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Haemorheological Profiles and its Pearson correlation among patients with nephrotic syndrome, South West Nigeria.

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Abstract

Sixty subjects diagnosed of Nephritic syndrome (NS) and 60 apparently healthy aged matched individuals were recruited as control for this project work. Written concent was obtained from the participants and ethical clearance was obtained from Ladoke Akintola University Teaching Hospital Osogbo. The parameters estimated were whole blood viscosity (WBV), Plasma Viscosity (PV), White blood cell count (WBC), Platelets count (PLT), Packed cell volume (PCV), and Fibrinogen concentration (FIBC). Statistically significant increased results were observed in WBV, PV, FIBC ($p<0.05$), while the results of PCV and PLT were significantly reduced compared with the control. Male subjects parameters were compared with the male control, statistically significant results were observed in PV, FIBC and PCV ($P<0.05$). In the female subjects when their parameters were compared with female controls, WBV, PV, FIBC, and PCV were statistically significant ($p<0.05$). The Pearson correlation showed positive linear correlation between WBV and PV ($p<0.05$), WBV and PCV ($p<0.01$), WBC and PLT ($p<0.05$).
Conclusion: The haemorheological parameters in Nephrotic syndrome patients were altered and these might lead to thrombotic episode as a result of Hyperviscosity of blood and also cardiovascular episode due to overwork of the heart.

Keywords: Blood viscosity, Plasma viscosity, Nephritic Syndrome, Fibrinogen, Hyperviscosity.

Introduction

Nephrotic syndrome (NS) is one of the major kidney diseases that affect man, both children and adult can be affected. Nephrotic syndrome is a condition that is often caused by any group of diseases that damage the kidney's filtering system (the glomeruli), such as glomerulonephrities which is the common cause; others include diabetic nephropathy and glomerulonephrities. Nephrotic syndrome is a chronic disorder, characterized by alterations of permiselectivity at the glomerular capillary wall, resulting in its inability to restrict the urinary loss of protein. Urinary losses of immunoglobulin as part of unselective proteinuria lower the patient's resistance to infections and increase the risk of serious sepsis and peritonitis (Bagga and Srivastava, 2005). Nephrotic syndrome is a leading cause of paediatric admission outside urinary tract infection (UTI) and post streptococcal glomerulonephritis in Nigeria (Eke and Eke, 1994; Aburrahman *et al.,* 1990). Most caucasian series report NS as a disease of pre-school aged children with peak age incidence of 2-3 years, affecting more males than females (McEnery and Frederic, 1976; Manoo *et al.,* 1990).

Nephrotic syndrome is characterized by massive loss of urinary protein (primarily albumin) leading to hypoproteinemia (hypoalbuminemia) and result into oedema because of reduction in the oncotic pressure (Roy *et al.,* 2013). Hyperlipidemia, hypercholesterolemia and increased lipiduria are usually associated. Thoug not common hypertension, haematuria and azotemia may occur (Gowenlock *et al*., 1988).

Nephrotic syndrome is categorized into primary and secondary forms. The primary nephrotic syndrome occurs without any previous disease and is designated as idiopathic nephrotic syndrome while secondary NS relates to some clinical disease such as systemic lupus erythromatus, diabetes mellitus, sickle cell disease or syphilis. The secondary type are rare in children (Niaudet, 2004). Nephrotic syndrome in adult or children may be caused by primary (idiopathic) renal disease or by a variety of secondary causes.

In adults, diabetes mellitus is the most common secondary cause; focal segmental glomerulosclerosis and membranous nephropathy are the most common primary causes (Roy *et al*., 2013). Venous thromboembolism is a possible complication; acute renal failure and serious bacterial infection are also possible, but much less common (Charles, 2009)

Materials and Methods

A total of 38 adult males and females age within 18 years and above, and 22 children age range between 2½ years and 17 years together with 60 apparently healthy non nephrotic syndrome subjects'(children age 3 to 17 years and adult age 18 years and above) were recruited for this work.

Inclusion and Exclusion criteria: Subjects with 24 hrs urinary protein ≥3.5g timed collection) or albumin >1.5g/24hr urine (random) (David and Christopher, 2003).

