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The Prevalence of the Metabolic Syndrome Among Ghanaian Pregnancy-Induced Hypertensive Patients Using the World Health Organisation and the National Cholesterol Education Program III Criteria

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The aim of this study was to determine the risk factors among Ghanaian women with Pregnancy-Induced Hypertension (PIH). Thirty women with preeclampsia, seventy with gestational hypertension and fifty normotensive pregnant women (controls) in the second half of pregnancy were recruited for this study. There was a significant increase in the prevalence of metabolic syndrome among the PIH subjects as compared to the normotensive pregnant women (controls) using the National Cholesterol Education Program (NCEPIII) and World Health Organisation criteria. Ghanaian women presenting with PIH are very prone to the development of the metabolic syndrome, thus the indices must be screened for during antenatal care.

Key words: Pre-eclampsia, gestational hypertension, syndrome X, high blood pressure, anthropometric data

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INTRODUCTION

Throughout the world 58,500 women die every year as a result of Pregnancy and childbirth and over 98% of all maternal mortality occurs in developing countries (Abouzahr and Royston, 1991; Bates *et al*., 2008). The most common cause of these maternal deaths are complications of pregnancy and child birth such as haemorrhage, sepsis, complications of unsafe abortions, hypertension disorders of pregnancy and obstructed labour (WHO, 1994). In developing countries, 17% of direct obstetric deaths are as a result of hypertension (Maine, 1987). Maternal and perinatal morbidity and mortality are also major health problems in developing countries like Ghana. Studies by Osei-Nketiah (2001) has shown that in Ghana, 40% of maternal deaths are as a result of hypertensive pregnancy, antepartum haemorrhage and post partum haemorrhage.

Pregnancy-Induced Hypertension (PIH) is hypertension that develops as a consequence of pregnancy and regresses after delivery. Pregnancy-induced hypertension can be differentiated from chronic hypertension, which appears before 20 weeks gestation or continues for more than six weeks after delivery usually (ACOG, 1996). Preeclampsia, which is a type of pregnancy-induced hypertension characterized by progressive hypertension and pathological oedema, is clinically defined as blood pressure greater than 140/90 mmHg after 20 weeks gestation coexisting with proteinuria (300 mg/24 h or greater than 1+protein on a dipstick sample of urine collected at random) (Davey and MacGillivray, 1988). Gestational hypertension which is another form of PIH is characterized by the presence of blood pressure of 140/90 mmHg or higher on at least two occasions, 6 h apart, after 20 weeks of gestation (Forest *et al*., 2005).

Despite advances in perinatal care, hypertensive disorders of pregnancy remain a major cause of maternal and fetal morbidity, occurring in 5-10% of all pregnancies (Lindheimer and Katz, 1985). Pregnancy-Induced Hypertension complicates 5-10% pregnancies in the United States and is a major cause of maternal, fetal and neonatal morbidity and mortality (Seely and Solomon, 2003). In Ghana, prevalence of preeclampsia amongst Ghanaian women has been shown to be 7.03% (Obed and Patience, 2006).

Several studies have shown that PIH-including gestational hypertension and preeclampsia is associated with increased prevalence of markers of metabolic syndrome or syndrome X. The coining of the term syndrome X renewed the drive to conduct research concerning this syndrome (Reaven, 1988). In his description of syndrome X, Reaven (1988) considered the following abnormalities: resistance to insulin-stimulated glucose uptake, glucose intolerance, hyperinsulinemia, increased VLDL triglycerides, decreased HDL cholesterol and hypertension. Other metabolic abnormalities that have been considered as part of the syndrome include abnormal weight or weight distribution, inflammation, microalbuminuria, hyperuricemia and abnormalities of fibrinolysis and of coagulation (Meigs, 2000). Varying degrees of insulin resistance are present in overweight and obesity and closely related to type 2 diabetes (DeFronzo and Ferrannini, 1991). Insulin resistance is associated with increased morbidity and mortality when accompanied by other cardiovascular risk factors such as abnormal glucose tolerance, hypertension, hyperlipidemia, or obesity (Isomaa *et al*., 2001; Lakka *et al*., 2002), components of the insulin resistance syndrome or the metabolic syndrome (DeFronzo and Ferrannini, 1991). The prevalence of the metabolic syndrome increases with increasing age and Body Mass Index (BMI) (Park *et al*., 2003). Overweight and diabetes have increased dramatically worldwide during the last decade (Young and Nestle, 2002; Kengne *et al*., 2005) probably due to a more sedentary lifestyle combined with a relative increase in high-energy food intake.

