
Prevalence of Microalbuminuria among Patients with Gestational Hypertension in Kano

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Abstract

Background: Gestational hypertension is prevalent in developing countries including Nigeria. Studies have shown that patients with this disorder have eleven-fold higher risk of developing end stage renal disease compared with normal pregnant women without the disorder. However, patients at risk can be identified by determination of urine albumin:creatinine ratio (UACR) and those with microalbuminuria could benefit from preventive and possibly more intensive therapeutic interventions.

Aim: To determine the prevalence of microalbuminuria in patients with gestational hypertension. This is with the aim of reducing pregnancy associated renal disease, through the use of a possible biomarker.

Methods: This research was a cross-sectional study conducted between August, 2014 and June, 2016. Urine albumin, urine creatinine were measured in 100 patients with gestational hypertension and 100 controls using hemo Cue albumin autoanalyser and Kenzaautoanalyser respectively.

Results: Prevalence of microalbuminuria in patients with gestational hypertension was 42%. Young age, primiparities were found to be associated with development of gestational hypertension. Increased number of parity was found to be associated with severity of hypertension.

Conclusions: Microalbuminuria is common among gestational hypertensives and routine screening for microalbuminuria as part of the initial evaluation of patients with gestational hypertension is advocated. This would result in preventive intervention against renal disease in affected individuals.

Keywords: Prevalence; Microalbuminuria; Gestational hypertension

Introduction

Gestational hypertension, which was formerly known as pregnancy induced hypertension,

is defined as sustained rise in systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg in a previously normotensive pregnant woman who is at ≥ 20 weeks of gestation and has no proteinuria or new signs of end-organ dysfunction (ACOG, 2013). The blood pressure readings should be documented on two occasions, at least four hours apart (WHO, 2005).

Prevalence of gestational hypertension vary substantially in different regions of the world ranging from 4 - 10% of all pregnancies (Yoder *et al*, 2009). Gestational hypertension is known to complicate up to 1 in 10 pregnancies in the United States (Rocella, 2000). Also an incidence rate of 13.9% has been reported in a study conducted in South Africa (Solange *et al*, 1999). Gestational hypertension is a major cause of maternal morbidity and mortality especially in developing countries like Nigeria (Olayemi *et al*, 2010). Incidence of gestational hypertension in a study conducted in an urban Nigerian city of Ibadan was found to be 28.9%⁶. However, the National average was found to be 3.7% according to National Centre for Health Statistics 2001 (Nwabueze *et al*, 2012).

Though the cause of gestational hypertension is unknown, there are some identified associated risk factors which include first pregnancy, advanced maternal age (> 35 years), high body mass index (BMI), family history of gestational hypertension, history of kidney disease and high serum uric acid level (Fang and Alderman, 2000; Zamoski and Green, 2001). Other notable risk factors include multiple gestation, placental abnormalities, family history of pre-eclampsia and African – American race (Zamoski and Green, 2001).

With the high prevalence of gestational hypertension locally and worldwide, only few researches have been conducted on the long term effects of the disorder (Hannaford *et al*, 1997). Studies have shown that women with hypertensive disorders during pregnancy are at higher risk of developing end-stage renal disease than other women without complicated pregnancy. Thus, the incidence of chronic kidney disease is almost 11-fold higher in those with gestational hypertension compared with normal pregnant women without the disorder (Karalliedde *et al*, 2004). Patients with gestational hypertension are also at increased risk of developing hypertension and cardiovascular disease later in life (Hannaford *et al*, 1997).

Early detection with the use of screening tests such as determination of the level of albumin excretion by the kidneys provides opportunity for early identification of individuals at risk who may benefit from preventive and more intensive therapeutic interventions to prevent the development of renal dysfunction (Mancia *et al*, 2007). Albumin excretion by the kidneys can be quantified by measuring the albumin excretion rate in the urine and excretion rate in the range of 30 – 299 $\mu\text{g}/\text{mg}$ of creatinine is termed microalbuminuria (Karalliedde *et al*, 2004). Microalbuminuria describes increase transcapillary escape rate of albumin and is regarded as a marker of microvascular disease (Karalliedde *et al*, 2004). It is a predictor of chronic kidney disease and also identifies group of individuals at increased risk of developing coronary artery disease (David, 2008). Interventions such as good control of blood pressure, and optimal glycaemic control may slow the rate of decline in renal function (Montalescot and Collet, 2005).

