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The Role of Plasm Lipid Profile on RBC Aggregation and Sedimentation Changes in Diabetic Patients

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Abstract:

The aggregation capacity of human reb blood cells lies between that of the non- aggregated arythrocyte and the remarkably full sedimentation. As the ability to aggregate is atributed to many factors such as the availability of macromolecules and plasma lipids , the role of plasm lipid profile on RBC aggregation and sedimentation changes in normal and diabetic patients is studied .Also serum lipid profile measurement (Total cholesterol, Triglyceride, HDL, LDL, VLDL) in normal and diabetic subjects were made .The principle of measurement includes detecting the transmitted laser light through a suspension of 10% diluted red blood cells in plasma .In all diabetics, the raulux formation and sedimentation rate is enhanced.

Introduction:

Vander Waals force is the only force that acts between very close particles [1]. The effect of this electrostatic force is extends for about 15 nm [2]. The mechanism of generation of Vander Waals force is started when two R.B.C's approach one another, so the negative charge is displaced to one side leaving the positive charge on the other side (dipole). This dipole in turn induces another dipoles. This process creates an attraction force responsible for the cohesion, adhesion and then aggregation between the red blood cells both in vivo and in vitro [3]. As the radius of aggregates increase, the attraction force also increases according to Newton's law [4]:

$$F = \text{const.} \ (m_1 \times m_2 / r^2)$$

Where: (m_1) and (m_2) are the masses of two adjacent R.B.C.'s , (r) is the distance between them.

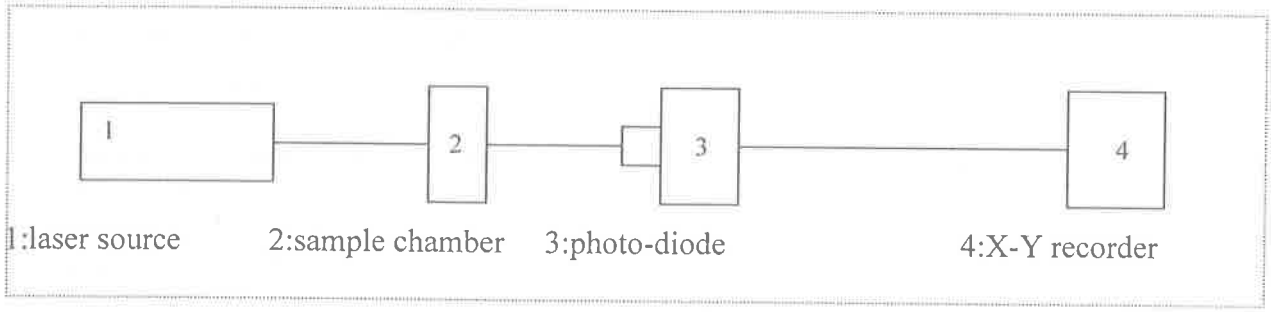
The process of attraction and aggregation is prevented by repulsion force induced by the negative charge on the cellular membrane, and enhanced by the adsorbing force provided by macromolecules ,such as ,plasma proteins, lipoproteins, lipid particles, must be available and act sufficiently. Aggregation of red blood cell, which is a normal physiological feature, increases blood viscosity, causing a slow flow rate [5]. The slowness of flow is essential normally for gases exchange of between R.B.C. and tissue, and visa versa. On the other hand abnormal increased aggregation may play a major role in the pathogenesiss of microangiopathy of many diseases like diabetic neuropathy and retinopathy [6]. The early observations of abnormal flow behaviour of the blood in the microcirculation of diabetic subject have raised the possibility that abnormal physiochemical properties of the vessel contents might act as a cause of the complication of the disease. The aim of this study is evaluate the relation of erythrocyte aggregation kinetics in normal and diabetic patients (during the different phases of sedimentation) to the level of different plasma lipid profiles.

Experimental technique:

A modified method of E. Muraldinharan, (1994) is used [7]. The principle of which includes detecting the transmitted laser light through a suspension of diluted red blood cells in plasma. The samples were prepared from fresh blood samples collected from 30 type I diabetic patients and 20 normal subjects. The blood sample is obtained by venepuncture in a test tube containing Heparin as an anticoagulant. The blood sample was centrifuged at 3000 rpm for 10 minutes to separate the red blood cells from the plasma. The separated plasma was divided into two parts, one for lipid profile tests, and the other to be used in aggregation and sedimentation test. For preparing 10% PCV, we add 100 microleter of packed cells to 900 microleter of plasma. This 10% PCV suspension was placed in a specially designed chamber (locally made of optically flat glass plates) having an internal dimensions of 10 x 0.1 x 0.1 cm. This chamber is then mounted between laser beam and the photo-detector, as shown in the block diagram of figure (1).

