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Some Coagulation Parameters among Primary Hypertensive Subjects Attending University of Abuja Teaching Hospital Abuja, Nigeria

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Abstract

Objective: *This study investigated some coagulation parameters of Primary hypertensive subjects to evaluate the effects of the disease on these parameters as well as age and gender based variations among the subjects.*

Materials and Methods: *Blood samples were collected from 76 known hypertensive subjects between 30 – 70 years attending the Cardiology clinic of the University of Abuja Teaching Hospital Gwagwalada. Another 37 normotensive subjects between 30 – 65 years served as the control. All subjects gave their consents. Prothrombin Time Test, Activated Partial Thromboplastin Time Test and Platelets count were determined following standard protocols.*

Results: *Results for Hypertensive and Control subjects were: Prothrombin, 14.10±1.677 and 10.86±0.71 seconds, Activated Partial Thromboplastin Time, 36.39±6.23 and 24.86±2.83 seconds, Platelet count, 248.7552.45 x10⁹/l and 284.95±27.66 x10⁹/l respectively. There was significant increase ($P<0.05$) in Prothrombin time, international normalized ratio and Activated Partial Thromboplastin Time, while Platelet was significantly decreased when compared with control. Across male and female genders, Prothrombin time, international normalized ratio and Activated Partial Thromboplastin Time, were significantly higher among the test subjects when compared with the control subjects; while Platelet count was significantly lower ($P<0.05$). the increase in Prothrombin time, international normalized ratio and Activated Partial Thromboplastin Time was more pronounced among the male hypertensive subjects. Also, the reduction in platelets count was more pronounced among the male hypertensive subjects.*

Conclusion: *These variations suggest possible haemostatic system dysfunction with regard to coagulation and thrombosis. Hence, there is need for constant monitoring to avoid haemostatic complications and to aid early and timely management of the patients. Platelets count, Prothrombin time (PT) and activated partial thromboplastin time (APTT) investigations should be used as prognostic indices for evaluating hypertensive patients for effective management.*

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Introduction

Hypertension (HTN or HT), also known as high blood pressure (HBP), is a long-term medical condition in which the blood pressure in the arteries is persistently elevated [1]. High blood pressure typically does not cause symptoms [2]. Long term high blood pressure, however, is a major risk factor for coronary artery disease, stroke, heart failure, atrial fibrillation, peripheral vascular disease, vision loss, chronic kidney disease, and dementia [3].

It is classified as either primary (essential) high blood pressure or secondary high blood pressure4. About 90-95% of cases are primary, defined as high blood pressure due to non-specific lifestyle and genetic factors [4]. Excess salt in the diet, excess body weight, smoking, and alcohol use are lifestyle factors that increase the risk [2]. High blood pressure due to an identifiable cause, such as chronic kidney disease, narrowing of the kidney arteries, endocrine disorder, or the use of birth control pills are categorized as secondary high blood pressure [4] .

For most adults, normal blood pressure at rest is within the range of 100 - 130 millimeters mercury (mmHg) systolic and 60 - 80mmHg diastolic [5]. If the resting blood pressure is persistently at or above 130/80 or 140/90mmHg, it is high [4]. Hypertension is rarely accompanied by symptoms, and its identification is usually through screening, or when seeking health care for an unrelated problem. Some people with high blood pressure report headaches (particularly at the back of the head and in the morning), as well as light headedness, vertigo, tinnitus (buzzing or hissing in the ears), altered vision or fainting episodes [6]. These symptoms, however, might be related to associated anxiety rather than the high blood pressure itself [7].

In hypertensive emergency, there is evidence of direct damage to one or more organs [8]. The most affected organs include the brain, kidney, heart and lungs, producing symptoms which may include confusion, drowsiness, chest pain and breathlessness [9].

The prothrombin time (PT) and its derived measures of prothrombin ratio (PR) and international normalized ratio (INR) are measures of the extrinsic pathway of coagulation. The APTT in contrast to the PT measures the activity of the intrinsic pathways of coagulation. Endothelial damage, platelet hyper activity, and other changes of blood coagulation may play a role in the vascular complications of essential hypertension [10].

Platelets, also called thrombocytes are a component of blood whose function (along with the coagulation factors) is to react to bleeding from blood vessel injury by clumping, there by initiating a blood clot [11]. Platelets have no cell nucleus: they are fragments of cytoplasm that are derived from the megakaryocytes of the bone marrow, and then enter the circulation [12].