Although there is a known association between microalbuminuria, chronic kidney disease

(CKD), and cardiovascular disease (CVD), determination of microalbuminuria is yet to be made routine for ante-natal patients with hypertensive disorders including those with gestational hypertension (Knight *et al*, 2003; Kano, 2013).

This study is therefore aimed at assessing the prevalence of microalbuminuria among gestational hypertensives with a view to creating awareness among attending obstetricians in order to prevent and reduce renal complications post pregnancy.

Materials and Methods:

The study was a cross-sectional study, conducted between August, 2014 and June, 2016 at the ante-natal clinic of Murtala Muhammad Specialist Hospital, Kano.

The study recruited 100 pregnant women diagnosed of gestational hypertension and 100 age-matched normotensive pregnant women served as controls. The gestational age of both gestational hypertensive patients and the controls was 20 weeks and above.

Excluded from the study were patients being managed for hypertension that predates the index pregnancy, patients with established renal disease or suspected renal disease, known diabetics and obese individuals, individuals with signs and symptoms of urinary tract infection, patients with acute illnesses, and patients with overt proteinuria.

The bio data of all study subjects were obtained with the use of pre-tested, structured interviewer- administered questionnaire. The questionnaire was administered at the time of sample collection.

Blood pressure measurement was done after 5 minutes of rest with the subject sitting upright and upper arm positioned at same level with the heart. With the appropriate sized cuff the systolic blood pressure was taken as the pressure on the mercury column at which the first Korotkoff sound was heard and the diastolic blood pressure is the pressure at which the fourth Korotkoff sound was barely audible. Measurement was taken three times and average value was taken and recorded.

An early morning fresh midstream urine sample was collected using sterile sample bottle. Samples collected were stored refrigerated at 2 - 8°C until analysis. The storage temperature was validated with the use of temperature monitoring chart on the refrigerator.

Visual inspection and dipstick examination of urine was carried out on the urine samples after collection. Samples with obvious turbidity were excluded.

Albumin and creatinine were measured quantitatively in all urine samples using HemoCue albumin analyser (which utilizes the principle of turbidatex immunoassay) and Kenzoautoanalyzer (which uses the principle of Jaffe colorimetry) respectively (Gilbert, 1994; Edmund and Christopher, 2008).

The urine albumin:creatinine ratio (UACR) was calculated mathematically. Values were interpreted using the American Diabetic Association (ADA) and National Kidney Foundation recommended albumin:creatinine ratio cut-off points. Microalbuminuria was defined as UACR between 30 and 299 µg/mg creatinine (ADA, 2008).

Results:

The baseline characteristics of the studied subjects are shown in Table 1.

The mean ages of the patients and controls were statistically similar ($p = 0.07$, Table 1). Although there were more primiparas among the patients than the controls, there was no significant statistical difference in the gestational age between the patients and the control

group.

Both mean systolic and mean diastolic blood pressures were clearly higher among the patients with gestational hypertension compared to the corresponding mean values observed among the control group.

Significant statistical difference was observed in the urine albumin values between patients and controls while no statistical difference was noted in urine creatinine values between them.

Over half of the gestational hypertensive patients were primipara while most of the control group was made up of almost equal proportions of both primipara and multipara.

Table 2 shows that the mean UACR value was significantly higher among the gestational hypertensives than the controls with the levels higher among the primipara compared to the values observed in both the multipara and the grand multipara. Also, patients with severe hypertension had higher levels of UACR than the patients with mild and moderate hypertension.

The prevalence of microalbuminuria was 42% among patients with gestational hypertension while the prevalence was 25% among the controls (Table 3).

