

The Use of Hormonal Contraceptives and Preeclampsia among Ghanaian Pregnant Women

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Abstract

Hormonal contraceptives (HC) are thought to play a role in the pathogenesis of cardiovascular diseases. The study evaluated the use of HC as a primary cause of preeclampsia (PE) among Ghanaians. This study comprised 30 preeclamptic women and 30 healthy normotensive pregnant women with over 20 weeks of gestation at the Comboni Hospital, Ghana using a randomized case-control study. Blood pressure, weight, height, socio-demographics, medical and previous obstetric history were taken and recorded. Blood samples were collected for the estimation of homocysteine and fasting lipids. Estimated foetal weight (EFW) and infant birthweight (BWT) were obtained from maternal records. This study was carried out in 2019. 80.0% of women with PE used the hormonal contraceptive “depot medroxyprogesterone acetate” (DMPA) prior to pregnancy. The use of DMPA was associated with about thirty-fold increase in the odds of developing PE (OR = 29.71, $p < 0.001$). Systolic blood pressure ($P < 0.001$), diastolic blood pressure ($P < 0.001$), triglycerides ($P = 0.024$), LDL-C ($P = 0.026$), and homocysteine levels ($P < 0.001$) were significantly elevated in the PE cases than the normal pregnant (NP) women, whilst EFW ($P < 0.001$), BWT ($P < 0.001$) and HDL-C levels ($P = 0.001$) were significantly reduced in the PE cases when compared to NP women. 50% and 47% of PE cases had intrauterine growth restriction and low birthweight infants, respectively. In conclusion, DMPA use predisposes women to a high risk of developing PE. DMPA could partly contribute to endothelial dysfunction, hyperhomocysteinaemia, dyslipidaemia and excessive weight gain, all of which characterize PE.

Keywords

Preeclampsia, Hormonal Contraceptives, Depot Medroxyprogesterone Acetate

1. Introduction

Hormonal contraception generally refers to birth control methods that act on the endocrine system. There are two main types of hormonal contraceptive formulations. These are combined methods that contain both estrogen and progestin, and progestogen-only methods which contain only progesterone or one of its synthetic analogues (progestins) [1]. Combined methods work by suppressing ovulation and thickening cervical mucus, while progestogen-only methods reduce the frequency of ovulation [1]. HC use is associated with hypertension, dyslipidemia, and insulin resistance, all of which characterize hypertensive disorders of pregnancy (HDP) [2].

HDP (which includes preeclampsia) is one of the commonest causes of maternal mortality [3] and accounted for 37,000 deaths in 1990 and 29,000 in 2013 [4]. Preeclampsia (PE) is characterized by hypertension and significant proteinuria [5]. The disorder occurs after 20 weeks of pregnancy and worsens over time [6]. In Ghana, the incidence of PE was reported to be 7.03% [7]. Another study conducted in Ghana also showed that the prevalence of PE was 6.55% [8].

Although the exact pathophysiologic mechanism is not clearly understood, PE is primarily a disorder of placental dysfunction leading to a syndrome of endothelial dysfunction with associated vasospasm. Epidemiological studies have indicated that HC may play a role in the pathogenesis of cardiovascular diseases [9]. However, the extent to which HC contributes to PE remains unknown, hence the basis of this study. Our study aimed at evaluating the use of hormonal contraceptives as a primary cause of preeclampsia (PE) among Ghanaians.

The objective of the study, therefore, was to determine the level to which DMPA could contribute to hyperhomocysteinaemia, endothelial dysfunction and PE.

2. Methods

2.1. Study Design/Site

The study was a systematic randomized case-control study conducted at Comboni Mission hospital, Sogakope, in the South Tongu District of the Volta Region of Ghana in 2019.