Women in whom PIH develop, enter into pregnancy overweight or obese and also demonstrate, during pregnancy, some risk factors characterizing atherosclerosis, such as dyslipidemia (Belo *et al*., 2002; Sattar *et al*., 1997) insulin resistance (Kaaja *et al*., 1999; Seely and Solomon, 2003) and endothelial dysfunction (Roberts, 1998). These metabolic anomalies (increased adiposity, hyperlipidemia, hyperglycemia and elevated blood pressure) are suggestive of the metabolic syndrome. However geographical, social, economic and racial differences are thought to be responsible for incidence rates up to 3 times higher in some populations (Lopez-Jaramillo *et al*., 2001; WHO, 1988). In some countries such as Columbia it is the main cause of maternal mortality. Up to 42% of maternal deaths are attributed to this disorder in Colombia (Lopez-Jaramillo *et al*., 2001). Because of the increased risk for morbidity and mortality associated with the metabolic syndrome, an understanding of the dimensions of this syndrome is critical both for allocating health care and research resources and for other purposes (Ford and Giles, 2003).

Obed and Patience (2006) have previously reported the prevalence of preeclampsia among Ghanaian women to be as substantial as 7.03%. In Ghana today, efforts are being made to reduce both maternal and neonatal mortality. There is increasing evidence that women who develop PIH also present with a syndrome similar to the metabolic syndrome and because of the increased risk of

morbidity and mortality associated with the metabolic syndrome there is, therefore, the need for assessment in this direction. The objective of the current study was, therefore, to determine if the metabolic syndrome is a risk factor among Ghanaian women with pregnancy-induced hypertension.

MATERIALS AND METHODS

Subjects: Between November, 2006 and December, 2007 two groups of age matched women, comprising of one hundred pregnant women with pregnancy-induced hypertension (seventy with gestational hypertension and thirty with preeclampsia) and fifty normotensive pregnant women visiting the Obstetrics and Gynaecology Department of the Komfo Anokye Teaching Hospital in Kumasi, Ashanti Region of Ghana were recruited for this study. All the subjects were of Ghanaian origin, their participation voluntary and informed consent was obtained from each of them. Those within the age range 17-45 years and after 20 weeks of gestation and with blood pressure 140/90 mmHg and above with or without proteinuria were considered, the diagnosis of hypertensive complications during pregnancy was assessed by a single qualified obstetrician and gynaecologist using the diagnostic criteria of the National High Blood Pressure Education Program Working Group. Briefly, the presence of high blood pressure on two occasions 6 h apart was considered gestational hypertension while pregnant women who had proteinuria level of 2+positive result on a dipstick, were considered as preeclampsia (Forest *et al*., 2005). Women with known renal diseases, diabetes, cardiovascular diseases and hypertension prior to pregnancy were excluded from this study (both test and control). Pregnant women with normal blood pressure and without proteinuria were included in the control group. The study was approved by the local Committee on Human Research Publication and Ethics (CHRPE/KNUST/KATH/15_03_08).

Sample collection and preparation

Biochemical analysis: Venous blood samples were collected after an overnight fast (12-16 h). About 5 mL of venous blood was collected and dispensed into fluoride oxalate tubes and vacutainer® plain tubes for separation into plasma and serum, respectively. This was then taken to the laboratory and centrifuged; the plasma was used for the assay of glucose and the serum for other biochemical assay. Serum biochemistry was performed with an ATAC® 8000 Random Access Chemistry System (Elan Diagnostics, Smithfied, RI, USA). Parameters that were determined include: Fasting Blood Glucose (FBS), Triglyceride (TG), High Density Lipoprotein (HDL), Creatinine Kinase (CK), Lactate Dehydrogenase (LDH) and Aspartate Transferase (AST). The methods adopted by the automated instrument for the determination of the above parameters are according to the reagent manufacturer's instruction - JAS™ diagnostics, Inc. (JAS Diagnostics, Inc. Miami Florida, USA).

