ORIGINAL ARTICLE

Maternal renal artery Doppler sonographic changes in pregnancy-induced hypertension in South West Nigeria

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ABSTRACT

Background: To evaluate the renal arterial hemodynamic changes caused by pregnancy-induced hypertension using Doppler ultrasonography. **Materials and Methods:** Eighty (80) subjects with pregnancy-induced hypertension (PIH) and 160 controls (80 pregnant normotensive women and 80 healthy, non-pregnant women) underwent triplex renal sonography prospectively to determine their renal volumes and right renal artery Doppler indices. **Results:** The peak systolic velocity, end diastolic velocity, pulsatility index, systolic/diastolic ratio and acceleration time were respectively significantly higher in the PIH group (68.67 cm/s, 21.55 cm/s, 1.23, 3.38, 123.2 ms) than the pregnant, normotensive group (65.19 cm/s, 20.27 cm/s, 0.88, 3.35, 61.14 ms) and healthy, non-pregnant group (52.06 cm/s, 18.27 cm/s, 0.84, 2.90, 68.48 ms). Resistivity index was also increased in the PIH group, but this was not statistically significant. Conversely, the systolic acceleration was significantly lower in the PIH group (6.06 m/s^2) compared to the pregnant, normotensive group (11.82 m/s^2) and healthy, non-pregnant group (8.26 m/s^2). The right renal volume of the PIH group (132.76 cm^3) was significantly higher that of the pregnant, normotensive group (125.29 cm^3) and healthy, non-pregnant group (91.66 cm^3). The same pattern was observed in the left renal volume which was 168.78 cm^3, 164.95 cm^3 and 113.80 cm^3 in the study groups, respectively. **Conclusion:** Renal Doppler ultrasound is clinically relevant in the diagnosis and follow-up of renal complications in patients with pregnancy-induced hypertension.

Key words: Doppler ultrasonography, hypertension, pregnancy, renal artery

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INTRODUCTION

Pregnancy-induced hypertension (PIH) is a syndrome of hypertension with or without proteinuria and edema. It is a major complication of pregnancy.[1] PIH encompasses gestational hypertension, pre-eclampsia (PET) and eclampsia.[2] PET/eclampsia causes 50,000 maternal deaths worldwide annually,[3,4] complicating 0.91% of all deliveries in a tropical centre.[5]

Blood flow velocity is directly related to peripheral vascular resistance[6,7] and there are changes in downstream resistance of the renal artery in PIH.[8-12]

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This study purposed to establish the effects of PIH on renal artery Doppler indices in this locality with the aim of detecting its renal complications early.

MATERIALS AND METHODS

This was a prospective, case-control, non-randomized study carried out from February 2011 to January 2012. The hospital's Ethics and Research Committee approved the study. Eighty newly diagnosed pregnant subjects with PIH were recruited while 80 normotensive pregnant subjects and 80 normotensive, non-pregnant female subjects formed the two other study groups. The inclusion criteria were:

1. Pregnant subjects (greater than 20 weeks gestational age) with systemic blood pressure of or greater than 140/90 mmHg; or systolic blood pressure exceeding 30 mmHg, or diastolic blood pressure exceeding 15 mmHg above the recorded baseline blood pressure on two occasions at least 6 hours apart, for the PIH group.
2. Healthy pregnant, normotensive subjects referred for routine ultrasound scan (second group of subjects).

3. Healthy non-pregnant female subjects without hypertension.

The exclusion criteria were: Chronic renal disease, chronic hypertension predating pregnancy, diabetes mellitus, multiple gestation, urinary tract infection and previous renal surgery. Verbal informed consent was obtained from all participants. At presentation, medical history was taken and the blood pressures of the subjects were recorded. Information such as presenting complaint(s), age and drug history were obtained from the subjects. Laboratory results were checked to detect the presence and/or degree of proteinuria. Serum creatinine, urinalysis and complete blood count results were also reviewed. Subjects were classified as proteinuric when greater than or equal to 0.3g/L of protein was present in their random urine specimen. Doppler ultrasound assessment was performed with a Mindray® DC-6 ultrasound scanner (Shenzhen Mindray Bio-medical Electronics, Nanshan, Shenzhen, China)equipped with a curvilinear probe of frequency ranging from 3.5 to 5 MHz. This was done before the commencement of medication by the Obstetrics and Gynecology team.