Discussion

The age distribution of subjects in this study is similar to the report of a local study conducted recently (Abdulsalam and Yahaya, 2015). The similarity may be due to the socio-cultural practice of early marriage in northern part of Nigeria where the study was conducted. However higher mean age among patients with gestational hypertension was reported in Canada, where girls are married off at older age (Rony et al, 2011). Many authors have identified extreme ages of reproductive years as risk factor for hypertension in pregnancy as observed in this present study (Mary et al, 2014). The higher number of primiparas among the patients with gestational hypertension in this study agrees with a previous report from a study conducted on patients with gestational hypertension in Kano (Yakasai and Gaya, 2011). Similar findings were reported from a study conducted on Ghanaian women with pregnancy induced hypertension (Tupin et al, 2008). This is in keeping with the fact that primiparity has been recognized as a risk factor for gestational hypertension (Elisabete, 2007). Many authors described association between primiparity and hypertension to be due to biological responses to pregnancy and the life-style risk factors associated with childbearing. These life-style risk factors include anxiety and stress associated with childbearing which were shown to lead to insulin resistance and increased risk of hypertension especially in primiparous patients (Jasovic et al, 2011).

Though, the mean gestational age of subjects in this present study was in agreement with the mean gestational age reported by Swati et al., (2014), a higher mean gestational age among gestational hypertensives was reported in Pakistan (Manusha *etal*, 2014). This difference may be related to the cross-sectional nature of this study and also the socio-demographic differences of the two study areas.

In this study, urine albumin value was found to be higher among gestational hypertensives and this finding was similar to what was reported from other studies in Nigeria and abroad (Ibeh et al, 2006; Irene et al, 2013). Studies have shown that increased albumin excretion among gestational hypertensives may be due to the abnormal differentiation pathway during cytotrophoblastic invasion of the uterus, which gives rise to abnormality in the nitric oxide

(NO) pathway for controlling vascular tone. The inhibition of the synthesis of NO induces higher sensitivity to vasoconstriction and thus chronic placental ischaemia and oxidative stress ensues. This oxidative stress induces release into maternal circulation of substances such as free radicals, oxidized lipids, cytokines and serum soluble vascular endothelial growth factor resulting in endothelial dysfunction. Consequently, depletion of vascular endothelial growth factor in the podocytes makes endotheliosis more able to block the slit diaphragm in the basement membrane leading to increase protein loss (Poon et al, 2008). The urine albumin: creatinine ratio (UACR) of this study was found to have positive association with severity of hypertension among gestational hypertensives i.e the higher the level of blood pressure the higher the mean UACR. This finding was consistent with the report of a study conducted in Uppsala, Sweden (Marques et al, 2015). This observation can be attributed to greater endothelial dysfunction and malignant sclerosis attributable to higher elevation in blood pressure seen among patients with severe hypertension (Marques et al, 2015).

The association between albuminuria and number of parity in our study was in agreement with previous reports (Raghupathy et al, 2014). Many authors postulated impaired trophoblastic invasion with atherosclerotic lesion as a causal mechanism for increased albumin excretion by the kidneys among patients with gestational hypertension (Raghupathy et al, 2014). The resultant narrowing of uterine arterioles leads to placental ischaemia. The resultant ischaemia causes alteration in the expression of various factors (vascular endothelial growth factor, placental growth factor, prostacyclin, nitric oxide) that have effect on endothelial function, from the vascular endothelium. Associated increased expression of angiogenic factors such as tumour necrosis factor- α , interleukin -1, together result in widespread endothelial dysfunction all over the body. This gives rise to glomerular changes with endothelial vacuolization and hypertrophy of cytoplasmic organelles leading to glomerular endotheliosis with net effect of reduced renal blood flow and impaired tubular reabsorptive capacity (Raghupathy et al, 2014).

The prevalence of microalbuminuria was found to be 42%. These finding was similar to a research work reported in New Delhi, India where a high prevalence of microalbuminuria was observed among pregnant women diagnosed of gestational hypertension (Raghupathy et al, 2014). This similarity can be attributed to the fact that Nigeria and India both have high prevalence of hypertension among their populations thus making prevalence of microalbuminuria high in the two regions (Risberg et al, 2004; James et al, 2015). Similarly, researchers in Northern Europe reported a prevalence of 27.3% among pregnant women with pregnancy-induced hypertension attending ante-natal clinic in Scandinavia (Habbal et al, 2010).