Enzyme-linked immunoassay for cardiac specific troponin-I: Troponin-I (Tn I) was analyzed using Enzyme–linked Immunoassay for cardiac specific Troponin-I using Ameritek USA assay kit. Assays were carried out as described by the manufacturer. This assay is based on the principle of solid phase enzyme- linked immunosorbent assay, USA Center for Disease Control/ National Institute of Health Manual, Biosafety in Microbiological and Biomedical Laboratories' (1984). Briefly, the desired coated wells were secured into the holder. The standard reference was diluted by using the reference standard dilution buffer to 2.0, 10, 20 and 50 ng mL^{-1}. Fifty microliters of negative control, each standards and specimens into appropriate wells. This was then incubated at room temperature (18-25°C) for about 30 min. The incubation mixture was removed by flicking plate contents into a waste container. The microtiter well was then rinsed and flicked 5 times with wash buffer. The wells were stroked sharply onto absorbent paper to remove all residual water droplets. One Hundred microliters of enzyme conjugate was also dispensed into appropriate well and the mixture was incubated for 30 min at room temperature (18-25°C). The washing of the microtiter wells was again repeated. One Hundred microliters of TMB reagent was dispensed into appropriate well and incubated at room temperature for about 15 min. The reaction was stopped by adding 100 μL of stop solution (2N HCl) to each well. This was gently mixed for 30 sec; it was ensured that all blue colour changed to yellow. Optical density was read at 450 nm with an ELx800™ Microplate Reader (Bio-Tek Instrument, Winooski, VT, USA) and results calculated from the standard curve using GraphPad Prism version 5.00 for windows (GraphPad software, San Diego California USA, www.graphpad.com).

Urinalysis: Early morning urine was collected in plastic containers from the respondents and urine protein was analyzed using the dip-stick qualitative method (CYBOW™ DFI Co. Ltd., Gimhae-City, Republic of Korea).

Anthropometric variables

Measurements: Anthropometric measurements included height to the nearest meter without shoes and weight to nearest 0.1 kg in light clothing. Subjects were weighed on a bathroom scale (Zhongshan Camry Electronic Co. Ltd,

Guangdong, China) and their height measured with a wall-mounted ruler. BMI was calculated by dividing weight (kg) by height squared (m^2). Waist circumference was measured with a Gulick II spring-loaded measuring tape (Gay Mills, WI) midway between the inferior angle of the ribs and the superior iliac crest, whereas hip circumference was measured at the outermost points of the greater trochanters. WHR and WHtR were recorded to the nearest 2 decimal places.

Blood pressure: Blood pressure was taken by trained personnel using a mercury sphygmomanometer and stethoscope. Measurements were taken from the left upper arm after subjects had been sitting for >5 min in accordance with the recommendation of the American Heart Association (Kirkendall *et al*., 1967). Duplicate measurements were taken with a 5 min rest interval between measurements and the mean value was recorded to the nearest 2.0 mmHg.

Metabolic syndrome: Because no single definition for the metabolic syndrome has been accepted worldwide and to give the possibility for comparison with the majority of studies on the same topic, we applied two widely used criteria to examine the prevalence of the metabolic syndrome: the NCEP III panel and the WHO. The various definitions are listed below.