The subjects were positioned supine or lateral decubitus on the examination couch. After appropriate exposure of the abdomen, acoustic gel was applied and both kidneys were scanned to rule-out gross abnormalities in renal size, shape and echogenicity. The longitudinal (L), transverse (T) and anteroposterior (AP) renal diameters were measured and the renal volume (RV) was calculated from these parameters using the following ellipsoid equation:

RV = L xTxAP x0.523

The right renal artery was used for the Doppler study. Subjects were placed in lateral decubitus position and asked to relax their abdominal muscles. Modified flank approach was employed to visualize the entire length of the artery. The probe was placed beneath the rib cage in sagittal orientation and rotated to the right until a longitudinal axis view of the abdominal aorta and inferior *vena cava* was obtained. With a Doppler angle of 60° or less, the pulsating right renal artery was then insonated by placing the pulsed Doppler sample volume gate within it to obtain an angle-corrected velocity waveform measurement during a period of suspended respiration. The Doppler sample volume was set at 3mm, and a 100 Hz pass filter was used to reduce the noise from the pulsating arterial wall. Pulse repetition Frequency (PRF) of 2500 Hz was used. The peak systolic velocity (PSV) and end diastolic velocity (EDV) were measured at the apex of the highest systolic peak and at the end of diastole, respectively. The acceleration time was measured from the beginning of the systolic upstroke to the highest systolic peak of the arterial waveform. Breaks in the systolic upstroke before reaching the peak were not included. The velocity waveform was analyzed for Resistivity Index (RI) of Pourcelot,[13]Pulsatility Index (PI) of Gosling *et al*.,[14] and systolic/diastolic ratio (S/D).[15]These indices were calculated automatically by the ultrasound machine's software.

The data were reported as mean and standard deviation for continuous variables, while categorical and dichotomous variables were reported as frequencies and percentages. The level of significance was determined at $P < 0.05$. Independent samples test was used to compare the mean age of subjects and controls. One-way analysis of variance (ANOVA) was used to compare means of variables involving three (3) or more groups. Analysis was done using the Statistical Package for Social Sciences (SPSS Inc., Chicago, IL, USA), version 17.0 for windows.

RESULTS

The mean age of the subjects with PIH was 32.15±5.29 years (range = 18-47 years). The 80 pregnant, normotensive women had a mean age of 30.5±5.25 years (range = 20-45 years) while the third group of healthy, non-pregnant women had a mean age of 30.63 ± 5.74 years (range = 19-47 years). There was no statistically significant difference between the ages of women in these groups ($P = 0.106$). No statistically significant differences in parity ($P > 0.05$) and gestational ages ($P = 0.846$) were seen between the pregnant, normotensive subjects and the subjects with PIH. Thirteen (16.25%) of the subjects with PIH had previous episodes of PIH [Table 1].

Analysis of Variance (ANOVA) showed that the systemic blood pressure (both systolic and diastolic) in subjects with PIH were significantly higher than those of normotensive pregnant subjects and healthy, non-pregnant subjects ($P < 0.05$). The healthy, non-pregnant subjects had a mean systolic blood pressure of 107.94± 8.57 mmHg, while those of normotensive, pregnant subjects and the subjects with PIH were 103.72 ± 6.98 mmHg and 158.68± 19.05 mmHg, respectively. The mean diastolic pressures were 75.55 ± 9.09 mmHg, 70.04 ± 8.95 mmHg and 102.88 ± 11.05 mmHg in the same order [Table 1].

The right and left renal volumes were significantly higher in the PIH group compared to the normotensive pregnant and healthy, non-pregnant groups. The right renal volumes were 132.76 ± 29.05cm^3, 125.29 ± 32.21cm^3 and 91.66 ± 19.30cm^3 in the PIH, normotensive pregnant and healthy, non-pregnant women respectively. The left renal volumes were 168.78 ± 36.81 cm^3, 164.95 ± 44.1 cm^3 and 113.80 ± 22.16 cm^3 in the PIH, pregnant normotensive and healthy non-pregnant groups, respectively ($P < 0.05$).

The peak systolic velocity (PSV),end diastolic velocity (EDV),acceleration time (AT), acceleration index, systolic/diastolic ratio and pulsatilityindex (PI)of the right main renal artery were significantly higher in women with PIH

Table 1: Characteristics of subjects

Characteristic	Healthy non-pregnant women (*n* = 80)	Normotensive pregnant women (*n* = 80)	Women with PIH (*n* = 80)	*P* value
Age (years)	30.63±5.74	30.51±5.25	32.15±5.29	0.106
Nulliparity (number, %)	NA	39 (48.8)	31 (38.8)	0.202
Primiparity (number, %)	NA	19 (23.8)	23 (28.8)	0.472
Gestational age at examination (weeks)	NA	31.36±5.38	31.52±5.21	0.846
Blood pressure (mmHg)				
Systole	107.94±8.57	103.72±6.98	158.68±19.05	0.000
Diastole	75.55±9.09	70.04±8.95	102.88±11.05	0.000

PIH – Pregnancy-induced hypertension

Table 2: Doppler indices in subjects

Doppler indices	Healthy non-pregnant women (*n* = 80)	Normotensive pregnant women (*n* = 80)	Women with PIH (*n* = 80)	*P* value
Peak systolic velocity (cm/s)	52.06±21.18	65.19±23.51	68.67±21.01	0.000
End-diastolic velocity (cm/s)	18.27±8.08	20.27±7.84	21.55±7.93	0.033
Resistivity inde	0.62±0.21	0.70±0.64	0.76±0.28	0.115
Acceleration index (m/s2)	5.26±2.26	7.72±4.54	6.67±4.37	0.000
Acceleration time (ms)	68.48±18.59	61.14±18.00	123.2±34.65	0.000
Systolic/diastolic ratio	2.90±0.83	3.35±0.97	3.38±0.70	0.000
Pulsatility index	0.84±0.12	0.88±0.65	1.23±0.20	0.000
Systolic acceleration (m/s2)	8.26±4.39	11.82±6.12	6.06±2.83	0.000

PIH – Pregnancy-induced hypertension

($P < 0.05$); while the systolic acceleration was significantly lower [Table 2]. Although RI was higher in subjects with PIH compared to the other groups, this was not statistically significant ($P > 0.05$).