One major function of platelets is to contribute to haemostasis: the process of stopping bleeding at the site of interrupted endothelium. They gather at the site and unless the interruption is physically too large, they plug the hole. First, platelets attach to substances outside the interrupted endothelium: adhesion. Second, they change shape, turn on receptors and secrete chemical messengers: activation. Third, they connect to each other through receptor bridges: aggregation [13]. Formation of this platelet plug (primary haemostasis) is associated with activation of the coagulation cascade with resultant fibrin deposition and linking (secondary haemostasis). These processes may overlap; the spectrum is from a predominantly platelet plug, or "white clot "to a predominantly fibrin, or "red clot "or the more typical mixture. Some would

add the subsequent retraction and platelet inhibition as fourth and fifth steps to the completion of the process and still others a sixth step wound repair [14]. Platelets also participate in both innate and adaptive intravascular immune responses [15,16].

Low platelet concentration is called thrombocytopenia, and is due to either decreased production or increased destruction. Elevated platelet concentration is called thrombocytosis, and is either congenital, reactive (tocytokines), or due to unregulated production: one of the myeloproliferative neoplasms or certain other myeloidneoplasms. A disorder of platelet function is a thrombocytopathy. Normal platelets can respond to an abnormality on the vessel wall rather than to hemorrhage, resulting in inappropriate platelet adhesion/ activation and thrombosis: the formation of a clot within an intact vessel. This type of thrombosis arises by mechanisms different from those of a normal clot: namely, extending the fibrin of venous thrombosis; extending an unstable or ruptured arterial plaque, causing arterial thrombosis; and microcirculatory thrombosis. An arterial thrombus may partially obstruct blood flow, causing downstream ischemia, or may completely obstruct it, causing downstream tissue death.Thrombus formation on an intact endothelium is prevented by nitricoxide, prostacyclin and CD 39 [17-19].

Endothelial cells are attached to the sub endothelial collagen by von Willebrandfactor (VWF) which these cells produce. VWF is also stored in the Weibel Palade bodies of the endothelial cells and secreted constitutively into the blood. Platelets store VWF in their alpha granules. When the endothelial layer is disrupted, collagen and VWF anchor platelets to the subendothelium. Platelet GP1b IX V receptor binds with VWF; and GPVI receptor and integrin α2β1 bind with collagen [20].

Therefore, this study is aimed to investigate the role of coagulation parameters in Primary hypertensive subjects attending university of Abuja teaching hospital Abuja, Nigeria.

Materials and Methods

Study Area

This research population were drawn from a group of subjects with primary hypertensive cases who have been on medications for 6-12months period in University of Abuja Teaching Hospital, Nigeria's Federal Capital Territory. The subjects were between 18-60 years of age.

Ethical Approval

Ethical approval was gotten from University of Abuja Teaching Hospital (UATH) Ethical committee on September 6, 2019 with approval number of UATH/HREC/PR/2019/023.

Research Protocol

Using aseptic precaution, 4.5mls of venous blood were collected into a plastic tube containing 0.5ml of aqueous tri-sodium citrate and were separated at 4000rpm for 15 minutes to obtain citrated plasma for coagulation Activated Partial Thromboplastin Time (APTT) and Prothrombin Time (PT). 5mls of blood were collected into K3EDTA bottle for platelet count. Coagulometry method was used for quantitative measurement of Activated Partial Thromboplastin Time (APTT), Prothrombin Time (PT) and platelet counts were analysed using electronic impedance principle in haematology analyzer.

Statistical Analysis

The data obtained were analyzed by SPSS software version 16.

Results

The mean ± SD value of coagulation factors of the hypertensive subjects (Test): prothrombin time (PT):14.10±1.677, INR: 1.22±0.14, APTT: 36.39±6.25 were significantly higher compared with the apparently normal subjects: 10.86±0.71, 1.10±0.08, 24.86±2.83 respectively ($p<0.05$) while Platelets count: 248.75±52.45 was significantly lower compared to apparently normal subjects: 284.95±27.66 ($p<0.05$) as shown in table 1.

Table 1: Shows the Mean±SD value for some coagulation parameters in hypertensive and normotensive subjects.

Parameter	Control	Test	T	P
PT	10.86±0.71	14.10±1.68	14.37	0.000
INR	1.10±0.08	1.22±0.15	5.58	0.001
APTT	24.86±2.83	36.39±6.24	13.49	0.003
PLT	284.95±27.66	248.75±52.45	4.79	0.002

P< 0.05 is significant
P>0.05 is not significant

Using one-way ANOVA, the mean± SD value for control, male and female were compared to hypertensive male and female as shown in table 2. For PT the mean ±SD value for hypertensive male was 14.24±1.61, for hypertensive female was 13.96±1.75 while for control male and female was 10.86±0.73 and 11.00±0.00 respectively. For INR, the Mean±SD value for male and female hypertensive were 1.24±0.15 and1.21±0.15 respectively, while for control male and female were 1.10±0.08 and 1.12±0.00 respectively. for APTT, the mean ±SD values for male and female hypertensive was 37.72±5.88 and 35.32±5.69 respectively while for control male and female control were 24.77±2.82 and 26.50±3.54. For Platelets, the mean ±SD values for male and female hypertensive were 235.52±59.17 and 253.32±50.42 while for control male and female control were 285.03±24.53and 283.50±84.15 respectively. PT, APTT and INR were significantly increased when compared with the control ($p<0.05$) while platelets significantly decreased when compared with control.