According to the NCEP III criteria (NCEP, 2001), a subject has the metabolic syndrome if she has three or more of the following criteria:

- Abdominal obesity: ≥88 cm
- Hypertriglyceridemia: ≥1.7 mmol L^{-1}
- Low HDL cholesterol: <1.3 mmol L^{-1}
- High blood pressure: ≥130/85 mmHg
- High fasting glucose: ≥110 mg dL^{-1} (6.1 mmol L^{-1})

According to the WHO criteria (Alberti and Zimmet, 1998) a subject has the metabolic syndrome if she has diabetes plus two or more of the following abnormalities:

- High blood pressure: ≥ 40/90 mmHg
- Hyperlipidemia: Triglyceride concentration ≥2.00 mmol L^{-1}) and HDL ≥1.00 mmol L^{-1})
- Central obesity: Waist circumference of ≥80 cm

Statistical analysis: Continuous variables are expressed as their Mean±SEM. Comparisons of the women with PIH (gestational hypertension and preeclampsia separately and combined) against the control group were performed using unpaired t-tests, χ^2-tests, or Fisher exact tests where appropriate. Odds ratio and their 95% confidence intervals were used to quantify the risk of women with PIH in comparison with controls. A level of $p<0.05$ was acceptable as statistically significant. GraphPad Prism version 5.00 for windows was used for statistical analysis (GraphPad software, San Diego California USA, www.graphpad.com).

RESULTS

From this study, the mean age of the PIH subjects is 31.81±0.60 (30.37±1.29 for the preeclamptic patients and 32.43±0.65 for the gestational hypertensive patients) and 30.22±0.57 for the normotensive control group. Demographics, metabolic and cardiovascular variables are shown in Table 1. With the exception of Hip Circumference (HC) and Waist-to-Hip Ratio (WHR) which

Table 1: Demographics and metabolic and cardiovascular variables in each of the studied group

Parameters	GH	p-value	PE	p-value	PIH (GH+PE)	p-value	CG
Age (years)	32.43±0.65	0.0170	30.37±1.29	0.2840	31.81±0.60	0.6834	30.22±0.57
GP (weeks)	29.43±0.72	0.1609	30.67±0.96	0.8021	30.40±0.83	0.9630	31.00±0.85
Weight (kg)	74.19±2.06	0.0436	76.43±2.75	0.0069	74.86±1.65	0.0194	68.86±1.32
Height (m)	1.60±0.01	0.9052	1.59±0.01	0.7533	1.59±0.01	0.7754	1.60±0.01
BMI (kg m^{-2})	29.23±0.79	0.0323	30.11±0.91	0.0016	29.49±0.61	0.0100	27.05±0.48
WC (cm)	106.30±1.69	0.0003	103.80±2.27	0.0282	105.50±1.36	0.0003	96.31±2.23
HC (cm)	113.30±1.85	0.0062	110.20±2.27	0.1597	112.40±1.46	0.0074	104.80±2.63
Waist/Hip ratio	0.94±0.01	0.0445	0.94±0.01	0.1089	0.94±0.01	0.0084	0.89±0.03
Waist/Height ratio	0.67±0.01	0.0005	0.65±0.01	0.0223	0.66±0.01	0.0001	0.59±0.02
SBP (mmHg)	147.10±1.63	<0.0001	133.80±4.25	<0.0001	149.00±1.65	<0.0001	105.80±1.54
DBP (mmHg)	94.13±1.13	<0.0001	99.00±2.89	<0.0001	95.59±1.19	<0.0001	67.20±1.07
FBS (mmol L^{-1})	3.79±0.12	0.0904	4.025±0.19	0.0078	3.86±0.10	0.0307	3.52±0.08
HDL-C (mmol L^{-1})	2.03±0.07	0.4843	2.01±0.11	0.6757	2.03±0.06	0.4970	1.95±0.077
TG (mmol L^{-1})	3.84±1.42	0.7282	2.49±0.20	0.0437	3.43±0.99	0.8944	3.24±0.26
TnI (ng dL^{-1})	7.91±0.49	0.4475	7.92±0.50	0.4368	7.91±0.38	0.3937	7.19±0.79
CK (U L^{-1})	121.30±16.67	0.1654	82.40±14.27	0.7611	111.60±13.07	0.0354	69.80±5.60
AST (U L^{-1})	15.80±1.12	0.2447	14.96±1.73	0.5656	18.82±2.61	0.2054	14.08±0.66
LDH (U L^{-1})	162.20±14.68	0.2922	150.40±22.06	0.2823	159.20±12.27	0.9917	159.40±16.08

