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Some Biochemical and Haematological Effects of Hypertention Among Pregnant Women of Ikwano L.G.A., Abia State, Nigeria.

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ABSTRACT

Liver enzymes and haemoglobin were estimated in the 10 hypertensive and 20 non-hypertensive pregnant women who attended antenatal clinic in Ikwuano L.G.A. of Abia satate Nigeria.Hypertension is a silent killer because many a times it does not give signal to the patient.Hypertension is so common to pregnant women and affect many systems.The study showed significant difference($P<0.05$) in liver enzymes.There was no significant change ($P>0.05$) in the haemoglobin level among the subjects.

Keywords: Hypertensive women, non-hypertensive women, liver enzyme and haemoglobin.

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INTRODUCTION

Hypertension (HTN) or high blood pressure, sometimes called arterial hypertension, is a chronic medical condition in which the blood pressure in the arteries is elevated.[Chobanian et al.,2003] Blood pressure is summarised by two measurements, systolic and diastolic, which depend on whether the heart muscle is contracting (systole) or relaxed between beats (diastole) and equate to a maximum and minimum pressure, respectively. Normal blood pressure at rest is within the range of 100-140mmHg systolic (top reading) and 60-90mmHg diastolic (bottom reading). High blood pressure is said to be present if it is persistently at or above 140/90 mmHg.

Hypertension is classified as either primary (essential) hypertension or secondary hypertension; about 90–95% of cases are categorized as "primary hypertension" which means high blood pressure with no obvious underlying medical cause.[Fisher and Williams,2005] The remaining 5–10% of cases (secondary hypertension) are caused by other conditions that affect the kidneys, arteries, heart or endocrine system.

Hypertension puts persistent strain on the heart, leading to hypertensive heart disease and coronary artery disease if untreated. Hypertension is also a major risk factor for stroke, aneurysms of the arteries , peripheral arterial disease and is a cause of chronic kidney disease. Even moderate elevation of arterial blood pressure is associated with a shortened life expectancy. Dietary and lifestyle changes can improve blood pressure control and decrease the risk of associated health complications, although drug treatment is often necessary in people for whom lifestyle changes are not enough or not effective.

Hypertension is rarely accompanied by any symptoms, and its identification is usually through screening, or when seeking healthcare for an unrelated problem. A proportion of people with high blood pressure report headaches back of the head, as well as lightheadedness, vertigo, tinnitus (buzzing or hissing in the ears), altered vision or fainting episodes.[Marshall et al.,2012] These symptoms, however, might be related to associated anxiety rather than the high blood pressure itself.[Wong and Mitchel.,2007]

On physical examination, hypertension may be suspected on the basis of the presence of hypertensive retinopathy detected by examination of the optic fundus found in the back of the eye using ophthalmoscopy.[O Brien et al.,2007] Classically, the severity of the hypertensive retinopathy changes is graded from grade I–IV, although the milder types may be difficult to distinguish from each other. Ophthalmoscopy findings may also give some indication as to how long a person has been hypertensive.

Some additional signs and symptoms may suggest secondary hypertension, that is hypertension due to an identifiable cause such as kidney diseases or endocrine diseases. For example, truncal obesity, glucose intolerance, moon face, a "buffalo hump" and purple stretch marks suggest Cushing's syndrome. Thyroid disease and acromegaly can also cause

hypertension and have characteristic symptoms and signs.[Papadopoulos et al.,2010] An abdominal bruit may be an indicator of renal artery stenosis , while decreased blood pressure in the lower extremities and/or delayed or absent femoral arterial pulses may indicate aortic coarctation . Labile or paroxysmal hypertension accompanied by headache, palpitations, pallor, and perspiration should prompt suspicions of pheochromocytoma.