2.2. Study Population

Stat Cal utility feature of Epiinfo Statistical software was used for sample size determination using confidence interval of 95% and power of 80%. A minimum size of 30 cases and 30 controls was obtained. A total of 60 participants com-

prising 30 PE cases and 30 healthy normotensive pregnant women (control group), aged 18 - 40 years were recruited. The study participants comprised pregnant women with gestational age above 20 weeks to 40 weeks. The cases were recruited according to their history, clinical presentation and proteinuria equivalent to 1+ or higher on a urine dipstick test. Furthermore, cases were singleton pregnant women with systolic blood pressure ≥ 140 mm Hg and/or diastolic blood pressure ≥ 90 mm Hg.

Healthy normotensive pregnant women without any pregnancy complications or any other disease condition formed the control group. Pregnant women with the history of previous hypertension, those with twin pregnancy history and those with known vitamin deficiency (folic acid, B12) were excluded. Additionally, pregnant women suffering from diabetes, malnutrition, and those with any hepatic disorder, chronic renal disease, hypothyroidism, severe anaemia, and chronic illnesses were excluded. Cigarette smokers, alcoholics, and those on folic acid and other medication that could introduce confounding factors such as folate antagonists and nitrous oxide were also excluded.

A well-structured pre-tested questionnaire was used to obtain detailed medical, obstetric, dietary and socio-demographic data from each participant (Appendix A).

2.3. Measurement of Blood Pressure

In accordance with recommendations of the American Heart Association [10], the blood pressure of each participant was measured by trained personnel using a mercury sphygmomanometer (Omron M2 basic, Netherland). The procedure was repeated for each patient between 5 - 10 minutes. Mean values of duplicate measurements were recorded as the blood pressure to the nearest 2.0 mmHg.

2.4. Anthropometric Measurements

Weight was measured with the Omron BF-506 Body Composition Monitor (IL, USA) to the nearest 0.1 kg in light clothing with subjects barefooted and standing erect. Height was measured with a portable Seca stadiometer (Hamburg, Germany) to the nearest 0.1 centimeter.

2.5. Blood Sample Collection

About 5 ml of venous blood sample was drawn from the antecubital vein of each participant after 8 - 14 hours overnight fast and immediately transferred into gel separator tubes. The clotted samples were centrifuged at 3000 rpm for 5 minutes and the sera stored at -80°C until assayed.

2.6. Biochemical Analysis

Boehringer-Mannheim dipstick proteinuria (Mannheim, Germany) was used to determine proteinuria from the urine sample of each participant. Homocysteine (HCY) and lipid profile (HDL-C, LDL-C, total cholesterol and triglycerides)

concentrations were determined using appropriate laboratory methods.

2.7. Determination of Serum Homocysteine

HCY was determined by an enzymatic UV method, using Human homocysteine liquid UV diagnostics kit (Wiesbaden, Germany). The procedure was followed according to the manufacturer's instructions and the final chromogen measured at 340 nm on a Mindray UV spectrophotometer (Shenzhen, China).

2.8. Determination of Serum Lipid Profile

Total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C) and triglyceride (TG) were performed using BioSystem kits on an A25 BioSystem autoanalyzer (Barcelona, Spain). However, LDL was calculated as follows: $LDL = TC - (HDL + TG/2.2)$ mmol/L.

2.9. Determination of Intrauterine Growth Restriction (IUGR) and Birth Weight

Estimated foetal weight (EFW) and birthweight (BWT) were obtained from the medical record of each participant. EFW for each study participant was determined using Sonoscape S6 by a certified clinical sonologist. Using the smoothed percentiles of estimated birth weight (grams) for gestational age [11] [12], IUGR was determined using the 10th percentile.

2.10. Statistical Analysis

IBM SPSS Statistics for Windows, Version 21.0 (Armonk, NY: IBM Corporation) was used. Continuous variables were expressed as mean \pm SD. The independent sample t-test and Fisher exact tests were used where appropriate to compare means of the women with PE and the control group. Pearson correlation coefficient test was used to determine the correlation between study parameters. Using a multivariate logistic regression with a 95% confidence interval (CI), the odds ratio (OR) was determined to quantify the risk of women with PIH in comparison with controls. Statistical significance level was set at $P < 0.05$.

