetic complications is not known and may be multifactorial. Major emphasis has been placed on the polyol pathway where in glucose is reduced to sorbitol by the enzyme aldose reductase

BLOOD RHEOLOGY AND COAGULATION

In the 1970's a no of communications suggested that abnormal viscosity in diabetes resulted in reduced blood flow and increased coagulation. Torpe et al[1983] found increased viscosity in those with proliferative retinopathy associated with increased fibrinogen levels. Interestingly fibrinogen levels have been found to correlate with degree of diabetes control, as indicated by the glycosylated haemoglobin levels[collar1978]. Recently a provocative mechanism for reduced fibrinolysis in diabetes has been proposed based on evidence for impaired degradation of fibrin by plasmin due to glycosylation of fibrin itself [Brownlee 1983].

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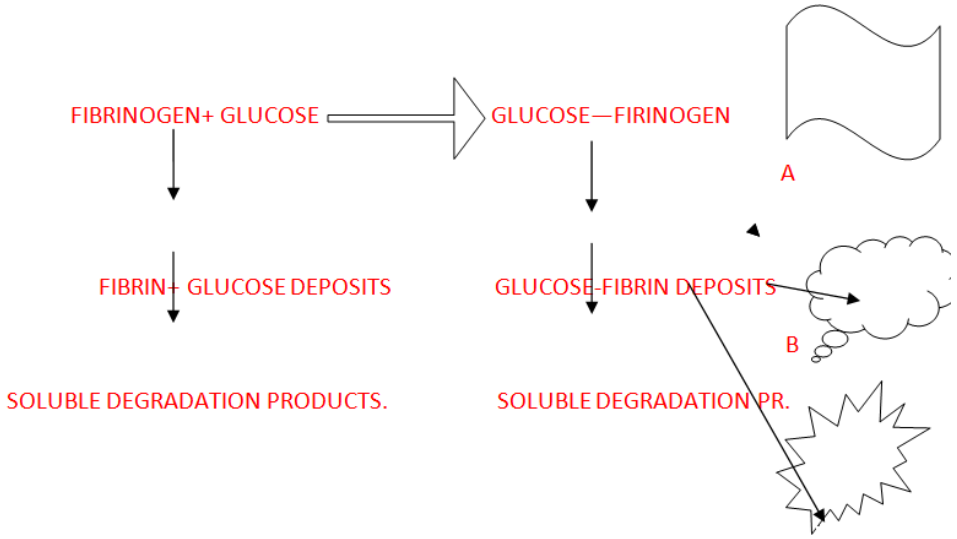

A=Collagen Basement Membrane.
B=Macrophage
C=Endothelial Cell.
Schematic representation of the mechanism by which excessive non-enzymatic glycosylation may lead to fibrin accumulation in various diabetic tissues.

MATERIALS AND METHODS

1. Estimation of Fibrinogen [Reinhold-1953]
Calorimetric Method using Biuret Reaction.
2. Estimation of blood sugar by GOD-POD Method.
3. Estimation of blood urea by Diacetyl Monoxime[DAM] method.
4. Estimation of serum Creatinine by Jaffe's kinetic method.

Parameters	Group—1 Controls	Groups –11 NIDDM without complications	Group—111NIDDM with complications	Significance
Blood sugar.	72.45	159.00	212.64	P<0.01
Blood urea.	20.80	27.50	54.60	P<0.05
Serum creatinine.	0.9	1.2	2.2	P<0.01
Fibrinogen	247.5	783.75	785.0	<0.05

DISCUSSION

Result of study indicate that predisposition of diabetes to develop complications such as retinopathy nephropathy and the predisposition to infection is multifactorial.

Fasting blood sugar levels were found to be raised in all patients who have already developed micro vascular complications control [72.45± 8.662]NIDDM without complications [159± 43.865] .NIDDM with complications [212.64± 0.2731].

Plasma fibrinogen levels were found to be raised in NIDDM with complications [785± 80.099] as compare to NIDDM without complications[783.75+56.899] and control group [247.5± 34.925] increased plasma fibrinogen levels have been reported in diabetes by others also. [Kohner1987].

Fibrinogen levels have also been reported to correlate with degree of diabetic control[Coller 1978].It is well known that increased plasma fibrinogen contributed to increased viscosity of blood and increased predisposition for clotting abnormal viscosity, increased coagulation and reduced blood flow may predispose to coagulation and conversion to fibrin. Glycosylation of fibrin has been reported to result in decreased fibrinolysis by plasmin probably due to decreased binding affinity of enzymes.[Ireland et al 1982]. Defective degradation of trapped of trapped fibrin, due to excessive non enzymatic glycosylation in vivo may result in fibrin accumulation in diabetic glomerular capillary with with poorly degraded plasma proteins trapped and glycosylated, could contribute to progressive microvascular occlusion that occurs in long term diabetes.

CONCLUSIONS

Diabetes Mellitus is a complex syndrome affecting all the metabolisms. This disease predisposes to large number of complications such as retinopathy, nephropathy, neuropathy and predispositions to infections etc. It is seen that plasma fibrinogen levels are higher in patients with high levels of glycosylated haemoglobin. Increased plasma fibrinogen levels could be due to impaired activity of hydrolytic enzyme for glycosylated Hb increased viscosity of blood and decreased blood flow through micro vasculature may lead to accumulation of glycosylated and poorly degraded fibrin and plasma proteins in capillary basement membrane glomeruli and retina.

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