Table 2: Shows the Mean±SD value for some coagulation parameters in hypertensive and normotensive subjects in different gender

Parameter	Male Control	Female Control	Male Hypertensive	Female Hypertensive	F	P
PT	10.86±0.73	11.00±0.00	14.24±1.61	13.96±1.75	39.34	0.000
INR	1.10±0.84	1.12±0.00	1.24±0.15	1.21±0.15	6.51	0.000
APTT	24.77±2.82	26.50±3.54	37.72±5.89	35.32±5.69	43.17	0.000
PLT	285.03±24.53	283.50±84.15	235.52±59.17	253.32±50.4	6.13	0.001

P< 0.05 is significant
P>0.05 is not significant

Using one-way ANOVA, Mean±SD values for hypertensive subjects in different age groups were analyzed for age groups of 31-40, 41-50, 51-60 and 61 above as shown in table 3. For PT, the Mean±SD for age group 31-40 was 14.16±1.60, for 41-50 was 14.31±2.21. For age group 51-60 was 14.22 ±1.88 and for 61 above was 13.79±0.71. There was no significant difference ($p>0.05$). For INR, the Mean±SD for age groups 31-40 was 1.23 ± 0.15, For 51-60 was 1.25±0.15 and for 61 above was 1.19±0.76. There was no significant difference ($p>0.05$). For APTT, the mean ±SD for 31-40 years was 38.47±7.48. For 41-50 years was 36.63 ± 5.79, for 51-60 years was 36.32 ±6.74, and for 61 above was 34.00 ±4.20. There was no significant difference ($P>0.05$). For PLT, the Mean±SD for 31-40 years was 256.79±54.27 and for 41-50 was 245.56 ±66.63. For 51-60 years was 241.14±51.54 and for 61 years above was 253.58±32.98. anao significant difference was observed too ($P>0.05$).

Table 3: Shows the Mean±SD value for some coagulation parameters in hypertensive subjects in different age groups

Parameter	31-40 years	41-50years	51-60years	61years above	F	P
PT	14.16±1.60	14.31±2.21	14.23±1.88	13.79±0.71	0.349	0.790
INR	1.22±0.15	1.22±0.19	1.25±0.15	1.19±0.76	0.433	0.730
APTT	38.47±7.48	36.63±5.79	36.32±6.74	34.00±4.20	1.66	0.180
PLT	256.79±54.27	245.56±66.63	241.14±51.54	253.58±32.98	0.38	0.770

P< 0.05 is significant
P>0.05 is not significant

Discussion

The PT and APTT in primary hypertensive subjects provide useful information about haemostatic functions and hypercoagulation. Platelets are essential parameters used in assessing thrombosis. This study shows that coagulation disorder is associates with hypertensive patients irrespective of the gender however, there is no significant on the age group.

The trend of PT, APTT and PLT observed in this study is comparable to the findings of Eledo et al., (2018) on Prothrombin Time, Activated Partial Thromboplastin Time and Platelets Count among Hypertensive Patients Attending a Tertiary Health Institution in Yenagoa, Nigeria. There was significant variation between the hypertensive and control candidates for each of the parameters. The variation suggests shift among the haemostatic parameters under study. Typically, a significant increase in both parameters suggests the tendency to cause an alteration in the haemostatic system. Dysfunctions of haemostatic system predispose the patients to atherosclerosis which a major risk factor of hypertension together with endothelial destruction and dysfunction, and hyper activation of the platelet[21].

Prothrombin time (PT) and activated partial thromboplastin time (APTT) have been shown to be associated with elevated systolic and diastolic blood pressures in hypertensive and normotensive patients[22]. In a study which involved the inclusion of one hundred and one patients with hypertension of mild to moderate grades, attending Al Najaf Teaching Hospital, Iraq, significant differences were found in PT and APTT between hypertensive and normotensive patients[23].

Conclusion

Our findings show that significant coagulation disorder is associated with primary hypertensive male and female patients irrespective of the age.

Recommendations

The need for early intervention on hypertensive subjects for appropriate management to avoid complications associated with coagulation and thrombosis is suggested. Platelets count, Prothrombin time (PT) and activated partial thromboplastin time (APTT) investigations should be used as prognostic indices for evaluating hypertensive patients for effective management.

To understand comphrehensive haemostatic system in hypertensive patients, we recommend further research in other coagulation factors not captured in this study.