3. Results

3.1. Socio-Demographic and Clinical Characteristics of Study Participants

PE cases and the normal pregnant (NP) women had similar ages. The majority of the study participants were within the age range of 25 - 34 years. Other age groups' percentages were as follows: 3.3%, 21.7%, 10.0%, and 3.3% for ages >40 , 35 - 39, 20 - 24, and <20 , respectively. About twenty-eight percent (28.3%) of the participants had completed Junior High School, whereas 20.0%, 20.0% and 13.3% had completed Senior High School, Tertiary, and Basic (Primary) education, respectively. About thirty-eight percent (38.3%) of the pregnant women were obese, compared to 35.0% and 26.7%, respectively, who were considered

overweight and normal weight. Additionally, fifty-eight percent (58.3%) of the study participants had normal systolic blood pressure (SBP), compared to 41.7% who had high SBP. Normal and high diastolic blood pressure (DBP) was observed in 68.3% and 31.7% of the pregnant women, respectively. The difference in the SBP and DBP of the PE cases compared to the NP women was statistically significant at $P < 0.05$ (Table 1).

Table 1. Socio-demographic and clinical characteristics of study participants.

Parameters	PE (n = 30)	NP (n = 30)	Total (n = 60) PE + NP
Age (yrs)	30.50 ± 5.78	29.13 ± 4.98	29.82 ± 5.38
Age category (yrs)			
<20	1 (3.3%)	1 (3.3%)	2 (3.3%)
20 - 24	2 (6.7%)	4 (13.3%)	6 (10.0%)
25 - 29	9 (30.0%)	10 (33.3%)	19 (31.7%)
30 - 34	8 (26.7%)	10 (33.3%)	18 (30.0%)
35 - 39	8 (26.7%)	5 (16.7%)	13 (21.7%)
≥40	2 (6.7%)	Nil (0.0%)	2 (3.3%)
Educational Level			
No Education	8 (26.7%)	3 (10.0%)	11 (18.3%)
Basic	6 (20.0%)	2 (6.7%)	8 (13.3%)
JHS	8 (26.7%)	9 (30.0%)	17 (28.3%)
SHS	5 (16.7%)	7 (23.3%)	12 (20.0%)
Tertiary	3 (10.0%)	9 (30.0%)	12 (20.0%)
BMI			
Normal	7 (23.3%)	9 (30.0%)	16 (26.7%)
Overweight	8 (26.7%)	13 (43.3%)	21 (35.0%)
Obese	15 (50.0%)	8 (26.7%)	23 (38.3%)
SBP			
Normal	5 (16.7%)*	30 (100.0%)	35 (58.3%)
High	25 (83.3%)	Nil (0.0%)	25 (41.7%)
DBP			
Normal	11 (36.7%)*	30 (100.0%)	41 (68.3%)
High	19 (63.3%)	Nil (0.0%)	19 (31.7%)

Chi-square or Fischer exact test whenever $n < 5$ was used to compare the PE group with the NP group. * $p < 0.05$ was considered a statistically significant difference. BMI of 19 Kg/m² - 24.9 Kg/m², 25.0 Kg/m² - 29.9 Kg/m² and >30 Kg/m² were categorized as normal, overweight and obese respectively; PE: Preeclampsia; NP: Normal pregnant control; JHS: Junior high school; SHS: Senior high school; BMI: Body Mass Index; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure.

3.2. The Clinical Variables of the Study Population

The clinical variables of the study population are shown in **Table 2**. There was no statistically significant difference in the mean age between the PE cases and NP women. Furthermore, BMI, SBP, DBP, EFW, BWT, TG, LDL-C and HDL-C of the PE cases were significantly different ($P < 0.05$) from the NP women. Gestational age and total cholesterol concentrations however, showed no statistically significant differences. Majority (65.0%) of the pregnant women were within 34 - 37 weeks of gestation.

