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Effects of Malaria and Hypertensive Diseases in Pregnancy on Foetal and Placental Morphometric Outcomes in Niger-Delta Region of Nigeria

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ABSTRACT

Introduction: available evidence suggest that malaria and hypertensive diseases in pregnancy affect placental morphometrics which impacts on fetal outcomes. Literature search did not reveal normative data for placental and fetal morphometry in this setting

Methods and Methods: a comparative cross-sectional study of two cohorts of women examined in labour as cases and controls. The cases were consecutive consenting parturients, with pregnancies complicated by hypertension and malaria and the controls were parturients with uncomplicated pregnancies. The birth weight, length of babies, placental weight, and diameter were measured.

Results: normative data showed average birth weight of 3318.16 +523.37g, fetal length was 54.39 + 4.45cm, placental weight was 618.18 + 122g, umbilical cord length was 59.96 + 12.65cm.

Pregnancies complicated by hypertension produced average birth weight of 3200 + 612.37g, fetal length was 53.38 + 5.65cm, placental weight was578.38 + 105.55g, and umbilical cord length was 53.38 + 23.40cm. The differences were statistically significant $p < 0.05$. Parturients whose pregnancies were complicated by malaria had birth weight: 3285 + 445.18g, fetal length: 54.95 + 3.46cm, placental weight: 597.80 + 125.76g, umbilical cord length: 60.55 + 11.00cm.

In pregnancies where malaria and hypertension were co-morbidities, birth weight: 3490 + 392.85g, fetal length: 56.40 + 3.06cm, placental weight: 671.43 + 101.89g, umbilical cord length: 70.60 + 0.03cm. The differences were statistically significant $p < 0.05$.

Discussion : hypertensive diseases and malaria, each, was associated with reduction of weight and size of babies and placentae, and this is believed to be mediated by placenta insufficiency. When these diseases occurred in the same pregnancy, we saw a paradoxical increase in weight and size of babies and their placentae. More studies are needed to evaluate this observation.

Keywords: Placenta; Hypertension; Malaria in pregnancy.

Introduction

Hypertensive disorders, infection (in our environment malaria is the commonest) and haemorrhage complicating pregnancy constitute the deadly triad resulting in maternal morbidity and mortality, related to pregnancy.[1]

The placenta is the first of the foetal organs to develop and has several critical functions including the supply of nutrition and maintenance of gaseous exchange for the foetus. All prenatal and peripartum injury can be explained by some combination of 3 basic mechanisms

1. Genetic causes of abnormal structures e.g karyotypic abnormalities, single gene defects, polygenic diseases or other genetic abnormalities e.g peroxisomal and mitochondrial disorders , imprinting disorders

2. Developmental abnormalities affecting fetoplacental reserve such as inadequate maternal supply line, nutritional deficiencies or abnormalities in the maternal immune response to pregnancy.

3. Stochastic (random) events causing specific damage such as exposure to teratogens, infections, vascular accidents or membrane rupture.[2,3,4]

Post delivery study of placental morphology helps explain 3 primary perinatal outcomes from the standpoint of prenatal and peripartum injury viz: preterm delivery, fetal growth restriction and hypoxic ischaemic injury.[5] This study focussed on effects of two maternal diseases (which can affect the placenta) on foetal and placental morphometric parameters. The diseases are hypertension and malaria.

Grossly, two different types of placental changes occur in preeclampsia and ecclampsia. The first and most common is a small placenta, often delivered prematurely, multiple infarcts and decidual vasculopathy, especially acute artherosis are usually present, and in severe cases the villi are small and slender in a pattern called accelerated maturation or distal villous hypoplasia. The second type is a large placenta with abundant villous cytotrophoblast that occurs mainly in association with diabetes mellitus and

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multifocal gestation. Preeclampsia occurs only in the presence of trophoblast, with or without the presence of a foetus. The treatment only improves symptoms but the condition itself can only be eliminated by delivery of the placenta.[2] According to Lachmeijer *et al*., 2002, there appears to be a genetic predisposition to preeclampsia/ecclampsia transmitted by both maternal and paternal routes.[6] Brosens *et al*. in 1972 reported that the mean diameter of myometrial spiral arterioles of 50 normal pregnant women was 500um.[7] The same measurement in 36 women with preeclampsia was 200um. Studies done in Bangladesh found that compared with controls, there was a trend of statistically significant lower weights and volumes of placenta in ecclamptic women.[8]