Exclusion criteria -Patients on oral anticoagulants, those who had recent blood/blood products transfusion, patients with other complications excluding nephrotic syndrome and recent surgery were excluded.

Blood collection

About 10ml of venous blood was collected by vene-puncture. 5ml was dispensed into Na_2^+ EDTA anticoagulant bottle for haemorheological parameters (Platelets count, White blood cell count, packed cell volume, whole blood and plasma viscosities) and 4.5ml into 0.5ml (3.8%) of sodium citrate bottle for fibrinogen concentration.

Urine specimen collection: A 24 hour urine sample was collected into a jar containing 1ml of 1N HCl (as preservative) from each of the subjects and random urine samples from the control subject for urinary protein estimation.

Haemorheological parameters: Reild and Ugwu (1987) method was employed for whole blood and Plasma viscosity while Ingram (1961) method was used for fibrinogen concentration. Dacie and

Lewis (2012) methods were employed for packed cell volume, White blood cell count and platelet count.

Statistical Analysis: The statistical analysis was performed using SPSS 25.0 software for haemorheological parameters for the subjects and unpaired students test, $p<0.05$ was accepted as statistically significant.

Results

Table 1.0 Overall haemorheological profiles of both children and adults with nephritic syndrome.

Statistically significant ($p<0.05$) increased results were observed in WBV, PV, FIBC, while PCV and PLT showed statistically significant ($p<0.05$) reduced value when compared to the control subject. There was no significant different in the white blood cell (WBC) ($p>0.05$).

Table 1.0 Haemorheological Profiles of Patients with Nephrotic Syndrome and Controls (mean±sd)

Parameters	Test Group n=60	Control Group n = 60	p-value
WBV (mPa.s)	5.79±1.53	4.84±0.89	<0.05
PV (mPa.s)	2.62±0.79	1.85±0.25	<0.05
FIBC (g/L)	4.82±1.34	3.08±0.79	<0.05
PCV (L/L)	0.35±0.07	0.41±0.04	<0.05
WBC ($x10^{12}$/L)	6543.80±2472.58	6197.00±3607.44	<0.05
PLT ($x10^{12}$/L)	212320.00±8041.69	2438080.00±5667.78	<0.05

Key: WBV= Whole blood viscosity, PV= Plasma viscosity, FIBC= Fibrinogen concentration, PCV= Packed cell volume, WBC= White blood cells, PLT= Platelets, X=Mean, SD = Standard deviation, ±= plus or minus, > = p greater than 0.05, < = p less than 0.05.

Table 2.0 Haemorheological profiles of male (adults and children) with nephrotic syndrome and males without nephrotic syndrome controls. The experimental differences seen in the whole blood viscosity was not statistically significant ($p>0.05$). Statistically significant increased ($p<0.05$) results were observed in PV and FIBC ($p<0.05$), PCV was statistically reduced. The differences existed between WBC was not statistically significant ($p>0.05$). The experimental differences observed in PLT was significantly lower in patients with NS ($p<0.05$).

Table 2.0 Haemorheological profiles of male with nephrotic syndrome and male control

Parameters	Test Group n=34	Control Group n = 34	p-value
WBV (mPa.s)	5.50±1.49	4.96±0.97	<0.05
PV (mPa.s)	2.53±0.74	1.92±0.25	<0.05
FIBC (g/L)	4.51±1.19	3.08±0.76	<0.05
PCV (L/L)	0.35±0.07	0.42±0.04	<0.05
WBC ($x10^{12}$/L)	6334.70±2066.17	5951.50±1623.71	<0.05
PLT ($x10^{12}$/L)	201880.00±74628.28	251210.00±53849.84	<0.05

Key: WBV= Whole blood viscosity, PV= Plasma viscosity, FIBC= Fibrinogen concentration, PCV= Packed cell volume, WBC= White blood cells, PLT= Platelets, X=Mean, SD = Standard deviation, ±= plus or minus, > = p greater than 0.05, < = p less than 0.05.