The highest percentage (28.3%) of participants were primiparous, whereas 26.7%, 21.7%, 15.0% and 8.3% were nulliparous, secundiparous, tertiparous and multiparous respectively. Majority (30.0%) of the participants were multigravida

About fifty-seven percent (56.7%) of the pregnant women were never on any contraceptive prior to the pregnancy; whereas 43.3% once used DMPA. Of the PE cases, 80.0% used DMPA, prior to getting pregnant. About twelve percent (11.7%) of the study participants had a family history of hypertension, with the majority amongst the PE cases (**Table 3**). There was IUGR if the estimated foetal weight (EFW) was below the 10th percentile for their gestational age. None of the normal pregnant (NP) women had IUGR, while 50.0% of the PE patients

Table 2. The clinical variables of the study population.

Parameters	PE (n = 30)	NP (n = 30)	P-value
Age (years)	30.50 ± 5.78	29.13 ± 4.98	0.330
Gestational Age (yrs)	34.50 ± 2.83	34.63 ± 2.00	0.834
Weight (Kg)	84.07 ± 21.33	72.20 ± 12.69	0.011
Height m	1.62 ± 0.09	1.56 ± 0.12	0.029
BMI (Kg/m ²)	30.35 ± 5.71	27.93 ± 4.02	0.063
SBP (mmHg)	155.67 ± 15.30	112.33 ± 10.06	<0.0001
DBP (mmHg)	99.33 ± 10.81	73.67 ± 9.64	<0.0001
EFW (Kg)	2.00 ± 0.62	3.00 ± 0.40	<0.0001
BWT (Kg)	2.50 ± 0.36	3.46 ± 0.62	<0.0001
TC (mmol/L)	4.17 ± 1.48	3.64 ± 1.45	0.162
TRG (mmol/L)	1.24 ± 0.84	0.85 ± 0.33	0.024
LDL (mmol/L)	2.92 ± 1.54	2.15 ± 1.03	0.026
HDL (mmol/L)	0.87 ± 0.50	1.31 ± 0.46	0.001
Homocysteine (µmol/L)	32.35 ± 11.08	9.32 ± 2.73	<0.0001

TC: serum cholesterol; TRG: triglycerides; LDL: Low Density Lipoprotein cholesterol; HDL: High Density Lipoprotein cholesterol. Data was expressed as mean ± standard deviation (SD). Independent (unpaired) t-test was to compare means of the clinical variables between the preeclamptic patients and the normal pregnant women.

Table 3. Obstetric history of studied participants.

Parameters	PE (n = 30)	NP (n = 30)	Total (n = 60) PE + NP
OBSTETRIC HISTORY			
Gestational Age at sampling			
<34 weeks	9 (30.0%)	8 (26.7%)	17 (28.3%)
34 - 37 weeks	18 (60.0%)	21 (70.0%)	39 (65.0%)
≥38 weeks	3 (10.0%)	1 (3.3%)	4 (6.7%)
Parity			
Nulliparous	10 (33.3%)	6 (20.0%)	16 (26.7%)
Primipara	6 (20.0%)	11 (36.7%)	17 (28.3%)
Secundipara	4 (13.3%)	9 (30.0%)	13 (21.7%)
Tertipara	6 (20.0%)	3 (10.0%)	9 (15.0%)
Multipara (≥4)	4 (13.3%)	1 (3.3%)	5 (8.3%)
Gravidity			
Primigravida	6 (20.0%)	5 (16.7%)	11 (18.3%)
Secungravida	7 (23.3%)	10 (33.3%)	17 (28.3%)
Tertigravida	4 (13.3%)	10 (33.3%)	14 (23.3%)
Multigravida (≥4)	13 (43.3%)	5 (16.7%)	18 (30.0%)
Type of contraceptive used			
DMPA	24 (80.0%)	2 (6.7%)	26 (43.3%)
No contraceptive used	6 (20.0%)	28 (93.3%)	34 (56.7%)
Family history of HTN			
Yes	5 (16.7%)	2 (6.7%)	7 (11.7%)
No	25 (83.3%)	28 (93.3%)	53 (88.3%)
Previous obstetric history			
No complication	23 (76.7%)	25 (83.3%)	48 (80.0%)
Induced abortion	2 (6.7%)	1 (3.3%)	3 (5.0%)
Spontaneous abortion	4 (13.3%)	3 (10.0%)	7 (11.7%)
Miscarriage	1 (3.3%)	1 (3.3%)	2 (3.3%)
PREGNANCY OUTCOME			
Foetal growth restriction			
Yes	15 (50.0%)	Nil (0.0%)	15 (25.0%)
No	15 (50.0%)	30 (100.0%)	45 (75.0%)
Birth weight			
Normal weight	16 (53.3%)	30 (100.0%)	46 (76.7%)
Underweight	14 (46.7%)	Nil (0.0%)	14 (23.3%)

