HAEMORHEOLOGICAL PATTERN IN HYPERCHOLESTEROLAEMIC PATIENTS IN MUTH ELELE

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ABSTRACT

BACKGROUND: Hypercholesterol is one of the predisposing factors to some disease conditions. AIM/OBJECTIVE: To investigate the haemorheological pattern of hypercholesterolemic patients. SUBJECTS: Thirty (30) hypercholesterolemic patients attending Madonna University Teaching Hospital Elele were used for this study. MATERIALS/METHOD: Fasting blood samples were collected from the patients and analyzed within 4 hours of collection. RESULTS: There was a significant decrease ($p<0.05$) in Packed Cell Volume (PCV), increase in Erythrocyte Sedimentation rate (ESR) and Plasma Viscosity (PV) in the test group compared to the control group while the whole blood viscosity (WBV) was not significant. There was a significant increase in Packed Cell Volume (36.2 ± 3.26) in the male test group compared to the female test group (33.30 ± 2.70). The other parameters were not significant ($p> 0.05$). CONCLUSION: The male hypercholesterolemic individuals appear to be more prone to hyperviscosity than the females because of poor blood flow.

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INTRODUCTION

Rheology in practice is the study of the mechanical properties of condensed matter and in particular complex fluid. The name rheology derives from the Greek word "Rheo" which means to "flow". A solid will usually respond to a stress force by deforming and storing energy elasticity. A liquid has a linear relationship between the shear rate and stress. Complex fluids are interesting because they exhibit a non-linear relationship between the shear rate and strain. The non-linear mechanical properties of complex fluids are attributed to damages in the organization of the molecules that comprise the fluid as they experience a deformatting force. Applying this to human blood, rheology becomes Haemorheology.

Haemorheology, which is the scientific study of the deformation and flow properties of cellular and plasmatic component of blood in macroscopic, microscopic and submicroscopic dimensions and the rheological properties of vessel structure with which the blood comes in direct contact. Many clinical disorders are associated with abnormalities in one or more of the fundamental determinants of blood flow.

Cholesterol is required in membrane of mammalian cell for normal cellular function, and is either synthesized in the endoplasmic reticulum or derived from diet; in which case, it is delivered by the blood stream in low density lipoproteins (LDL)[1]. These are taken into the cell by LDL receptor mediated endocytosis in clathrin-coated pits and then hydrolyzed in lysosome[2].Cholesterol is primarily synthesised from acetyl Co A through the HMG - Co A pathway in many cells and tissues. About 20-25% of total daily production (1g/day) occurs in the liver, other sites of higher rate of synthesis include the intestine, adrenal gland and reproductive organs[3]. Cholesterol is transported towards peripheral tissues by the lipoprotein chylomicrons, very low-density lipoproteins (VLDL) and low-density lipoprotein (LDL)[4]. Large numbers of small dense LDL (sdLDL) particle are strongly associated with the presence of atheromatous disease with the arteries. For this reasons LDL is referred to as "bad cholesterol"[5].

On the other hand, high-density lipoprotein (HDL) particles correlate with better health outcomes and hence it is commonly called good cholesterol[4]. In contrast, having small amounts of large HDL particle is independently associated with atheromantous disease progression within the arteries[6]. Hypercholestrolaemia therefore refers to conditions of presence of high level of cholesterol in the body especially small dense LDL (sdLDL) particles that are associated with atheroma formation in the walls of arteries, a condition known as atherosclerosis, which is the principal cause of coronary heart disease and other forms of cardiovascular disease[7]. Hypercholesterolaemia is not a disease but a metabolic derangement that can be secondary to many diseases and can contribute to many forms of diseases notably cardiovascular

diseases[8]. In contrast, HDL particles (especially large HDL) have been identified as a mechanism by which cholesterol and inflammatory mediator can be removed from atheroma. Increased concentration of HDL correlates with lower rates of atheroma progression and even regression[9].

Hyper-cholesterol is one of the numerous predisposing factors to some disease condition such as atherosclerosis and coronary heart disease, which mostly have to do with the haemorheological pattern of the body. It is therefore important to assay the level of some haemorheological parameters (plasma viscosity, whole blood viscosity, packed cell volume and erythrocyte sedimentation rate) in hyper-cholesterolaemic patients.

MATERIALS AND METHODS

SUBJECTS

The subjects were patients attending clinic at Madonna University Teaching Hospital Elele, Rivers State. The subjects also consist of 15 males and 15 females aged between 21 – 50 years with fasting serum cholesterol equal to or greater than 5.4 mmol/L.

CONTROL

The control consist of 15 males and 15 females of apparently healthy individuals also between the ages of 21 – 50 years with fasting serum cholesterol less than 5.4 mmol/L.

SAMPLE COLLECTION

Sixty (60) blood samples were collected for this work, thirty (30) samples from patients diagnosed as having hypercholesterolaemia through the test of fasting blood cholesterol with values of equal or greater than 5.4 mmol/L and thirty (30) from apparently healthy individuals with normal blood cholesterol levels. Verbal consent was obtained from all the subjects before their blood samples were collected for the tests.

6 mL of blood was collected from the antecubital vein. 1 mL was introduced into a plain dry blood container and allowed to clot. It was then centrifuged at 12,000 revolution per minute for 5 minutes and serum was gotten. 5 mL was introduced into a specimen container containing ethylene diamine tetra-acetic acid (EDTA) anticoagulant.

SAMPLE ANALYSIS

Plasma and Whole blood Viscosity[10]

PRINCIPLE

The test is based on the comparison of the rate of flow of plasma/whole blood and distilled water under equal pressure and constant temperature.