GP: Gestational Period, BMI: Body Mass-Index, WC: Waist Circumference, HC: Hip Circumference, BP: Blood Pressure, FBS: Fasting Blood Sugar, HDL: High-Density Lipoprotein, TG: Triglyceride, TnI: Troponin, CK: Creatinine Kinase, AST: Asparte Transaminase, LDH: Lactate Dehydrogenase, PIH: Pregnancy-Induced Hypertension, Data are expressed as Mean±SEM or number (%). Unpaired t-test for means and χ^2-test for categorical variables compared with controls. Each comparison is performed between hypertensive groups individually (GH: Gestational Hypertension, PE: Preeclampsia, PIH: Gestational Hypertension+preeclampsia combined) and the CG: Control Group

Table 2: The distribution of obesity, hypertension, diabetes and dyslipidemia amongst the general study population

Parameters	PIH (n = 100)	PE (n = 30)	GH (n = 70)	Control (n = 50)
WC (cm)				
Normal	2(2)	1(3)	1(1)	2(4)
Central obesity	98(98)	29(97)	69(99)	48(96)
BMI (kg m^{-2})				
Normal	24(24)	4(13)	20(29)	24(48)
Under wt.	21(21)	5(17)	16(23)	14(28)
Obesity	55(55)	21(70)	34(48)	12(24)
WHR				
Normal	2(2)	1(3)	1(1)	2(4)
Obesity	98(98)	29(97)	69(99)	48(96)
WHtR				
Normal	2(2)	1(3)	1(1)	2(4)
Obesity	98(98)	29(97)	69(99)	48(96)
SBP (mmHg)				
Normal	5(5)	3(10)	2(3)	50(100)
HPT	95(95)	27(90)	68(97)	0(0)
DBP (mmHg)				
Normal	7(7)	2(7)	66(94)	50(100)
HPT	93(93)	28(93)	4(6)	0(0)
FBS (mmol L^{-1})				
Normal	93(93)	27(90)	60(94)	50(100)
Diabetic	7(7)	3(10)	4(6)	0(0)
TG (mmol L^{-1})				
Normal	40(40)	12(40)	28(40)	8(16)
Dislipidemia	60(60)	18(60)	42(60)	42(84)

Table 3: Prevalence of hyperglycemia and the incidence of its compounds among women in the hypertensive group and controls

Criteria	PIH	p-value	PE	p-value	GH	p-value	CG
NCEP III criteria							
Abdominal obesity	107.30±1.24	<0.0001	105.60±2.03	0.0003	108.00±1.53	<0.0001	98.63±0.92
Hypertriglyceridemia	4.27±1.38	0.7044	2.86±0.22	0.072	4.88±1.98	0.5378	3.55±0.27
Low HDL-C	2.19±0.05	0.7268	2.17±0.10	0.6738	2.20±0.06	0.815	2.06±0.07
SBP	149.10±1.55	<0.0001	155.20±3.39	<0.0001	146.80±1.60	<0.0001	0.00±0.00
DBP	97.38±1.02	<0.0001	102.20±2.09	<0.0001	95.45±1.07	<0.0001	0.00±0.00
High glucose	8.22±1.71	<0.0001	6.47±0.15	<0.0001	9.54±2.98	<0.0001	0.00±0.00
WHO criteria							
Central obesity	106.30±1.29	0.0001	104.80±2.12	0.0032	106.90±1.60	0.0001	98.63±0.92
Hypertriglyceridemia	4.75±1.65	0.7044	3.09±0.24	0.0720	5.46±2.35	0.5378	3.59±0.27
Low HDL-C	0.76±0.12	0.2003	0.80±0.19	0.3176	0.74±0.17	0.1991	0.45±0.00
SBP	149.70±1.56	<0.0001	156.20±3.38	<0.0001	147.10±1.60	<0.0001	0.00±0.00
DBP	97.52±1.04	<0.0001	102.70±2.11	<0.0001	95.22±1.09	<0.0001	0.00±0.00
High glucose	8.22±1.71	<0.0001	6.47±0.15	<0.0001	9.54±2.98	<0.0001	0.00±0.00