Severely elevated blood pressure (equal to or greater than a systolic 180 or diastolic of 110 — sometimes termed malignant or accelerated hypertension) is referred to as a "hypertensive crisis", as blood pressures above these levels are known to confer a high risk of complications. People with blood pressures in this range may have no symptoms, but are more likely to report headaches (22% of cases) and dizziness than the general population. Other symptoms accompanying a hypertensive crisis may include visual deterioration or breathlessness due to heart failure or a general feeling of malaise due to renal failure. Most people with a hypertensive crisis are known to have elevated blood pressure, but additional triggers may have led to a sudden rise.[Gibson,2009]

A "hypertensive emergency", previously is diagnosed when there is evidence of direct damage to one or more organs as a result of the severely elevated blood pressure. This may include hypertensive encephalopathy, caused by brain swelling and dysfunction, and characterized by headaches and an altered level of consciousness . Retinal papilloedema and/or fundal hemorrhages and exudates are another sign of target organ damage. Chest pain may indicate heart muscle damage or sometimes aortic dissection, the tearing of the inner wall of the aorta. Breathlessness, cough, and the expectoration of blood-stained sputum are characteristic signs of pulmonary edema, the swelling of lung tissue due to left ventricular failure an inability of the left ventricle of the heart to adequately pump blood from the lungs into the arterial system. Rapid deterioration of kidney function and microangiopathic hemolytic anemia may also occur. In these situations, rapid reduction of the blood pressure is mandated to stop ongoing organ damage. In contrast there is no evidence that blood pressure needs to be lowered rapidly in hypertensive urgencies where there is no evidence of target organ damage and over aggressive reduction of blood pressure is not without risks. Use of oral medications to lower the BP gradually over 24 to 48h is advocated in hypertensive urgencies.

Hypertension occurs in approximately 8–10% of pregnancies. Two blood pressure measurements six hours an apart of greater than 140/90 mm Hg is considered diagnostic of hypertension in pregnancy.[Rodriguez et al.,2010] Most women with hypertension in pregnancy have pre-existing primary hypertension, but high blood pressure in pregnancy may be the first sign of pre-eclampsia, a serious condition of the second half of pregnancy and puerperium. Pre-eclampsia is characterised by increased blood pressure and the presence of protein in the urine. It occurs in about 5% of pregnancies and is responsible for approximately 16% of all maternal deaths globally. Pre-eclampsia also doubles the risk of perinatal mortality. Usually there are no symptoms in pre-eclampsia and it is detected by routine screening. When symptoms of pre-eclampsia occur the most common are headache, visual disturbance , vomiting, epigastric pain, and edema. Pre-eclampsia can occasionally progress to a life-threatening condition called eclampsia, which is a hypertensive emergency and has several

serious complications including vision loss, cerebral edema, seizures or convulsions, renal failure, pulmonary edema, and disseminated intravascular coagulation .[Dionne et al (2012)]

Primary (essential) hypertension is the most common form of hypertension, accounting for 90–95% of all cases of hypertension. In almost all contemporary societies, blood pressure rises with aging and the risk of becoming hypertensive in later life is considerable.[Lifton(2001] Hypertension results from a complex interaction of genes and environmental factors. Numerous common genetic variants with small effects on blood pressure have been identified[He and MacGregor.,2009] as well as some rare genetic variants with large effects on blood pressure[He et al.,2013] but the genetic basis of hypertension is still poorly understood. Several environmental factors influence blood pressure. Lifestyle factors that lower blood pressure include reduced dietary salt intake,[Haslam and James,2005] increased consumption of fruits and low fat products , exercise,[Whelton et al,2002] weight loss[] and reduced alcohol intake.[Ospina et al,2007] Stress appears to play a minor role with specific relaxation techniques not supported by the evidence[Mesa et al,2011;Vaidya and Forman,2010. The possible role of other factors such as caffeine consumption,[Sorof and Daniel,2002] and vitamin D deficiency[Lawlor and Smith,2005] are less clear cut. Insulin resistance, is also thought to contribute to hypertension.[Dluhy and Williams,1998] Recent studies have also implicated events in early life (for example low birth weight, maternal smoking and lack of breast feeding) as risk factors for adult essential hypertension,[Grossman and Messerli,2012] although the mechanisms linking these exposures to adult hypertension remain obscure.