Malaria is the commonest infectious disease in the world, but congenital disease is not often reported.[9] It is the commonest cause of fever in pregnancy in Nigeria. Four species of *Plasmodium* that cause human malaria are *vivax*, *ovale*, *malariae and falciparum*. By far the most malignant of these is *Plasmodium falciparum*.[10] Pregnancy enhances the severity of *falciparum malaria*, especially in non immune nulliparous women.[11,12,13] Increased foetal loss may be related to placental and foetal infections. Placental reactions to malaria is characterised by clogging of the intervillous space by macrophages and the extent of reaction is proportional to the severity of infection.[14] Parasite sequestration in the placenta is believed to trigger a pathological process, that contributes to decreased foetal viability that leads to low birth weight and placental weight.[15] Achidi *et al*., in 2005 found that neonates born from parasite-positive placentae had a significantly higher incidence of low birth weight (13.5%) than those from parasite negative placentas (6.8%).[16] Using a murine model to study the pathogenesis of severe malaria in pregnant women, Neres *et al*., in 2008 found significant thickening and disorganisation of labyrinthine, distension and disarrangement of perivascular space as well as presence of parasitized red blood cells in the maternal blood space.[17] Further analysis with a computerised morphometric method showed that the average sinusoid space fell from 52.0$\pm$ 4.0 in the control group to 34.7$\pm$7.5 in the infected group. Thus supporting earlier belief that the effects of malaria on the neonate are caused by placental insufficiency.[18] The outcomes of placental insufficiency are expected to be seen as restrictive effects on foetal and placental growth.

Placenta morphometrics most studied include placenta weight, thickness and diameter, of these, only placenta weight have been shown to vary with ethnicity even in cases where there was no significant difference in birth weight.[19] It also has a significant role in foetal growth in terms of weight, body length, and cord length,[20] and several studies have shown that low placental weight is associated with IUGR.[21,22] Several cohort studies have shown evidence of a correlation between placental weight and placental to birth weight ratio and future development of chronic diseases especially hypertension and diabetes in adult life.[23,24,25,26,27]

Hypertension in an offspring later in life has been associated with reduced placental weight and lower fetoplacental ratio in the offspring. This association of hypertension later in life with low placental weight was accompanied by reduced placental surface area but not reduced thickness.[23,35,36,37] This association highlights the necessity of routine placental morphometric measurement for every child so that preventive risk assessment counselling can be given to mother as regarding other risk factors for development of hypertension later in life including obesity and cigarette smoking , routine post natal examination of the placenta can hence assume a significant part of preventive health care.

We could find no record of any study done, documenting the effect of these two, occurring as co-morbidities in the mother, on the placenta. Clearly, there is a dearth of normative data on the morphology of the placenta, little is known about the structural changes in the placenta which are compatible with health or disease, and very little is known about the effects of these common complications of pregnancy on the morphology of the fetus and placenta in the Niger-Delta region of Nigeria.

This study was therefore undertaken to document and compare placental and foetal morphometric indices in uncomplicated and uncomplicated pregnancies in Central Hospital Warri, located in the Niger-Delta region of Nigeria. We believe that the information obtained in this study will provide baseline data on morphology of the placenta in Delta state, which will aid the understanding of perinatal morbidity in the study area. This will also aid medicolegal adjudication in cases involving perinatal morbidity and mortality.

Methods and Methods

It was comparative cross-sectional study of two cohorts of women examined in labour in the delivery suites of Central Hospital Warri, as cases and controls. The cases were consecutive consenting parturients, with pregnancies complicated by hypertension, malaria and or both, and the controls were parturients with uncomplicated pregnancies matched for parity, maternal age, maternal height and gestational age. The birth weight, length of babies, the placental weight, and diameter as well as the umbilical cord length were measured, whilst the placenta and feto-placental ratio was calculated. Central Hospital Warri is located between latitude 5 36^0 and 6^0 north of the equator and longitude 5 30^o and 6^o east of the Meridian. It is one of the secondary health care facilities in the Niger-Delta region of Nigeria. The Maternity unit is overseen by 4 Consultant Obstetricians and provides ante natal care

and labour management of pregnant women with an average of 4500 deliveries per year. It has a maternal mortality ratio of 2,232/100,000 live birth.[28] Common complications seen here in pregnant women include malaria and hypertensive diseases of pregnancy, and the latter is a common cause of maternal mortality.[28]

Selection Criteria

Inclusion:

1. Booked parturients with documented weight, height, parity and ethnicity at the booking clinic
2. Unbooked parturients presenting for delivery and had their weight, height, parity and ethnicity documented
3. Singleton pregnancies
4. Spontaneous vaginal deliveries with complete placental delivery or suitably delivered placentae from caesarean section
5. Preterm/premature delivery or spontaneous miscarriage with wholesome recovery of the placentae
6. Normal/uneventful pregnancies
7. Pregnancies complicate by:
    a. Hypertensive diseases
    b.Malaria

Exclusion:

1. Parturients with incomplete placenta at delivery
2. Parturients with undocumented weight, height, parity and ethnicity
3. Multiple pregnancies
4. Pregnancies complicated by diabetes
5. Pregnancies in which the parturient has sickle cell disease, cardiac disease, renal disease.

Specifically, the indices measured included:

- **Placenta:** weight, diameter, thickness, volume, surface area, placental weight index (feto-placenta ratio).
- **Umbilical cord:** length, insertion, number of umbilical arteries, cord-foetal length ratio.
- **Fetal:** birth weight, fetal length, fetal body mass index.

Placenta weight and birth weight was measured using the Docbelbraun infant weighing scale by docbel industries japan manufactured in 2000. Infant length, cord length and placenta diameter were measured using a tape rule and recorded in cm. In addition, site of cord insertion, presence of single umbilical artery if present was also recorded. Placenta diameter was measured using the maximal diameter of the surface since the placental surface is more oval than circular and placenta weight index (PWI) was estimated by dividing the placental weight by the fetal weight; vice versa was done to get the fetal placental ratio.

Parturients who presented for labour were selected consecutively. The purpose and procedure of the study was explained in detail including the benefits of the study, no harm to mother and child, and the mothers' liberty to withdraw from the study at any time she chose. Those who gave written informed consent and fulfilled the inclusion criteria were recruited.Ethical approval for the study was obtained from the Ethics Committee of the College of Health Sciences ,Delta State University Abraka (FBMS/2006/32), permission was also sought and obtained from the Management of the General Hospital Warri for the study to be undertaken in their facility.

Ante-natal records of consenting subjects were accessed to obtain demographic and clinical data. Singleton pregnancies with no documented complications, those with documented history of malaria in pregnancy (≥2+ MP on blood smear) and parturients with documented hypertensive disease of pregnancy were followed up to delivery. Their babies and placentae were studied. A total of 131 parturients were studied and of this total, 32.82% were cases and 67.17% were controls. This sample size was obtained using the standard formula for comparative cross-sectional study.[29]

Following resuscitation where necessary immediate post-delivery, the unclad newborn is weighed to obtain the birth weight and the length measured. These were documented for each parturient in a specific data collection proforma sheet designed for the study.

Placentas were examined within an hour of delivery and the following indices recorded, with the placenta on a flat surface. The placenta was first inspected, the shape, colour of maternal surface (CMS) was noted, and then the presence of accessory lobes, infarcts, tumors, nodules, were noted and recorded. For the placenta membrane the Colour, lustre and velamentous vessels if present were s recorded and noted. The cord and membranes were cut from placenta before weighing.[30] The diameter of the placenta, and the length of the umbilical cord were also measured.

All data derived from this study as recorded in the proforma sheet were collated, coded and fed into the computer using the Statistical Package for Social Scientists (SPSS PC+, SPSS Inc., Chicago Ill, USA), and analysed. Univariate and bivariate analysis were done as appropriate, and test of statistical significance was based on 95% confidence interval and a p-value of $P<0.05$ using students t-test or chi-squared test. Yates or Fischer correction were done when necessary.

Results

A total of 131 parturients were studied. Uncomplicated pregnancies constituted 67.18% (88) and complicated pregnancies constituted 32.82% (43). Malaria in pregnancy occurred in 45.24%, hypertensive diseases occurred in 30.88% and malaria and hypertensive diseases in 23.81%.

Table 1. Comparison of morphometric features in uncomplicated (control) pregnancies and pregnancies complicated by hypertensive diseases alone.