DISCUSSION

Hypertensive disorders occur in 5-7% of all pregnancies.[2] PIH is said to occur when the systolic blood pressure is greater than 140 mmHg and diastolic blood pressure greater than 90 mmHg taken on at least two occasions, 6hours apart.[2]

The initiating events and the factors responsible for the pathogenesis of PIH are yet to be fully elucidated. Reduced uteroplacenta perfusion sequel to abnormal cytotrophoblast invasion of spiral arterioles and the resultant placental ischemia is thought to lead to widespread activation or dysfunction of the maternal vascular endothelium. This results in enhanced formation of endothelin and thromboxane, increased vascular sensitivity to angiotensin II and decreased formation of vasodilators such as nitric oxide and prostacyclin. Consequently, this triggers series of hemodynamic disorders including significant elevation of total peripheral resistivity and marked reduction in renal blood flow.[16]

In this study, the mean renal volume of subjects with PIH was significantly higher than those of the two control groups. The mean renal volume of the normotensive pregnant women was also higher than that of the healthy, non-pregnant women. These findings are similar to those of the study by Lumbominova *et al.*[17] who documented a significant increase in right renal volume in the PIH subjects compared to the pregnant, normotensive and healthy, non-pregnant women. Renal volume was also noted to be generally greater in the left than the right kidney in all the groups studied. This finding is similar to earlier observations on sonographic estimation of renal volume by Emamian *et al*.[18] and Egberongbe *et al*.[19]

The systolic blood pressures of the three groups showed statistically significant differences ($P < 0.05$). This has been attributed to the increased plasma volume in the course of normal pregnancy which leads to systemic vasodilatation and hence a decrease in blood pressure in pregnant, normotensive compared to healthy, non-pregnant women.[20] These renal and cardiovascular adjustments are important for successful pregnancy outcome. In contrast, PIH is associated with increased peripheral vascular resistance.

Several investigastors have attempted to evaluate renal circulation by performing Doppler sonography in healthy pregnant women and women with PIH.[21-23] However, the parameters analysed in these studies were restricted to the most common parameters of distal or downstream vascular resistance such as systolic/diastolic ratio, RI and PI.

In this study, the right main renal artery had a statistically significant higher PSV, EDV, S/D ratio and PI in women with PIH ($P < 0.05$). The RI was also higher in the PIH subjects but the difference was not statistically significant. Similar findings to these were reported by Sohn and Fenden[9] in Germany who studied 31 non-pregnant, 52 normotensive pregnant and 12 pregnant women with PIH. They found significant differences between the non-pregnant and normotensive pregnant subjects as well as between the non-pregnant and those with PIH. In addition to increase in renal artery resistance, the hypertensive women had markedly different flow velocity waveform which manifested as prolongation of acceleration time and reduction of the Acceleration Index. Conversely, Lubomirova *et al*.[17]did not find any differences in renal PI and velocities unlike in the indexed study which showed significant differences in these parameters.

Acceleration time (AT) is the time interval from the onset of systole to the peak of the velocity. Miyake *et al*.[24] found that the AT of the segmental and interlobar arteries were significantly prolonged in the PIH group compared to normotensive pregnant women and concluded that AT of 100milliseconds was best cut-off for distinguishing healthy pregnant women and women with PIH. This had a sensitivity and specificity of 83% and 92%, respectively.[24] Renal artery AT of the hypertensive pregnant subjects in this study was prolonged, with a mean of 123.25 ± 34.65 ms compared to values of 61.14± 18.00 ms and 68.48 ± 18.59 ms obtained in the normotensive pregnant women and healthy, non-pregnant women, respectively ($P < 0.05$).

Systolic Acceleration (SA) is defined as the ratio of PSV to AT. It is also an important indicator of hemodynamic changes in the renal artery. The SA was much lower in PIH subjects compared to the normotensive, pregnant subjects. A similar pattern was reported Yuan *et al*.[20]

The systolic/diastolic ratio was significantly higher in the PIH group than the two other study groups. However, in the study done by Kuo *et al.*,[15] 12 pregnant women with pre-eclampsia demonstrated significantly lower S/D ratio of 2.07 as compared to a normotensive, pregnant women with S/D ratio of 2.41. However, Kuo's results are not consistent with the established concept of renal arteriospasm as part of the pathophysiology of preeclampsia.

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