were increased though not significant, all the other indicators of obesity (Weight (WT), Body Mass Index (BMI), Waist Circumference (WC) and Waist-to-Height Ratio (WHtR) were significantly higher in the entire studied group (Pregnancy Induced-Hypertension (PIH), Gestational Hypertension (GH) and Preeclampsia (PE) as compared to the control group. In general, fasting glucose levels were significantly higher in women with PE and PIH and triglyceride levels were lower in women with PE as compared to the control group. The cardiac profile generally did not show any significant change when all the studied groups were compared to the control group.

The percentage prevalence of obesity, hypertension, diabetes and dyslipidemia among the various studied groups as compared to the control group are shown in Table 2. Notable was the 31, 46 and 24% increase in BMI obesity as compared to the control group for PIH, PE and GH, respectively. Also, there was a 7, 10 and 6% increase in hyperglycemia as compared to the control group for the PIH, PE and GH, respectively. As shown in Table 3, with the exception of TG and HDL, the mean value of each of the components of metabolic syndrome was significantly higher in the entire studied group as compared to the control group using the two criteria (the WHO and NCEP adapted definitions).

The proportions of women with components of the metabolic syndrome are shown in Table 4. The rate of women with hyperglycemia, hypertension and dyslipidemia were increased in women in the studied group as compared to the control group. From the same Table 4, fasting hyperglycemia and blood pressure are significant predictors of the metabolic syndrome in all the studied group of women using the NCEP and the WHO criteria. There were no cases of metabolic syndrome in women with normal pregnancy according to the WHO adapted definition while 10% of them had metabolic syndrome according the NCEP adapted definition. When comparing WHO and NCEP adapted definitions in each

Table 4: Prevalence of metabolic syndrome and the incidence of its components among women in the hypertensive group and controls

Criteria	GH (n = 70)	*p-value	PE (n = 30)	p-value	PIH (n = 100)	*p-value	OR(95% CI)†	CG (n = 50)
WHO criteria§								
FBS≥6.1 mmol L^{-1}	4(6)	0.1472	3(10)	0.0594	7(7)	0.0979	----------	0(0)
WC≥80 cm	69(99)	1.0000	29(97)	1.0000	98(98)	1.0000	1.0(0.6-1.7)	48(96)
HDL-C<1.00;	4(6)	0.6476	2(7)	0.5559	6(6)	0.4288	3.0(0.4-25.6)	1(2)
TG≥2.0 mmol L^{-1}								
BP≥140/90 mmHg	63(90)	<0.0001	26(87)	<0.0001	89(89)	< 0.0001	----------	0(0)
Metabolic syndrome	6(9)	0.0803	4(13)	0.0240	10(10)	0.0316	----------	0(0)
NCEP III criteria‡								
BP≥130/85 mmHg	65(93)	<0.0001	27(90)	<0.0001	92(92)	<0.0001	----------	0(0)
FBS≥6.1 mmol L^{-1}	4(6)	0.1472	3(10)	0.0594	7(7)	0.0979	----------	0(0)
HDL<1.3 mmol L^{-1}	10(14)	0.5902	4(13)	0.7271	14(14)	0.6130	1.4(0.5-4.1)	5(10)
TG≥1.7 mmol L^{-1}	50(71)	0.5777	22(73)	0.7285	72(72)	0.5179	0.8(0.5-1.4)	43(86)
WC≥88 cm	66(94)	1.0000	28(93)	1.0000	94(94)	1.0000	1.0(0.6-1.6)	47(94)
Metabolic syndrome	43(61)	<0.0001	43(61)	0.0004	62(62)	<0.0001	6.2(2.3-16.4)	5(10)