DMPA: Depot medroxyprogesterone acetate; HTN: Hypertension.

demonstrated IUGR. Babies who weighed < 2.5 Kg at birth were considered underweight babies while those weighing > 2.5 Kg at birth were considered normal weight babies. While none of the NP women gave birth to underweight babies, 46.7% of the babies by PE women were underweight (**Table 3**). Positive correlations for PE were as follows: SBP and DBP ($p = 0.05$; $r = 0.858$); GA and BWT ($p = 0.05$, $r = 0.425$); TG and SBP ($p = 0.05$, $r = 0.457$); TG and TC ($p = 0.05$, $r = 0.397$); TG and LDL ($p = 0.05$, $r = 0.397$); LDL and TC ($p = 0.001$, $r = 0.908$); HCY and DBP ($p = 0.05$, $r = 0.441$). Negative correlations for PE were as follows: GA and EFW ($p = 0.05$, $r = -0.403$); GA and BWT ($p = 0.05$, $r = -0.337$); BWT and DBP ($p = 0.05$, $r = -0.366$); B12 and HDL ($p = 0.05$, $r = -0.368$); Folate and TC ($p = 0.05$; $r = -0.414$).

3.3. Multivariate Logistic Regression of Factors Associated with PE

The use of HC prior to pregnancy was associated with a 30-fold (29.71) increase in the odds of developing PE (**Table 4**).

4. Discussion

4.1. Main Findings

In this study, the risk of developing PE among women who used DMPA, prior to pregnancy was assessed. The findings of this study showed that 80.0% of the preeclamptic patients have at some point in their life-time used DMPA. The use of DMPA prior to pregnancy was associated with about thirty-fold increase in the odds of developing PE (OR = 29.71, $p < 0.001$). The study demonstrated that serum homocysteine and lipid profile levels were significantly altered with the use of HC.

4.2. Interpretation of Results

Depot medroxyprogesterone acetate (DMPA), an injectable progestin-only contraceptive, is a popular contraceptive of choice, particularly for younger premenopausal women because of the ease of use and high compliance. Ultimately, DMPA is a potent contraceptive with consequences [13] [14] [15].

Table 4. Multivariate logistic regression of factors associated with PE.

Variable	OR	(95% CI)	P-value
Contraceptive use			
Yes	26.00	8.12 - 83.25	<0.001
No*	1.00	-	-
Type of contraceptive used			
None*	1.00	-	-
DMPA	29.71	9.01 - 97.96	<0.001

OR: Odds Ratio; CI: Confidence Interval; Reference group.

Estrogens are known to have a cardio-protective influence in women by improving endothelial function and low-density lipoprotein (LDL) concentrations while decreasing concentrations of endothelin-1 (ET-1) and homocysteine. However, DMPA has been shown to counteract the beneficial effects of estrogens in animals and in some studies, in postmenopausal women [13]. DMPA creates a hypoestrogenic effect, hence diminishing estrogen's protective role on blood vessels [14].