Secondary hypertension results from an identifiable cause. Renal disease is the most common secondary cause of hypertension. Hypertension can also be caused by endocrine conditions, such as Cushing's syndrome, hyperthyroidism, hypothyroidism, acromegaly, Conn's syndrome or hyperaldosteronism, hyperparathyroidism and pheochromocytoma.[Conway,1982] Other causes of secondary hypertension include obesity, sleep apnea, pregnancy, coarctation of the aorta, excessive liquorice consumption and certain prescription medicines, herbal remedies and illegal drugs.[Palatin and Julius,2008]

In most people with established essential (primary) hypertension, increased resistance to blot accounting for the high pressure while cardiac output remains normal.[Anderson et al,2004] There is evidence that some younger people with prehypertension or 'borderline hypertension' have high cardiac output, an elevated heart rate and normal peripheral resistance, termed hyperkinetic borderline hypertension.[Folkow,1982] These individuals develop the typical features of established essential hypertension in later life as their cardiac output falls and peripheral resistance rises with age. Whether this pattern is typical of all people who ultimately develop hypertension is disputed.[Struijker et al,1992] The increased peripheral resistance in established hypertension is mainly attributable to structural narrowing of small arteries and arterioles,[Safar and London,1987] although a reduction in the number or density of capillaries may also contribute.[Schiffrin,1992] Hypertension is also associated with decreased peripheral venous compliance[Chobanian,2007] which may increase venous return, increase cardiac preload and, ultimately, cause diastolic dysfunction. Whether increased active vasoconstriction plays a role in established essential hypertension is unclear.

Pulse pressure is frequently increased in older people with hypertension. This can mean that systolic pressure is abnormally high, but diastolic pressure may be normal or low — a condition termed isolated systolic hypertension.[Navar,2010] The high pulse pressure in elderly people with hypertension or isolated systolic hypertension is explained by increased arterial stiffness, which typically accompanies aging and may be exacerbated by high blood pressure.[Esler et al,2010]

Many mechanisms have been proposed to account for the rise in peripheral resistance in hypertension. Most evidence implicates either disturbances in renal salt and water handling [Versari et al,2009] and/or abnormalities of the sympathetic nervous system.[Marchesi et al,2008] These mechanisms are not mutually exclusive and it is likely that both contribute to some extent in most cases of essential hypertension. It has also been suggested that endothelial dysfunction and vascular inflammation may also contribute to increased peripheral resistance and vascular damage in hypertension[Loscalzo et al,2008;Padwal et al,2009]

Much of the disease burden of high blood pressure is experienced by people who are not labelled as hypertensive. Consequently, population strategies are required to reduce the consequences of high blood pressure and reduce the need for antihypertensive drug therapy. Lifestyle changes are recommended to lower blood pressure, before starting drug therapy. The 2004 British Hypertension Society guidelines proposed the following lifestyle changes consistent with those outlined by the US National High BP Education Program in 2002[Siebenhofer et al,2011] for the primary prevention of hypertension:

- maintain normal body weight for adults (e.g. body mass index 20–25 kg/m^2)
- reduce dietary sodium intake to <100 mmol/ day (<6 g of sodium chloride or <2.4 g of sodium per day)
- engage in regular aerobic physical activity such as brisk walking (≥30 min per day, most days of the week)
- limit alcohol consumption to no more than 3 units/day in men and no more than 2 units/day in women
- consume a diet rich in fruit and vegetables (e.g. at least five portions per day);

Effective lifestyle modification may lower blood pressure as much an individual antihypertensive drug. Combinations of two or more lifestyle modifications can achieve even better results.