OR: Odds Ratio, CI: Confidence Interval, WHO: World Health Organization, NCEP: National Cholesterol Education Program, HDL-C: High-Density Lipoprotein Cholesterol, BP: Blood Pressure, FBS: Fasting Blood Sugar, BP: Blood Pressure, TG: Triglycerides. Data are presented as n (%). Each comparison is performed between hypertensive groups individually (GH: Gestational Hypertension, PE: Preeclampsia, PIH: Gestational Hypertension +preeclampsia) and the CG: Control Group. Chi-squared test or Fisher exact test whenever n<5 compared with controls. †Odds ratios are presented for the preeclampsia and gestational hypertension group combined. ‡Three or more criteria are diagnostic of the metabolic syndrome. §One of the 3 criteria of insulin resistance and at least 2 other criteria are diagnostic of the metabolic syndrome

studied group, the proportion of women with the metabolic syndrome was highest in women with PE (13%) followed by PIH (10%) and GH (6%) using the WHO adapted definition. Using the NCEP adapted definition, the metabolic syndrome was higher in PIH (62%) and exactly the same in women with preeclampsia (61%) and gestational hypertension (61%) (Table 4).

DISCUSSION

Available literature suggests that women who develop pregnancy induced-hypertension and/or PE have metabolic abnormalities similar to those present in patients with insulin resistance syndrome (Kaaja *et al*., 1995; Sattar *et al*., 1997). A high type 2 diabetes in patients who developed PIH (PE and GH) compared to patients at risk of but without hypertensive disorders was also reported (Gans *et al*., 1996). The significance increased in all the indicators of obesity from this study which is in accordance with earlier report (Gans *et al*., 1996), suggesting that the association between the metabolic syndrome and PIH was determined by the degree of obesity.

These increased obesity across the study population can be suggested to be a result of the Ghanaian woman's passion for increases in size and weight especially after marriage, this they consider cosmetic (Personal view and observation). Although, times are changing now but, majority of Ghanaians still hold this view. Ghanaians, generally associate plumpness with beauty in women and success in both sexes. It is, therefore, not surprising that some women and indeed some men go out of their way to put on weight in order to appear beautiful or prosperous. Ghanaian men are also perceived to prefer plumb and overweight women to thin ones (Owiredu *et al*., 2008), so their women opt for means of getting fat. This may possibly contribute to the higher BMI and WC in both groups.

Whereas no information was given regarding lipid profile by Gans *et al*. (1996) and Kaaja *et al*. (1995) reported high HDL-cholesterol, triglyceride and fasting glucose concentrations in both proteinuric and non-proteinuric hypertensive women. Though this study confirms the high fasting blood sugar, HDL-cholesterol did not show any significant difference with triglyceride decreasing in PE patients. Obesity is known to underline disturbances of lipid and glucose metabolisms that are posing one of the greatest threats to health in the world today (Stage *et al*., 2004; Wing *et al*., 1998).

Despite the fact that all of these variables are known to be related to the metabolic syndrome (Catalano *et al*., 1993), the issue of whether PE and other hypertensive states in pregnancy are associated with the presence of abnormalities observed in patients with the metabolic syndrome was not well established. In the present report, we found that women with PIH, PE and GH were characterized by increased FBS and high obesity.

The differences in carbohydrate metabolism between PIH (7% hyperglycemia), PE (10% hyperglycemia) and GH (6% hyperglycemia) agree in part with previous reports showing a significantly elevated frequency of type 2 diabetes and presumably increased insulin resistance, among women in whom transient hypertension developed during pregnancy but not among women in whom PE developed (Solomon *et al*., 1994). The observation of a 31% (PIH), 46% (PE) and 24% (GH) increase in obesity in our patients with hypertension compared to controls could explain, at least in part, the increase in metabolic

abnormalities seen in this group. Indeed, it has been shown that obesity plays a role in the development of the metabolic syndrome in pregnancy (Sivan *et al*., 1997). However, it has recently been suggested that, in non-pregnant subjects, obesity alone does not account for the presence of the metabolic syndrome (Zavaroni *et al*., 1993).