Table 3.0 Haemorheological profiles of female patients with nephrotic syndrome and female without nephrotic syndrome (controls). Statistically significant higher values (p>0.05) were obtained in WBV, PV and FIBC in patients with nephrotic syndrome (p>0.05). Significantly lower result was obtained in the PCV (p>0.05). The results of WBC, and PLT showed experimental difference but the differences were not statistically significant (p>0.05).

Table 3.0 Haemorheological profiles of female patients with nephrotic syndrome and Controls

Parameters	Test Group n=26	Control Group n = 26	p-value
WBV (mPa.s)	6.28±1.41	4.57±0.91	<0.05
PV (mPa.s)	2.77±0.87	1.77±0.21	<0.05
FIBC (g/L)	5.28±1.42	3.08±0.85	<0.05
PCV (L/L)	36.92±5.99	42.35±2.62	<0.05
WBC ($x10^{12}$/L)	6384.00±2962.44	5414.00±525.47	<0.05
PLT ($x10^{12}$/L)	223860.00±8591	249380.00±67410.43	<0.05

Key: WBV= Whole blood viscosity, PV= Plasma viscosity, FIBC= Fibrinogen concentration, PCV= Packed cell volume, WBC= White blood cells, PLT= Platelets, X=Mean, SD = Standard deviation, ±= plus or minus, > = p greater than 0.05, < = p less than 0.05.

Table 4.0 Comparison of haemorheological profiles of male and females patients with nephrotic syndrome. Statistical significant lower differences were noticed in WBV and FIBC in male with nephrotic syndrome patients. Experimental differences noticed in other parameters such as PV, PCV, WBC and PLT were not statistically significant (P<0.05).

Table 4.0 Comparison of male and female haemorheological profiles of Patients with nephrotic syndrome.

Parameters	Male Group n=34	Results X ± SD Female Group n = 26	p-value
WBV (mPa.s)	5.49±1.48	6.28±1.41	<0.05
PV (mPa.s)	2.51±0.70	2.77±0.87	<0.05
FIBC (g/L)	4.51±1.19	5.28±1.42	<0.05
PCV (L/L)	35.06±7.40	36.96±5.83	<0.05
WBC ($x10^{12}$/L)	6334.70±2066.00	6794.20±2953.11	<0.05
PLT ($x10^{12}$/L)	201880.00±7468.28	233350.00±76444.98	<0.05

Key: WBV= Whole blood viscosity, PV= Plasma viscosity, FIBC= Fibrinogen concentration, PCV= Packed cell volume, WBC= White blood cells, PLT= Platelets, X=Mean, SD = Standard deviation, ±= plus or minus, > = p greater than 0.05, < = p less than 0.05.

Table 5. Pearson correlation of haemorheological profiles of patients with nephritic syndrome. Positive significant correlations were observed between the following parameters PV and WBV (r=0.286*, p<0.05), WBV and PCV (r=0.381**, p<0.01), WBC and PLT (r=0.254*,p<0.05).

Table 5. Pearson correlation of haemorheological profiles of patients with nephritic syndrome

Parameters	Parameters	Correlation Coefficient (r)	P-value
WBV	PV	.286*	0.05
WBV	PCV	.381**	0.01
PLT	WBC	.254*	0.05

* Correlation is significant at the 0.05 level

** Correlation is significant at the 0.01 level

Key: WBV= Whole blood viscosity, PV= Plasma viscosity, PCV= Packed cell volume, WBC= White blood cells, PLT= Platelets

Discussion

Haemorheology is the study of how blood flows within the vascular system of the body and this depends on blood viscosity. Blood viscosity is a measurement of the thickness and stickiness of a individuals blood. This important hemodynamic biomarker determines the amount of friction against the blood vessels, the degree to which the heart must work, and the quantity of oxygen delivery to the tissues and organs. It has been reported that elevated blood viscosity is a strong independent predictor for cardiovascular events (Koenig *et al*., 1998). In the Edinburgh Artery study, it was reported that elevated blood viscosity was the strongest predictor of stroke risk after controlling all other major risk factor (Ciuffetti *et al*., 2005: Lee *et al*., 1998). The result of this research work showed that the haemorheological profiles of patients with nephrotic syndrome were altered compare to the apparent healthy individuals.