Generally, prevalence of microalbuminuria is known to be high among various groups of hypertensives with reported worldwide prevalence of 58.3% (Habbal et al, 2010). Racial difference was shown to greatly influence albumin excretion rate among whites and blacks as reported by some researchers who found 32% and 14% prevalence of microalbuminuria among black and white hypertensives in the United States of America respectively (Prujijim et al, 2008). Similarly, among hypertensive women in East Africa, a prevalence of 26.6% in those with grade I hypertension and prevalence of 42.6% in those with grade II hypertension was reported in 2008 (Marques et al, 2015). The similarity in the two reports can be attributed to the similar techniques employed by the researchers in analysing urine albumin and also regional and racial similarity between the two study groups. The finding of this study is also similar to a report from Portugal where a prevalence of 43% was found among gestational

hypertensive patients (Marques et al, 2015).

In contrast to the high prevalence rate of microalbuminuria observed among gestational hypertensives in this study, some researchers in Italy found a lower prevalence of microalbuminuria among patients with gestational hypertension in a large clinical trial that involved 787 patients. (Pontremoli et al, 1997). The discrepancy may be due to different criteria used in patient selection i.e, the severity of the hypertension, size of the study population, age, race, exclusion criteria, and the techniques used for urine albumin determination.

This study has also shown that severity of albuminuria positively correlated with the severity of hypertension. Similar observation was described in a multi-centered VIP study in United Kingdom (Bramhan et al, 2013).

Conclusion:

The prevalence of microalbuminuria was found to be high (42%) among women with gestational hypertension. Younger age and primiparity were found to be associated with development of gestational hypertension. Increased number of parity was also found to be positively associated with severity of hypertension.

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Table 1: Baseline characteristics of gestational hypertensive patients and controls

Variable n = 100	Patients n = 100	Controls	P-value
Age (years)	28.3 ± 6.2	26.7 ± 5.3	0.07
Gestational Age (months)	34.6 ± 6.2	33.7 ± 6.1	0.26
Systolic Blood Pressure (mmHg)	158.9 ± 17.9	112.5 ± 12.2	0.01*
Diastolic Blood Pressure (mmHg)	105.3 ± 11.7	71.04 ± 8.8	0.01*
Body Mass Index (Kg/m ²)	29.6 ± 6.2	27.9 ± 3.9	0.02*
Parity			
Primipara	52 (52%)	45 (45%)	0.01*
Multipara	33 (33%)	43 (43%)	0.30
Grand multipara	15 (15%)	12 (12%)	0.93
Urine Albumin (mg/L)	198.3 ± 25.4	26.8 ± 3.8	0.01*
Urine Creatinine (mg/L)	6226 ± 33.5	5190 ± 32.9	0.33

Results are presented as means ± standard deviations, or as numbers with percentages in brackets.

*p-value statistically significant;

Table 2: Urine Albumin:Creatinine ratio (UACR) by Parity and hypertension in gestational hypertensive patients and Controls.

Variable	UACR (µg/mg)		P value
	Patients	Controls	
Parity			
Primipara	298 ± 1.5	54.7 ± 9.7	0.01*
Multipara	291 ± 2.7	66.0 ± 1.4	0.01*
Grand multipara	295 ± 3.8	75.9 ± 9.0	0.01*
Hypertesion			
Mild	164 ± 10.6	56.7 ± 9.5	0.01*
Moderate	215 ± 3.0	56.0 ± 2.2	0.01*
Severe	294 ± 5.3	76.3 ± 5.3	0.01*

Results are presented as mean ± standard deviation; *p-value statistically significant

Table 3: Prevalence of Microalbuminuria by Parity and hypertension in patients with gestational hypertension and Controls

Variable	Patients n = 100	Controls n = 100	P value
Parity			
Primipara	27 (27%)	6 (6%)	0.01*
Multipara	12 (12%)	8 (8%)	0.01*
Grand multipara	3 (3%)	11 (11%)	0.01*
Hypertension			
	42 (42%)	25 (25%)	0.02*

Results are presented as numbers with percentages in brackets