Parameter	Uncomplicated (Control)	Hypertensive diseases	Significant difference at P<0.05
Mean birth weight (g)	3316.18 + 523.37	3200.00 + 612.37	Significant
Mean foetal length (cm)	54.39 + 4.45	53.38 + 5.65	Not significant
Mean placental weight(g)	618.18+122.28	578.38+ 105.55	Significant
Mean placental thickness (cm)	1.91 + 0.48	1.99 + 0.42	Not significant
Mean placental diameter (cm)	18.91 + 2.89	18.74 + 2.07	Significant
Mean umbilical cord length (cm)	59.96 + 12.65	53.58 + 23.40	Not significant
Mean placental surface area (cm2)	574.61 + 182.09	557.85 + 121.73	Significant
Mean foeto-placental ratio	5.47 + 0.86	5.72 + 1.45	Significant
Mean umbilical-foetal ratio	1.11 + 0.22	0.99 + 0.45	Not significant

Table 2. Comparison of morphometric features in uncomplicated (control) pregnancies and pregnancies complicated by malariaalone.

Parameter	Uncomplicated (control)	Malaria	Significant difference at P<0.05
Mean birth weight (g)	3316.18 + 523.37	3285.0 + 445.18	Significant
Mean foetal length (cm)	54.39 + 4.45	54.95 + 3.46	Significant
Mean placental weight (g)	618.18+ 122.28	597.82+ 125.76	Not significant
Mean placental thickness (cm)	1.91 + 0.48	2.03 + 0.49	Significant
Mean placental diameter (cm)	18.91 + 2.89	18.86 + 2.17	Significant
Mean umbilical cord length (cm)	59.96 + 12.65	60.55 + 11.00	Significant
Mean placental surface area (cm2)	574.61 + 182.09	565.55 + 132.66	Significant
Mean foeto-placental ratio	5.47 + 0.86	5.44 + 0.80	Significant
Mean umbilical-foetal ratio	1.11 + 0.22	1.10 + 0.19	Not significant

Table 3. Comparison of morphometric features in uncomplicated (control) pregnancies and pregnancies complicated by hypertensive diseases and malaria.

Parameter	Uncomplicated (control)	Hypertensive disease and fever (malaria)	Significant difference at P<0.05
Mean birth weight (g)	3316.18 + 523.37	3490.00 + 392.85	Significant
Mean foetal length (cm)	54.39 + 4.45	56.40 + 3.06	Significant
Mean placental weight (g)	618.18+ 122.28	671.43+ 101.89	Significant
Mean placental thickness (cm)	1.91 + 0.48	2.06 + 0.46	Significant
Mean placental diameter (cm)	18.91 + 2.89	20.10 + 2.60	Significant
Mean umbilical cord length (cm)	59.96 + 12.65	70.60 + 0.03	Significant
Mean placental surface area (cm2)	574.61 + 182.09	644.27 + 170.26	Significant
Mean foeto-placental ratio	5.47 + 0.86	5.49 + 0.87	Not significant
Mean umbilical-foetal ratio	1.11 + 0.22	1.25 + 0.14	Significant

Discussion

The birth weights observed was largely normally distributed irrespective of pregnancy complications studied and showed average normal birth weight (2.5kg-3.9kg) despite hypertensive diseases or malaria in pregnancy. The effect of good antenatal care is highlighted by the observation that despite the distress imposed by complications studied, 93.75% of babies weighed at least 2.5kg.

Birth weight as an anthropometric variable has been shown to vary with race and altitude. India is reported to have one of the highest levels of low birth weight in the world.[31]All the babies were African.

The reduction effect of hypertensive diseases and malaria in pregnancy on birth weight as seen in this study is most probably mediated via the placental

insufficiency associated with these complications, more so with hypertensive disease in pregnancy.[2] It was observed in this study that all cases of large birth weight (>4kg) in complicated pregnancies involved malaria. In fact, where malaria and hypertensive disease occurred together in pregnancy, the babies were significantly heavier and bigger at birth. A possible explanation is that Malaria in pregnancy appears to counteract the insufficiency occasioned by hypertensive diseases. We could find no study which had reported this observation. The exact reason(s) for this we could not fathom, and it is a basis for further research to unravel how synergistic effect of both disease entities is associated with increased new-born's weight.