The whole blood viscosity which is one of the parameters of haemorheology was significantly higher in individuals with nephrotic syndrome. This result corroborates the previous findings by other researchers, (Coppola *et al.*, 2000; de la Torre, 1999; Ernst *et al.*, 1991). The decreased oxygen -carrying capacity of higher-viscous blood affects cognitive function, as well as the function of any tissue to which robust oxygen delivery is essential. The consequences of hyper viscous blood primarily results in damage to the blood vessel, over work of the heart, and decreased delivery of oxygen to the tissues. Highly viscous blood which was as a result of lost of albumin with the resultant of increase in globulin that contribute to the hyper viscosity pounding against the walls of the blood vessel lead to abrasion of the single-cell layer of the intima in the carotid, pulmonary, and coronary arteries. The body responds with a protective adaptation, creating a scab (plaque), which eventually calcifies in an effort to protect the body vessel. The longer term result is increased turbulence and an ever-narrowing channel for blood flow. This result requires the heart to work harder, pushing the viscous blood out at even higher pressures, further damaging the intimal layer. At the other extreme of the vascular tree, decreased perfusion of the tissues as the stiffened erythrocytes of viscous blood scour the capillary lining occur. The body responds by thickening the capillary walls decreasing diffusion of oxygen and nutrient into tissues. This effect is most pronounced in tissues where healthy capillaries are essential for unimpaired function such as the kidney, eyes, fingers and toes (Kensey and Cho, 2007; Sloop, 1996). Elevated blood viscosity is a strong independent predictor of cardiovascular events (Koenig *et al.*, 1998).

The plasma viscosity was found to be significantly increased in nephrotic syndrome patients compared to the control in this study. Plasma viscosity refers to the thickness of the fluid portion of blood which contains plasma proteins, hormones, vitamins and other body essentials nutrients. McGinley *et al*, (1983) also reported significant increased blood viscosity in nephrotic syndrome in their work.

The male patients with nephrotic syndrome plasma viscosity was compared with male control plasma viscosity, significant increased level was recorded. This observation was the same when the female patients with nephrotic syndrome plasma viscosity were compared with apparently healthy female control. This result corroborates the result of previous study done (McGinley *et al.,* 1983). Elevated plasma viscosity has been shown to have a deleterious effect on oxygen delivery to the ischemic myocardium (Gordon *et al*., 1974; Wells, 1972). The comparison of male and female patients with nephrotic syndrome haemorheological profiles were done, significant differences were noticed only in the whole blood viscosity (WBV) and fibrinogen concentration (FIBC). These differences may be prompted by the hormonal differences in the male and female gender. The experimental differences noticed in other parameters were not statistically significant.

The packed cell volume (PCV) result of nephrotic syndrome patients indicates relative aneamia compared to the control. This was also reported by Adedoyin *et al*, (2006), Vogt and Avener (2004). Davis and Avener (2004) reported the same in their study. Kidney is involved in the production of red cells by the bone marrow by stimulating the production of an hormone called erythropoietin. The production of this hormone is reduced or completely ceased in some renal diseases depending on the severity of the renal impairment (Vogt and Avner, 2004). Urinary losses of erythropoietin in nephritic syndrome have been shown to cause erythropoietin deficiency anaemia and prevent normal increase in plasma erythropoietin level in response to anaemia in hypoxia (Shibasaki *et al*., 1994). The consequence is a reduction in red cell production by the bone marrow resulting in varying degrees of anaemia. The capacity of blood to carry oxygen to the tissue is directly correlated with haematocrit. It is also inversely correlated with blood viscosity. The relationship of these two parameters is expressed as the oxygen delivery index, the oxygen delivery index was calculated as the ratio of haematocrit to the whole blood viscosity (Bowers *et al.,* 2008)

The packed cell volume of male patients with nephrotic syndrome were compared to the control, significantly reduced level was observed. The result of packed cell volume obtained from female patients with nephrotic syndrome when compared with the female controls was not different from that of male. This corroborate the previous work by Shibasaki *et al* (1994) where they recorded significant lower haematocrit in nephrotic syndrome patients in relation to apparently healthy individual controls. Studies have also shown that in both human and animal there are significant urinary losses of both erythropoietin (EPO) and transferring in nephritic syndrome and prevent the normal increase in plasma EPO level in response to anaemia and hypoxia in nephrotic syndrome (Vaziri, 2001). No significantly difference was recorded when the PCV of male and female patients with nephritic syndrome were compared. This indicates that anaemia in nephrotic syndrome has no gender influence but on the severity of the disease.