As of 2000, nearly one billion people or ~26% of the adult population of the world had hypertension.[Burt et al,2011] It was common in both developed (333 million) and undeveloped (639 million) countries. However rates vary markedly in different regions with rates as low as 3.4% (men) and 6.8% (women) in rural India and as high as 68.9% (men) and 72.5% (women) in Poland. In Europe hypertension occurs in about 30-45% of people as of 2013.[Burt et al,1995]

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Hypertension is more prevalent in men and in those of low socioeconomic status.

MATERIAL AND METHODS

Study Area: Ikwano,Abia State,Nigeria.

Subjects:10 Hypertensive and 20 non-hypertensive pregnant women attending antenatal clinic in Ikwuano L.G.A.Health Centres.

Sample Collection: Venous blood samples were collected from hypertensive and non-hypertensive women(control) into EDTA anti-coagulated containers for haemoglobin estimation and the other transferered into plain tubes for serum.

Grouping: I is the non-hypertensive group and II is the hypertensive group.

Ethics: Oral consents were made to the subjects prior to sample collection.

Statistical Analysis: The data were analysed using t-test and level of significance set at $P<0.05$.

RESULTS

Table1:Serum Alanine Aminotransferase In Non-Hypertensive Pregnant And Hypertensive Pregnant Women

Group	Mean	±SD	P-Level
I	8.55	6.75	
II	10.84	7.36	P<0.05

Table 2:Serum Aspartate Aminotransferase(Ast) Levels In Hypertensive Non-Hypertensive And Hypertensive Pregnant Women

Group	Mean	±SD	P-Level
I	17.00	10.01	
II	22.61	7.34	P<0.05

Table 3:Serum Alanine Phosphatase In Non-Hypertensive And Hypertensive Pregnant Women

Group	Mean	±SD	P-Level
I	111.00	15.12	
II	150.21	16.63	P<0.05

Table 4:Total Serum Bilirubin In Non-Hypertensive And Hypertensive Pregnant Women

Group	Mean	±SD	P-Level
I	23.86	12.23	
II	27.71	11.56	P<0.05

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Table 5:Total Serum Cholesterol Levels In Non-Hypertensive And Hypertensive Pregnant Women

Group	Mean	±SD	P-Level
I	11.12	5.40	
II	16.67	8.05	P<0.05

Table 6:Mean Body Weight Of Non-Hypertensive And Hypertensive Pregnant Women

Group	Mean(Kg)	±SD	P-Level
I	61.70	8.51	
II	70.00	13.45	P<0.05

Table 7:Haemoglobin Level In Non-Hypertensive And Hypertensive Pregnant Women

Group	Mean(g/dl)	±SD	P-Level
I	11.45	2.10	
II	12.71	1.30	P>0.05

DISCUSSSION

In this study,liver enzymes and haemoglobin tests were carried out to determine if there was any significant damage on the liver and to know how the enzyme activities were affected and from results in table 1,it was found that may be as a result of the inflammation of the hepatic cells caused by hypertension resulted in slight elevation in the alanine aminotransferase(ALT) which was seen in group ii.This significant increase(P<0.05) may be as a result of cell death,because when the cells are damaged,ALT leaks into the blood stream leading to a rise in the serum enzyme levels.Since ALT is the most sensitive marker for the liver cells damage and can be dictated by its elevation in the blood.The result showed that the mean serum level of ALT though slightly elevated but not above the normal value,both groups fal within normal value of 3-15 iu/l(P<0.05).This result collaborate with the report of Haber(1995),which wrote that ALT levels can be normal in the presence of liver disease.

The result obtained in table 2 showed significant difference(P<0.05) when the mean AST values were compared relative to the non-hypertensive pregnant women.Transaminases are elevated in some diseases such as myocardial infarction,hypertension,infectious hepatitis or other damages either to the heart of liver (Ohaeri,2001).In group ii,there was an increase in the level of AST of hypertensive pregnant patients beyond the normal range.The rise in the AST level can be attributed to paranchymal liver cell breakdown as a result of increase in the basal metabolic rate of the women.This result showed significant difference(P<0.05).