References

1. Naish J, Court D S (2014) Medical sciences (2nd edition) 9: 562-563. https://books.google.co.in/books?id=K21_AwAAQBAJ&pg=PAPA562&redir_esc=y#v=onepage&q&f=false.
2. CDC (2015) High Blood Pressure Fact Sheet. Archived from the original on 6 March, 2016 http://www.cdc.gov/dhdsp/data_statistics/fact_sheets/fs_bloodpressure.htm.
3. Hernandorena I, Duron E, Vidal J S, Hanon O (2017) Treatment options and considerations for hypertensive patients to prevent dementia. Expert Opinion on Pharmacotherapy (Review) 18: 989-1000.
4. Poulter N R, Prabhakaran D, Caulfield M (2015) Hypertension. Lancet 386: 801-812.
5. Giuseppe M, Fagard R, Narkiewicz K, Redon J, Zanchetti A, et al. (2013) ESH/ESC Guidelines for the management of arterial hypertension:

The Task Force for the management of arterial hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). European Heart Journal 34: 2159-2219.

6. Fisher N D, Williams G H (2005) Hypertensive vascular disease. Harrison's Principles of Internal Medicine (16th edition). New York, McGraw-Hill 3: 1463-1481.
7. Marshall I J, Wolfe C D, McKevitt C (2012) Lay perspectives on hypertension and drug adherence: systematic review of qualitative research. British Medical Journal (Clinical Research Edition) 345: 39-53.
8. Perez M I, Musini V M, Wright J M (2008) Pharmacological interventions for hypertensive emergencies. The Cochrane Database of Systematic Reviews 4: 36-53.
9. Marik P E, Varon J (2007) Hypertensive crises: challenges and management, chest 131: 1949-1962.
10. Blumenthal J A, Babyak M A, Hinderliter A, Watkins L L, Craighead L, et al. (2010) Effects of the DASH diet alone and in combination with exercise and weight loss on blood pressure and cardiovascular biomarkers in men and women with high blood pressure: the ENCORE study. Archives of internal medicine 170: 126-135.
11. Laki K (1972) Our ancient heritage in blood clotting and some of its consequences. Annals of the New York Academy of Sciences 202: 297-307.
12. Machlus K R, Thon J N, Italiano J E (2014) Interpreting the developmental dance of the megakaryocyte: a review of the cellular and molecular processes mediating platelet formation. British Journal of Haematology 165: 227-236.
13. Yip J, Shen Y, Berndt M C, Andrews R K (2005) Primary platelet adhesion receptors. International Union Biochemistry and Molecular Biology 57: 103-108.
14. Berridge M J (2014) Module 11: Cell Stress, Inflammatory Responses and Cell Death. Cell Signaling Biology 14: 261-289.
15. Gaertner F, Massberg S (2016) Blood coagulation in immunothrombosis-At the frontline of intravascular immunity. Seminars in Immunology 28: 561-569.
16. Hampton T (2018) Platelets' Role in Adaptive Immunity May Contribute to Sepsis and Shock. Journal of the American Medical Association 319: 1311–1312.
17. Palmer R M, Ferrige A G, Moncada S (1987) Nitric oxide release accounts for the biological activity of endothelium-derived relaxing factor 327: 524-526.
18. Jones C I, Barrett N E, Moraes L A, Gibbins J M, Jackson D E (2012) Endogenous inhibitory mechanisms and the regulation of platelet function. Platelets and Megakaryocytes. Methods in Molecular Biology 788: 341-366.
19. Marcus A J, Broekman M J, Drosopoulos J H, Olson K E, Islam N, et al. (2005) Role of CD39 (NTPDase-1) in thromboregulation, cerebroprotection, and cardioprotection. Seminars in Thrombosis and Hemostasis 31: 234-246.
20. Dubois C, Panicot Dubois L, Merrill Skoloff G, Furie B, Furie B C (2006) Glycoprotein VI-dependent and -independent pathways of thrombus formation in vivo. Blood 107:3902-3906.
21. Eledo B O, Izah S C, Okamgba O C (2018) Prothrombin Time, Activated Partial Thromboplastin Time and Platelets Count among Hypertensive Patients Attending a Tertiary Health Institution in Yenagoa, Nigeria. American Journal of Blood Research 1:3.
22. Nnamani, Nnenna Adaeze, Uchenna Emeribe A, Abdullahi Nasiru I, Babayo A, et al. (2014) Evaluation of prothrombin time and activated partial thromboplastin time in hypertensive patients attending a tertiary hospital in calabar, Nigeria. Advances in hematology 932039.
23. Giacchetti G, Turchi F, Boscaro M, Ronconi V(2009) Management of primary aldosteronism: its complications and their outcomes after treatment. Current Vascular Pharmacology 7: 244-249.