A new-born's length is a measure that indicates how big a baby is. In this study approximately 80% of babies measured 50-59cm long (mean 54.53±4.16cm). Whereas hypertensive diseases in pregnancy is associated with significantly smaller babies, malaria appeared to have the opposite effect such that malaria and hypertensive diseases co-existing, was associated with significantly larger babies. The increase in length occasioned by malaria is obviously relatively higher than the increase in fetal weight, this led to an overall reduction in body mass index as observed. A possible explanation for this phenomenon of apparently counteracting effects of malaria on hypertensive diseases in pregnancy is an alteration in the permeability of nutrients across the placenta such that soft tissue growth is restricted in favour of skeletal growth. This would lead to our observation of markedly increased length but a marginally increased weight with overall lower body mass index. The BMI in babies whose gestation was complicated by hypertensive remained about the same as in uncomplicated pregnancies (11.23kg/m^2 vs 11.21 kg/m^2) but in babies whose gestation was complicated by malaria fever alone and malaria co-existing with hypertensive diseases, it was observed that there was a net drop in BMI (10.88kg/$m^2$and 10.97 kg/m^2 respectively). This suggests that whereas, hypertensive disease in pregnancy is associated with smaller and lighter babies' malaria is associated with lighter but longer babies.

Placental weight observed in this study in uncomplicated pregnancies was less than the finding at Birmingham in U.K even among afro carribeans who are residing there and Chinese babies.[20,32] It agrees with the finding at Lithuania but higher than the finding in India and even south western Nigeria.[22,26] Environmental influences especially nutritional status would account for this variation more than race or ethnicity. However in individuals from the same environment, ethnicity and race has been shown to influence variation in the normal term placenta.[19,32] As placental weight is usually positively correlated with birth weight as seen in this and other studies,[20] the variation in placental weight is also usually mirrored in birth weight.

The mean placenta weight was significantly ($p<0.05$) reduced in pregnancy complicated by hypertensive diseases. This is in agreement with the work in India.[22] According to Kraus *et al* 2004, small placenta is expected in such pregnancies.[2]

The reduction in placental weight seen with hypertensive diseases in pregnancy is attributable to a reduction in placental exchange membrane which has been dubbed a definition of placental insufficiency.[33] It also probably involves a restriction of the physiological hyperplasia as recorded by Maynaw& colleagues.[34] Infact peripheral villous hypoplasia has been attributed to severe uteroplacental under perfusion culminating in restriction of placental growth and further decrease in feto-placental vascular bed.[35]

These changes all lead to reduced uterine blood flow with attendant changes in the placenta and reduced birth weight as seen in this study. This Increases the risk of later life hypertension in the offspring.[36,37,39,40]

As expected from literature, both birth weight and placental weight from pregnancies complicated by malaria was significantly lower than in uncomplicated pregnancies.[9] This is probably due to the attendant placental sequestration of *Plasmodium falciparum* trophozoites and inflammation.[18] It has also been demonstrated that malaria parasites may still remain sequestered in the intervillous spaces after apparently adequate therapy.[9] The reduction in placental weight seen here was not statistically significant and this is probably due to the significant placental tissue thickening and reduced blood sinusoid space secondary to the prominent inflammatory response in the placenta.[17] It has been suggested that the malaria parasite is unlikely to be directly responsible for the pathology observed with placental malaria but leukocytes through the production of non chemotatic cytokines might occasion the thickening of trophoblast membranes.[41] The changes in fetal and placental morphometry with malaria seen in this study can therefore be said to be due to combination of placental insufficiency secondary to reduction of maternal sinusoids spaces, fibrinoid deposition, haemosiderin deposition and placental inflammation with attendant thickening of trophoblast basement membrane.

In women whose pregnancies were complicated by hypertensive disease and fever, we found significantly heavier placentae, even though the feto-placental ratio was not significantly different from that in uncomplicated pregnancies. There is a paucity of studies on the combined effects of placental malaria and hypertensive diseases in pregnancy. Since feto-placental ratio is not significantly different in thecomplicated and uncomplicated pregnancies (5.49±0.87 vs. 5.47±0.86), it suggests a match in the increase in both placental weight and birth weight. This is possible if there is a uniform reversal of the placental insufficiency caused on the one hand by reduction in placental exchange membrane seen

in hypertensive diseases in pregnancy and on the other hand, the thickening of trophoblastic basement membrane seen in placental malaria. It is suggested that both effects seem to cancel each other leading to enhanced placental and foetal growth. Another possible reason for this observation maybe the much-enhanced health care and attention these women must have received because of the existence of co-morbidities in pregnancy in a secondary healthcare facility having qualified and competent obstetricians. We hypothesize that it is possible that placental malaria and hypertensive diseases coexisting may cause the release of a new factor which counteracts the effects of each and encourages foetoplacental growth. More detailed studies are needed to evaluate this phenomenon.