Furthermore, it has been indicated that using HC pills can adversely affect homocysteine [16]. As a result, the significant hyperhomocysteinaemia and dyslipidaemia observed in the preeclamptic women might partly be due to the hypoestrogenic effect of DMPA.

Endothelial dysfunction plays a critical role in the pathogenesis of PE [17]. Widespread endothelial dysfunction is hypothesized to mediate the characteristic generalized vasospasm manifesting as hypertension, a central feature of established PE [18]. In a study where the authors evaluated DMPA damage to blood vessels via flow-mediated dilation [(FMD)—FMD measures endothelial function], the data showed significant differences in FMD among DMPA users and non-hormonal contraceptive users as well as combined oral contraceptive (COC) users and non-hormonal contraceptive users [19]. Between the two HC groups, there was no significant difference in FMD. Data of the study group showed that DMPA users also had lower total cholesterol as well as lower LDL-C, whereas non-hormonal contraceptive users had a higher HDL-C. The findings of Meendering and his colleagues [13] support previous literature that suggests that oral progesterone (which may be considered to have properties similar to DMPA) has androgenic effects that are harmful to the cardiovascular system.

Although the antioxidant status and markers of oxidative stress were not assessed in this study, DMPA is known to induce ovarian oxidative stress [20]. A study to determine the protective effect of vitamins C and E on DMPA-induced ovarian oxidative stress *in vivo*, found elevated levels of malondialdehyde and reduced level of superoxide dismutase in DMPA users when compared to the control group [20]. The findings of that study indicate that DMPA increases lipid peroxidation of ovarian tissue. As a result, oxidative stress resulting from the use of DMPA may play a pivotal role in endothelial cell dysfunction in preeclampsia [21].

Hyperhomocysteinaemia is recognized as an important and independent risk factor for cardiovascular diseases [22]. HC increases homocysteine concentration, leading to hyperhomocysteinaemia [16]. Consequently, the presence of significant hyperhomocysteinaemia in the preeclamptic women when compared to normal healthy pregnant women in this study may partly be due to the use of DMPA by majority of the women with preeclampsia prior to pregnancy. This may further have contributed to the development of PE.

Other cardiovascular risk markers were significantly altered in women with

PE than in the healthy normotensive pregnant women in this study. The preeclamptic women demonstrated significant dyslipidemia in comparison with the normal pregnant women. Although studies that assessed lipid profile in pregnant women who used progestin-only contraceptive prior to pregnancy remain scarce, data on women who used Combined Oral Contraceptive (COC) indicated that COC uptake results in a more atherogenic lipid profile by increasing LDL-C and decreasing HDL-C concentrations [23]. It has also been suggested that perhaps free radicals produced during metabolism of hormonal contraceptives change the amounts of atherogenic lipids [24].

Furthermore, weight gain is a commonly perceived side effect of hormonal contraception [24]. Our study showed that the preeclamptic women had a higher BMI than the control group (NP), though statistically insignificant. Given the serious public health problem posed by overweight and obesity worldwide [15], the public health impact of any contraceptive on obesity could be substantial. Weight gain and changes in body composition have previously been reported among DMPA users [25]. In a 36-months longitudinal study of 703 women taking contraceptives, it was observed that DMPA users had a 5.1 Kg weight gain when compared to the 1.5 Kg weight gain among oral contraceptive users and the 2.1 Kg weight gain among non-hormonal contraceptives users [26]. A meta-analysis of maternal BMI and PE showed that the risk of developing PE was doubled with every 5 to 7 unit increase in pre-pregnancy BMI [27]. Moreover, some authors reported that the risk of preeclampsia during pregnancy doubled in overweight women while it was 4.5 times higher in obese women [27]. Consequently, changes in weight resulting from the use of DMPA further increases the risk of developing PE in later life.

4.3. Strengths and Limitations of Study

The small sample size used is a limitation of this study. Nevertheless, this research adds a lot to the existing knowledge, as there is paucity of data on the role DMPA in the aetiology of PE.