However,in this investigation on ALP,which is said to be useful in the diagnosis of hepatic ciliary's disease,ALP has optimal activity when PH is in the neighbouhood of 9.8.They act on large variety of physiological and and non-physiological substrates.Results in table 3 showed significant differnce9P<0.05).Alkaline phosphatase level increases progressively during normal pregnancy.While the increase in group ii may be due to inflammation of the biliary tract cells but still not specific to biliary tract because,this enzyme is equally found in bone where

increase in ALP level may result in osteoblastic bone tumour and also disease such as hepatobiliary disease,cholangitis,and a double increase in ALP level may result in such disease like,hepatocellular jaundice and cholestatic jaundice(Friedinan et al,1996).

Renal or intestinal damage can also lead to a rise in ALP.ALP may also be elevated in primary biliarr cirrhosis,hepatitis,hypertension,gallstone etc.They catalyse a group of hydrolysis of phosphate esters in the alkaline environments generating organic radical and inorganic phosphate.

The result obtained in table 5 showed that the mean serum cholesterol levels for group i and ii when compared had significant difference($P<0.05$).The increase in serum cholesterol level in group ii may be as result of atherosclerosis of the coronary such as hypertension and other arteries and circulating levels of lipoprotein.It can also be a result of environmental and dietary factors which may detremine the expression of the increase in cholesterol level.The result in table 6 showed that the bilirubin level in group ii was slightly increased than in group i and this increase may be as a result of the increase in the bilirubin load as a result of haemolysis.It can also be as a result of impaired binding of bilirubin to ligandin or impaired conjugation with glucuronate in the liver.

Table 7 showed no significant change in haemoglobin among the subjects($P<0.05$).

CONCLUSION

The study showed that liver enzymes are seriously affected in the hypertensive pregnant women as well as total cholesterol levels but no much effect on the haemoglobin levels.More care and monitoring is needed in women who are pregnant becuase they are easily exposed to high blood pressure and proteinuria.

REFERENCES

[1] Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo Jr. JL, Jones DW, Materson BJ, Oparil S, Wright Jr. JT, Roccella EJ (2003). "Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure". Hypertension (Joint National Committee On Prevention) 42 (6): 1206–52.

[2] Fisher ND, Williams GH (2005). "Hypertensive vascular disease". In Kasper DL, Braunwald E, Fauci AS, et al.. Harrison's Principles of Internal Medicine (16th ed.). New York, NY: McGraw-Hill. pp. 1463–81.

[3] Marshall, IJ; Wolfe, CD; McKevitt, C (2012). "Lay perspectives on hypertension and drug adherence: systematic review of qualitative research". BMJ (Clinical research ed.) 345: e3953.

[4] Wong T, Mitchell P (2007). "The eye in hypertension". Lancet 369 (9559): 425–35.

[5] O'Brien, Eoin; Beevers, D. G.; Lip, Gregory Y. H. (2007). ABC of hypertension. London: BMJ Books.