RESULTS:

Figure (2a and 2b) showes the aggregation pattern in normal people and in diabetics respectively [10].



Figure(1) : The system layout

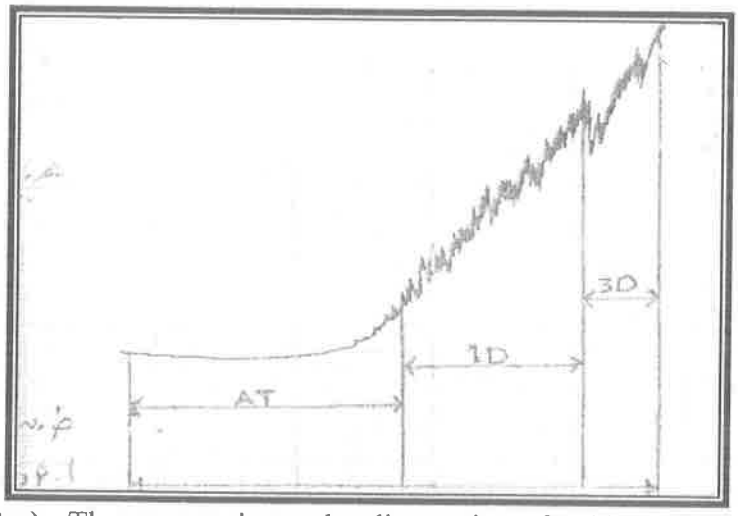


Figure (2-a) : The aggregation and sedimentaion of red blood cells pattern in 10% concentration of normal sample .

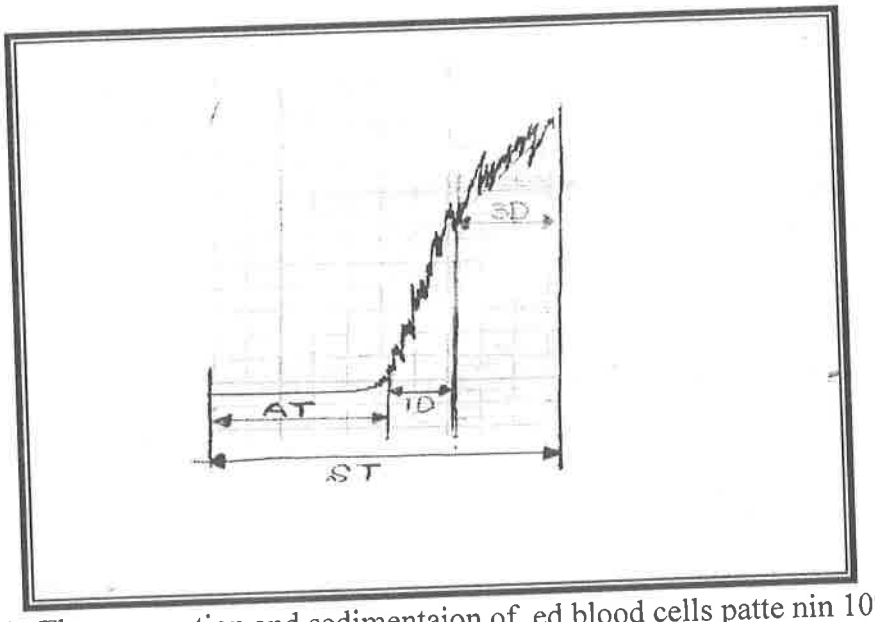


Figure (2-b): The agg egation and sedimentaion of ed blood cells patte nin 10% concent ation of diabetic sample.

In all diabetics, the aulux fo mation and sedimentation ate is enhanced, and showed a significantly sho te du ation. While the time needed fo one and th ee dimension agg egate fo mation, although it is sho ted but it is statistically insignificant (table 1).

Table(1)

The mean + S.D. of agg egation time at diffe ent stages of agg egation,(AT= oulox fo mation, 1D=one dimension agg egate time, 3D=th ee dimensional agg egate and ST= sedimentation time) fo no mal and diabetic voluntee s.