5. Conclusion

Overall, the study demonstrated that the use of DMPA prior to pregnancy is a primary cause of PE. DMPA could partly contribute to endothelial dysfunction, hyperhomocysteinaemia, dyslipidaemia and excessive weight gain, all of which contribute to the development of PE. Women who decide to use DMPA should be well educated on the benefits and harmful effects of this contraceptive.

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Contribution to Authorship

All authors collaborated to carry out this work. Authors L. Asare, W. K. B. A. Owiredu, C. Obikorang and G. A. Asare conceptualized and designed the study. Participants recruitment and data generation were carried out by authors L. Asare, W. Tashie, L. Seidu and G. A. Asare. Authors L. Asare, W. K. B. A. Owiredu, C. Obikorang, W. Tashie and G. A. Asare analyzed the data. Authors L. Asare, W. K. B. A. Owiredu, C. Obikorang, W. Tashie, L. Seidu, G. A. Asare drafted the manuscript. Authors L. Asare, W. K. B. A. Owiredu, C. Obikorang, E. Appiah and G. A. Asare reviewed the manuscript for intellectual content and each author approved the final manuscript.

Ethical Approval

Ethical approval for the study was granted by the facility authorized committee in a letter (KAD/CHS/ADM/026/2014]).

Consent

Informed consent was obtained from participants before enrollment into this study.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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Appendix A

Research Questionnaire

Personal History

Identification number: Date of visit:/...../.....
 Name:..... Age: years.
 Residential area: H/No:
 Health Facility: Mobile No:
 Marital Status: Educational background:

Obstetric History

Gestational Age:..... (Weeks). Expected Date of Delivery:
 Parity: Gravidity:
 Weight:(Kgs) Height: (M)
 Body Mass Index: Blood pressure: (mmHg)
 Have you been pregnant before? Yes No
 If yes, number of pregnancies..... Miscarriages:
 Still births, Induced abortion Abortion:
 Preterm delivery, Live births, Multiple births
 Birth weight
 Intrauterine Growth Restriction

Medical History

Do you have any of these?

Pre-eclampsia	Yes <input type="checkbox"/>	No <input type="checkbox"/>
Heart disease	Yes <input type="checkbox"/>	No <input type="checkbox"/>
Hypertension	Yes <input type="checkbox"/>	No <input type="checkbox"/>
Kidney disease	Yes <input type="checkbox"/>	No <input type="checkbox"/>
Liver diseases	Yes <input type="checkbox"/>	No <input type="checkbox"/>
Diabetes mellitus	Yes <input type="checkbox"/>	No <input type="checkbox"/>
Sickle cell disease	Yes <input type="checkbox"/>	No <input type="checkbox"/>
Severe anaemia	Yes <input type="checkbox"/>	No <input type="checkbox"/>
Folic acid deficiency	Yes <input type="checkbox"/>	No <input type="checkbox"/>
Vitamin B ₁₂ deficiency	Yes <input type="checkbox"/>	No <input type="checkbox"/>

Have you ever being diagnosed of any of the above condition before this pregnancy? Yes No
 If yes which?.....
 When was the first time you were diagnosed of this condition?
 Are you currently on any medications or injections? Yes No
 If yes, what are they?
 Any other health complication?
 Do you have any history of alcoholism? Yes No
 Have you currently drunk alcohol? Yes No
 If yes, how long did you drink?
 Have you ever smoked? Yes No

If yes, how long did you smoke?

Have you ever used any contraceptive prior to this pregnancy?

Yes No

If yes which?

Family History

Do you have family history of any of the following conditions?

- Pre-eclampsia Yes No
- Heart disease Yes No
- Hypertension Yes No
- Kidney disease Yes No
- Liver diseases Yes No
- Diabetes mellitus Yes No
- Sickle cell disease Yes No
- Multiple pregnancies Yes No
- Birth defects (specify) Yes No

.....

Nutritional Data

How often do you eat the following foods		None	Daily	Twice a week	Weekly
I	Meat				
II	Fish				
III	Green leafy vegetables				