The total white blood cells (WBC) count in the study groups were not significantly different and these indicate that the disease does not affect WBC counts. It is however not known from this study if any other aspect of WBC functions were affected. The result of this study corroborates the findings of Adedoyin *et al*, (2006). The experimental differences observed when the male nephrotic syndrome patients WBC was compared to the male controls and female nephrotic patients were compared to females controls were not statistically, this also was observed when WBC of both males and females nephrotic patients were compared together.

Platelet is a none nucleated cells produced from the bone marrow and actively involved in blood coagulation. In this study, platelet count in nephrotic syndrome patients was significantly lower than the control. This was in agreement with the study of Yalcinkaya *et al*, (1995) where similar report was recorded in patients with nephrotic syndrome and the control. This was contrary to the report of Farida *et al*, (2011), Mortazavi and Majidi (2008) and Ananda *et al*, (1996) where they found a significant increase in platelet counts of patients with nephrotic syndrome.

The role of platelet in the generation of hypercoagulability seen in patients with nephrotic syndrome as reported by some researcher (Sirolli *et al*., 2002) might not be due to the quantitative aspect of platelet but on the qualitative. The platelets counts of nephrotic syndrome of male patients were significantly lower when compared to the male control as observed in this study. Experimental differences observed when the females with nephrotic syndrome platelet and female control were compared were not statistically significant. This was also observed when males and females patients with NS platelets were compared.

Haemorheological profiles of male with nephrotic syndrome were compared to their male control counterpart. Significantly increased differences were observed in plasma viscosity (PV) and fibrinogen concentration (FIBC) but significant lower differences were observed in PCV and PLT while the experiment differences observed in WBV and WBC were not statistically significant. This also confirms a derangement of haemorheological profiles in patients with nephrotic syndrome.

Haemorheological profiles of females with nephrotic syndrome and female controls were compared, statistically significantly increased values were observed in the WBV, PV, and FIBC while the difference seen in PCV was lower. Experimental differences were seen in WBC and PLT but were not statistically significant. This also confirmed that the haemorheological profiles of male and female nephrotic syndrome patients were altered compared to apparent healthy individuals of the same gender.

Male and female patients with nephrotic syndrome haemorheological profiles were compared, significant lower differences were obtained in WBV and FIBC of male ($P<00.5$). This implies that the whole blood viscosity and fibrinogen concentration of female nephrotic syndrome were higher than their male counterpart. Other parameters such as PV, PCV, WBC and PLT showed experimental differences but not statically significant. Reason for this might be due to the monthly loss of blood being experienced by the females.

It has been reported that blood viscosity is more strongly related to left ventricular hypertrophy than any other haemodynamic (Devereux *et al*., 1984), and is associated both with the degree of peripheral atherosclerosis (Lowe *et al*., 1993), and coronary artery disease (Lowe *et al*., 1980). An increase in blood viscosity indicate an important cardiovascular risk factor (Coul *et al*., 1991).

The linear correlation between plasma viscosity and whole blood viscosity observed in this study corroborate the work of George and Mayer (1966), Celik *et al*, (2017) where it was observed that an increase of plasma viscosity lead to increase level of whole blood viscosity. Whole blood viscosity is affected by a number of factors among which plasma proteins are major components. They exert their effects directly or through their influence on red cell aggregation (Gayatri and Jyotismita, 2015). A strong linear relationship between the whole blood viscosity and packed cell volume was observed also in this work. This was also in line with the previous work done (George and Mayer, 1966). It was observed also that a linear correlation exist between total white blood cell count and platelets, these are part of cellular components of blood. It has been confirmed that increase in these two components can also be a contributed factor to the viscosity of blood (Chao-Hung, 2004).

Conflict of interest: Authors declare that there is no conflict of interest.

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