Pa amete	Du ation of phase (ave age min)		T test
	No mal	Diabetic	
AT	14.13 ± 5.08	9.7 ± 2.08	0.011 *
1D	5.6 ± 2.64	2.98 ± 2	0.08
3D	8.61 ± 2.59	6.11 ± 4.52	0.44
ST	23.78 ± 7.37	23.78 ± 7.3	0.0001*

Plasma lipid p ofile measu ement (Total choleste ol, T iglyce ide, HDL, LDL, VLDL) in no mal and diabetic subjects we e made . Significantly high choleste ol and VLDL in diabetic patients we e found. The LDL and the HDL we e significantly lowe

than that of the normal subjects (figure 3). We correlate the duration of the different phases of aggregation and sedimentation in diabetic patients with their lipid profile values. There was a significantly negative correlation between the plasma cholesterol value and 1D , 3D as well as sedimentation duration. While the level of VLDL showed a significantly negative correlation only with the 1D duration.

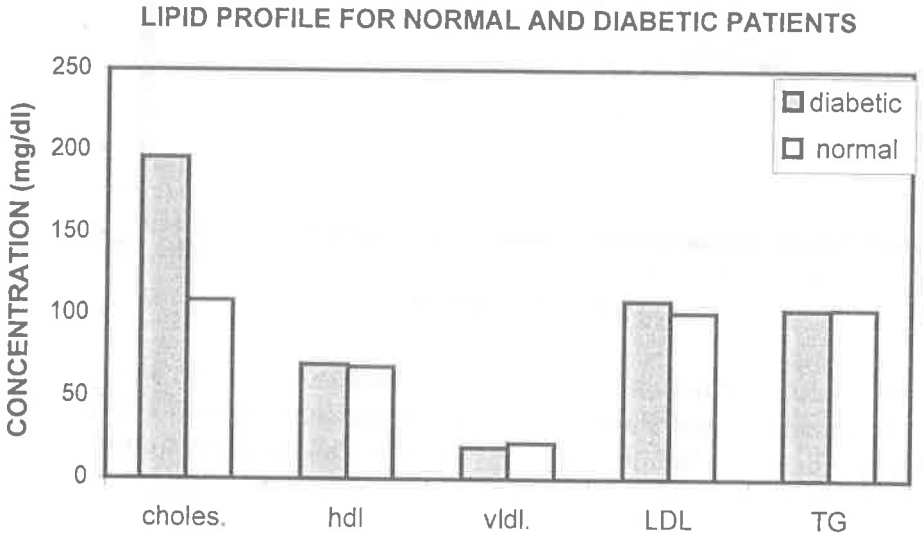


Figure (3): The concentration of cholesterol, triglyceride, and lipid profile (high density, low density, very low-density lipoproteins) for normal and diabetic patients As an average value and a standerd diviation.

Table(2):

The slop (R) of relationship between the aggregation phases and the concentration of cholesterol, triglyceride, and lipid profile (high density, low density, very low-density lipoproteins) for diabetic patients.

Parameter	slop of relationship (R)				
	Cholesterol	triglyceride	high density lipo protein	low density	very low-density
AT	-0.057	-0.128	-0.43	-0.11	-0.11
1D	-0.0871	-0.422	-0.054	-0.096	-0.29
3D	-0.31	-0.427	-0.026	-0.022	-0.36
ST	-0.244	-0.411	-0.128	-0.0028	-0.262
T test (nor. & diab.)	0.1086	0.3383	0.18	0.089	0.576

DISCUSSION:

The lipid represents a large group of compounds that are present in the plasma, it can be separated clinically into the following types cholesterol, triglyceride, LDL, HDL, and VLDL [12]. Cholesterol and triglyceride are the two most predominant lipids found in plasma. The other types of lipid profiles like LDL and HDL were also present in blood of variable level. The association of all these lipid profiles with some pathological changes like atherosclerosis explains the test requested in some disease like diabetes were atherosclerosis is a consequence or a complication. A part from the effect of these substances on the endothelium of the blood vessels, these substances affect many rheological factors related to blood flow and blood aggregation.[13] In our study, there is a significant correlation of different aggregation phases slop (R), with the concentration of cholesterol, triglyceride, and other lipid profile. These findings suggest that the rheological changes explain at least in part the pathophysiological changes in diabetic microangiopathy. This is through their effect on the process of aggregation and sedimentation.[14]

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