Placental thickness has been variously studied as an ultrasonographic index for estimating fetal weight.[42,43] In this study, placental thickness showed a unique distribution, whereas it showed no significant difference between placentae from uncomplicated pregnancies and that from pregnancies complicated by hypertensive disease it showed a significant increase in placentae from pregnancies complicated by malaria and where both placental malaria and hypertensive diseases had occurred. It had earlier been known that villous inflammation as seen in placental malaria is associated with increased thickness of trophoblast basement membrane, hence it follows that placental malaria that placental is associated with increased thickness. Yet apparently the placental insufficiency associated with hypertensive disease affects villous branching more than growth of main villous stem. Inhibition of placental villous branching would occasion a restriction of both placenta weight and diameter as seen in this study in cases where there is hypertensive disease alone or malaria alone.

Yet where both diseases occurred, a possible reversal of insufficiency or a cancelling out effect or a new factor effect leads to enhanced growth in both main stem and branches. This hypothesis would explain the effects seen here. How these complications would preferentially restrict villous branching but not main stem growth needs to be evaluated by further studies.

Placental diameter is a measure of the surface area available for exchange. The mean placental diameter in pregnancies complicated by hypertensive diseases was significantly lower than from uncomplicatedpregnancies. This trend is in concordance with the finding in Bangladesh.[8]

The significant reduction in weight and diameter but not in thickness suggest placental growth restriction focused more on branching villi and less on the main stem villi.

The reduction in placental surface area which has been associated with hypertension in offspring later in life also implies reduced placental diameter.[38] Mothers with hypertensive diseases has been shown to have increased probability (almost two times) of producing placentae which are restricted for placental weight and chorionic plate areas.[44] This leads to disproportionately small placentae associated with hypertensive diseases in pregnancies which probably results from poor nutrient supply or hypoxia and leads to foetal growth restriction.[44,45]

The mean placental surface area in pregnancy complicated with hypertensive diseases was significantly lower than that in uncomplicated pregnancies. This follows from a reduced placental diameter and is in agreement with the findings at India.[46]

The reduced placental surface area seen in hypertensive pregnancies and the accompanying reduced placental weight has been associated with increased risk of hypertension in the offspring later in life.[23,36,37,47] This association between hypertension and placental size is probably initiated through restricted placental growth in the weeks after implantation resulting in reduced placental area that limits transfer of nutrients to the baby.[47]

A woman who had hypertension during her pregnancy with these attendant placental abnormalities gives birth to a baby who has an almost double risk of developing hypertension later in life. Routine examination of the placenta in addition to good antenatal care would isolate babies who carry such risk. These children and parents can then be advised to consciously reduce other risk factors for developing hypertension later in life for example obesity, hyperlipidaemia and cigarette smoking. This gives routine morphometric examination of the placenta not just forensic value but intrinsic value in preventive health.

The mean foeto-placental ratio in pregnancies complicated by hypertensive diseases was significantly higher than in uncomplicated pregnancies. The higher foeto-placental ratio in hypertensive pregnancies was also seen in a study at India.[46] The increase in foeto-placental ratio in hypertensive pregnancies is to be expected since we have already seen a reduction in birth weight in hypertensive pregnancies, that apparently the growth restriction affected the placenta more than it affected the foetus. Hypertension in the offspring in later life has been shown to be more common in low birth weight with associated reduced placental weight and placental surface area as in this case.[36,37,47]At the other end of the spectrum it is also associated with cases of high placental weight relative to birth weight. - High placental ratio.[23,37] These associations are thought to reflect foetal progressing which is a process through which foetal malnutrition and consequent small size leads to lifelong changes in body system and organs in ways which lead to diseases in later life.[47] Our search show these associations have not been conducted in Nigeria as records of birth and placenta dimensions are

not usually kept well in hospitals. This morphometric parameter should be ascertained for every child born whose pregnancy was complicated by hypertensive diseases so that preventive health counselling can be offered to the mother.

The umbilical cord length did not show any significant difference with hypertensive disease in pregnancy and uncomplicated pregnancy but with malaria in pregnancy it showed significantly longer cords, with longer but lighter babies.[48]

In conclusion, the babies were mostly normal birth weight both in complicated and uncomplicated pregnancies but there was significant variation of birth weight with maternal disease of hypertension and malaria in pregnancy. The placenta also showed gross significant variation with hypertensive disease and /or malaria in pregnancy.