It has been postulated that the more insulin resistant an individual and the higher the resultant plasma glucose concentration, the greater the increase in hepatic VLDL-TG synthesis and secretion and plasma TG concentration (Reaven *et al*., 1967; Tobey *et al*., 1981). Contrary to this assertion there was no significant change in plasma HDL and TG in this study (except in PE where there was a significant decrease in TG) even though the FBS was significantly increased. This lack of a statistical significant difference could be due to the small sample size.

In this study, the prevalence of the metabolic syndrome was 10% (WHO) and 62% (NCEP III) in women who had PIH compared to those with normotensive pregnancy which was 10 and 0% for the NCEP III and WHO criteria respectively. This demonstrates that using the NCEPIII criteria more pregnant women (PIH) were seen to have the metabolic syndrome and that metabolic syndrome was higher in the PIH women as compared to the controls using both criteria. In their study, Bartha *et al*. (2008) and Forest *et al*. (2005) similarly found a higher prevalence of the metabolic syndrome in PIH subjects as compared to the control group using both criteria. These findings suggest that the metabolic syndrome appears early in a division of women after an episode of PIH.

Even though, Bartha *et al*. (2008) did not find any significant differences in the prevalence of metabolic syndrome within the same hypertensive patient sub-group using the WHO and the NCEP III criteria, this study is in agreement with the study of Forest *et al*. (2005). It was found that, using the WHO criteria, women with preeclampsia and gestational hypertension were both significantly more likely to have metabolic syndrome, whereas using the NCEP III criteria, the prevalence of the metabolic syndrome was significantly higher only in the gestational hypertension group.

These differences re-emphasize the need for a universal criteria for the metabolic syndrome because generating estimates has been complicated by the use of many definitions of the metabolic syndrome, with no standard definition been routinely used (Ford and Giles, 2003). Over the last decade, the link between the metabolic syndrome and cardiovascular disease has become well established (Isomaa *et al*., 2001; Lakka *et al*., 2002). Consequently, this relationship was explored in the subjects by analyzing for some cardiovascular disease markers. Interestingly, no such association was found between cardiovascular disease and Pregnancy-induced hypertension in this study. Although, this study has demonstrated low cardiovascular risk profiles in the subjects, major retrospective studies involving Pregnancy-induced hypertensive patients have found an increased risk of cardiovascular disease mainly in women who delivered before term (Smith *et al*., 2001) and later in life (Forest *et al*., 2005; Seely and Solomon, 2003).

By appropriately recognizing the metabolically challenged pregnancy, we could have the opportunity to prevent or delay the onset of clinical disease and because of the increased risk of morbidity and mortality associated with the metabolic syndrome, an understanding of the presentations of this syndrome is vital especially among pregnant women. It must be noted that, since the studied population is largely of Ghanaian origin, the study findings apply mainly to this population and may not be transferable to other population, ethnic or cultural groups without additional research and studies because of differences in ethnic and cultural beliefs and norms and also because of disparities in the prevalence of the metabolic syndrome.

CONCLUSION

In conclusion, the results of this study suggest that alterations in carbohydrate and to some extent lipid metabolism are present in women with PIH. These metabolic features could play a role in the pathogenesis of vascular dysfunction in these patients. The findings in this study support an association between the metabolic syndrome and the hypertensive state associated with PIH. Also, the results of this study has shown that women with PIH (PE and GH) have a higher prevalence of clusters of the metabolic syndrome (hypertension, obesity, diabetes mellitus) than their controls which is in good agreement with the notion that women prone to PIH have the ability towards developing metabolic syndrome (Sattar and Greer, 2002).

As such, women who wish to conceive should be screened for features of the metabolic syndrome such as glucose intolerance, central obesity and lipid abnormalities with the view to preventing PIH which has adverse effects on both the mother and the foetus. In addition, obstetricians should be encouraged to collaborate with nutritionist and physiotherapist in the management of pregnant women to ensure that only acceptable weight gain is countenanced.

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