PROCEDURES

The 1 mL syringe was clamped on the retort stand in a vertical position.

- The well-mixed whole blood was suctioned by pulling the plunger of the syringe so that the plunger rises above the upper measuring line.
- The suction was then released by gently pulling out the plunger and the whole blood was allowed to flow through the barrel.
- A stop watch was started when the meniscus got to the upper measuring line, and the time required for the meniscus to pass the lower measuring line was determined, recorded and repeated twice.
- The whole blood was removed from the syringes and then rinsed twice with normal saline and dried.
- The syringe was reclamped and distilled water was determined the same way as whole blood.

CALCULATION OF RESULTS

$$\text{Plasma Viscosity} = \frac{\text{Flow time of plasma (secs)}}{\text{Flow time of D/water (secs)}}$$

$$\text{Whole blood viscosity} = \frac{\text{Flow time of whole blood (secs)}}{\text{Flow time of D/water (secs)}}$$

Erythrocyte Sedimentation rate (ESR) was analyzed using Westergren Method and Packed Cell volume (PCV) was analyzed using microhaematocrit method[11]

RESULTS

A total of 60 samples were analysed. 30 samples (test) from hyper-cholesterolemic age-matched subjects (15 Males and 15 Females) and 30 samples (control) from apparently healthy individuals with normal cholesterol levels (15 Males and 15 Females). The results were analyzed using Statistical Package for Social Science (SPSS) version 14. P value less or equal to 0.05 was taken as significant while p value greater than 0.05 was taken as not significant.

TABLE 1

The mean ± SD of TC, PCV, ESR, RWBV and RPV for hyper-cholesterolaemic subjects (test) and controls.

Parameters	Test (n = 30)	Control(n=30)	P- value
Cholesterol (mmol/L)	6.86 ± 0.88	3.06 ± 0.75	P< 0.05
PCV (%)	34.27± 3.16	37.93 ± 3.35	P< 0.05
ESR (mm/hr)	13.23± 3.43	9.07 ± 0.85	P< 0.05
RWBV (m.Pas)	4.63 ± 0.44	4.63 ± 0.37	P> 0.05
RPV (m.Pas)	1.86 ± 0.22	1.58 ± 0.43	P< 0.05

TABLE 2

The mean ± SD of TC, PCV, ESR, RWBV and RPV for female test and male test.

Parameters	Male test(n=15)	Female test(n=15)	P-value
Cholesterol (mmol/L)	6.78 ± 0.78	6.91 ± 0.94	P> 0.05
PCV (%)	36.20± 3.26	33.30 ± 2.70	P< 0.05
ESR (mm/hr)	12.70± 3.37	14.50 ± 3.52	P> 0.05
RWBV(m.Pas)	4.71 ± 0.28	4.58 ± 0.50	P> 0.05
RPV(m.Pas)	1.93 ± 0.22	1.82 ± 0.22	P> 0.05

Key:

PCV = Packed Cell volume.

ESR = Erythrocyte Sedimentation Rate

RWBV = Relative Whole blood Viscosity

RPV = Relative Plasma Viscosity

n = Number of Total Sample

DISCUSSION AND CONCLUSION

Hyper-cholesterolemia is a condition in which a patient has a cholesterol level above the normal healthy value of that individual considering the sex, age and race. We set out to investigate the effects (if any) of hyper-cholesterol on haemorheological parameters.

Packed cell volume (PCV) shows a statistical decrease ($p<0.05$) in test group (34.27 ± 3.16) compared to control (37.93 ± 3.35). This could be as a result of increased cholesterol level in the test group. Ho (2004) states that PCV could affect the whole blood viscosity (WBV).

For prediction of blood in vivo, erythrocyte sedimentation rate (ESR) can be supplemented by measurement of whole blood viscosity[12]. Erythrocyte sedimentation rate (ESR) showed a statistical significant increase ($P<0.05$) in the test group (13.23 ± 3.43) compared to the control (9.07 ± 0. 85).

Increased blood viscosity occurs in several cardiovascular diseases[13]. We recorded a statistical increase ($P< 0.05$) in plasma viscosity (PV) with values of 1.86 ± 0.22 and 1. 58 ± 0.43 in test group and controls respectively[14,15]. Though plasma fibrinogen was not measured in this study, it could be the cause of increase in plasma viscosity. Plasma fibrinogen contributes to dyslipidemia-induced morbidity[16]. There is a strong correlation between plasma viscosity and fibrinogen[14].

Whole blood viscosity (WBV) was not statistically significant ($p>0.05$). This could be attributed to the lowered packed cell volume (PCV) of the test group. Fibrinogen and cholesterol did not correlate with whole blood viscosity[17]. Low-density lipoprotein (LDL) is the principal lipoprotein independently influencing whole blood viscosity[18]. This low-density lipoprotein (LDL) can be reduced by a single low density lipid lipoprotein apheresis resulting in significant reduction in whole blood viscosity at all shear rates[19]. The male test group was also compared with the female test group.

All the parameters were not significant ($P>0.05$) with the exception of packed cell volume (PCV) with values of 36.20 ± 3.26 and 33.30 ± 2.70 for males and females respectively. This could be attributed to the normal lowered packed cell volume (PCV) of females. Whole blood viscosity or plasma viscosity are increased by male sex, obesity, high sodium intake, aging and black race[13]. Higher packed cell volume (PCV) have also been documented in males with hypercholesterol[20]. Also whole blood viscosity is strongly related to the severity of the metabolic syndrome[21].

We therefore concluded that male hypercholesterolemic individuals appear to be more prone to hyperviscosity than the females because of poor blood flow.

CONFLICT OF INTEREST

We declare that we have no conflict of interest.

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