[6] Papadopoulos DP, Mourouzis I, Thomopoulos C, Makris T, Papademetriou V. Blood Press 2010;19(6): 328–36.
[7] Gibson Paul. 2009, Hpertension and Pregnancy. eMedicine Obstetrics and Gynecology. Medscape.
[8] Rodriguez-Cruz, Edwin; Ettinger, Leigh M. 2010, Hypertension. eMedicine Pediatrics: Cardiac Disease and Critical Care Medicine. Medscape.
[9] Dionne JM, Abitbol CL, Flynn JT. Pediatr Nephrol 2012;27(1):17–32..
[10] Lifton RP, Gharavi AG, Geller DS. Cell 2011;104(4):545.
[11] He FJ, MacGregor GA. J Hum Hyp 2009;23(6):363–84.
[12] He FJ, Li J, Macgregor GA. BMJ (Clinical research ed.) 2013:346.
[13] Dickinson HO, Mason JM, Nicolson DJ et al. J Hypertens 2006;24(2).
[14] Haslam DW, James WP. Lancet 2005;366:9492.
[15] Whelton PK, He J, Appel LJ, Cutler JA, Havas S, Kotchen TA et al. JAMA 2002;288(15):1882–8.
[16] Dickinson HO, et al. J Hypert 2006;24(2):215–33.
[17] Ospina MB, Bond K, Karkhaneh M et al. Evid Rep Technol Assess (Full Rep) 2007;155: 1–263.
[18] Mesas AE, Leon-Muñoz LM, Rodriguez-Artalejo F, Lopez-Garcia E. The American J Clin Nutr 2011;94(4):1113–26.
[19] Vaidya A, Forman JP. Hypertension 2010;56(5):774–9.
[20] Sorof J, Daniels S. Hypertension 2002;40(4): 441–447..
[21] Lawlor DA, Smith GD. Curr Opinion Nephrol Hypert 2005;14(3): 259–64
[22] Dluhy RG, Williams GH eds. 1998, Endocrine hypertension. In Wilson JD, Foster DW, Kronenberg HM. Williams textbook of endocrinology (9th ed. ed.). Philadelphia ; Montreal: W.B. Saunders. pp. 729–49.
[23] Grossman E, Messerli FH. Am J Med 2012;125(1):14–22.
[24] Conway J. Physiol Rev 1984;64 (2): 617–60.
[25] Palatini P, Julius S. Curr Hypertens Rep 2009;11(3):199–205.
[26] Andersson OK, Lingman M, Himmelmann A, Sivertsson R, Widgren BR. Blood Press 2004; 13(6):350–4.
[27] Folkow B. Physiol Rev 1982;62(2):347–504.
[28] Struijker Boudier HA, et al. J Hypertens 1982;Suppl 10 (7): S147–56.
[29] Safar ME, London GM. Hypertension 1987;10(2):133–9.
[30] Schiffrin EL. Hypertension 1992;19 (2 Suppl): II1–9.
[31] Chobanian AV. N Engl J Med 2007;357 (8): 789–96.
[32] Zieman SJ, Melenovsky V, Kass DA. Arterioscler Thromb Vasc Biol 2005;25 (5): 932–43.
[33] Navar LG. J Appl Physiol 2010; 109 (6): 1998–2000.
[34] Esler M, Lambert E, Schlaich M. J Appl Physiol 2010;109 (6): 1996–8.
[35] Versari D, Daghini E, Virdis A, Ghiadoni L, Taddei S. Br J Pharmacol 2009;157(4):527–36.
[36] Marchesi C, Paradis P, Schiffrin EL. Trends Pharmacol Sci 2008;29(7): 367–74.
[37] Loscalzo Joseph, et al. 2008, Harrison's principles of internal medicine. McGraw-Hill Medical.
[38] Padwal RS, Hemmelgarn BR, Khan NA et al. Canadian J Cardiol 2009;25 (5): 279–86.
[39] Siebenhofer A, et al Cochrane Database of Systematic Reviews 9(9):CD008274.

[40] Burt VL, Whelton P, Roccella EJ et al. Hypertension 1995;25 (3): 305–13.
[41] Burt VL, Cutler JA, Higgins M et al. Hypertension 1995;26 (1): 60–9.
[42] Haber MM, West AB, Haber AD, Reuben. Cam J Gastroenterol 1995;90:1250-1257.
[43] Ohaeri OC, Umokoro SE. JOMP 2001; I2:54-57.
[44] Friedman LS, Martin P, and Munoz J. 1996, Liver Function Test and Objective Evaluation of the Patients with Liver Disease.In Hepatology:a Textbook of Liver Disease. WB. Saunders Company.pp791-801.