Hypertensive diseases in pregnancy was associated with smaller, lighter placenta, higher feto-placental ratio and no significant change in umbilical cord length.

The association of lower placental weight and smaller placental surface area with increased risk of hypertension and early cardiovascular death later in life,[27,46] makes it imperative to recommend that placenta be examined at birth as a surveillance tool for risk of cardiovascular disease especially hypertension, later in life. This would enable counselling of parents and later the children on avoidance of other risk factors like obesity and cigarette smoking.

As might be expected, we observed a reduction in birth weight, placental weight , placental diameter in both hypertensive disease and malaria but when both occurred the effect was a paradoxical and significant increase in birth weight, placental weight and placental diameter suggesting a cancelling effect or release of a foeto-placental growth enhancing factor. This phenomenon is intriguing and more studies are needed to evaluate it.

It was also observed that the placental growth restriction seen in malaria in pregnancy differed from that in hypertensive disease in that it tended to affect villous branching more than growth of stem villi. More studies to elaborate this are needed.

References

1. Cunninghams FG, Gant NF, Levensleueno KJ, Gilstrap III, L.C, Hauth JC, WenstormKD,(EdS) Williams obstetrics 21st ed . 2001 macgraw Hill, U.S.A pp154.
2. Kraus F.T , Redline RW, Gersell DJ, Nelson D.M , Dicke JM, 2004, placental pathology , Atlas of non tumour pathology first series fascile 3, amarican registry of pathology ,U.S.A. pp283-285.
3. Allen WR, Skidmore JA, Stewart F, Antczarkde. Effects of fetal genotype and uterine environment on placental development in equids. J. Reprod. Fertile 1993;98:55-60.
4. Altshuller G and Hyde S, 1996, clinicopathologic implications of placenta pathology. Clin. Obstet. Gynaecol 39:549-570.
5. Baghado-Singh RO, Dashe J, Deran O, Daftary G, Copel JA, Ehrenkranz RA. Prenatal predictions of neonatal outcome in the extremely low birth weight infant. Am j. ObstetGynaecol 1998; 178:462-8.
6. Lachmeijer AM, Dekker GA, Pals G,Aanoudse JG, ten kate LP, arngrimsson R, searching for preeclampsia genes : the current position .Eur J ObstetGynecolReprodBiol 2002:105: 94-113.
7.Brosens I and Dixon H. G, 1963, the anatomy of the maternal side of the placenta. Br J ObstetGynaecol 33:357.
8. Sultana S,Hossain GA, Rahman H, Hassan N, Mannan S, Zannat S. Gross morphometry o human placenta in eclampsia . mymensingh med J. Jan 15(1):10-4 2006.
9. Bernischke K, Kaufmann P, Baergen R pathology of the human placenta , 5th ed, Springer , U.S.A 2006,pp722-724.
10. Health organisation, Geneva, Switzerland. WHO/CD/CSR/EPH/2002. WHO.,2008. Nigeria: World malaria report. http:www.who.int/malaria/wmr/2008-sumkey-EN.pdf
11. Nathwani D, curie PF, Douglas JG, Green ST, Smith NC:plasmodium falciparum malaria in pregnancy a review Br J. ObstetGynaecol 99:118,1992.
12. Robier c, Tall A, diagne N, FontanilleD,Spiegel A, Trape JF: plasmodium falciparum clinical malaria: lessons from longitudinal studies in Senegal . parasitologia 41:255,1999
13. Menendez C,Ordi J, Ismail MR, Ventura PJ, Aponte JJ, Kahigwa E, Font F, Alonson PL: The impact of placental malaria on gestational age and b irthweight .J infect Dis 181 : 1740,2000
14.Ngassa P.C 2004, power point presentation post graduate research training in reproductive health faculty of medicine . BMJ 320:490, 2000.
15. McgregorIA , Wilson ME , Bilemicz WZ. Malaria infection of the placenta in the Gambia , West Africa ; its incidence and relationship to still birth and placental weight. Trans R.soc Trop med Hyg 1983:77: 232 -44.
16. Achidi EA, Anchang J.K, Minang JT, Boyo M Achimbom,mokube J.A , Marita T.B , the effect of maternal umbilical cord and placental malaria parasitaemia on the birth weight of newborns from southwestern Cameroon, Acta paeditrica Vol 94, issue 7, july 2005 pp 917-923.
17. Neves S, Marinho CRF, Goncalves LA, Caterino MB, penha – Gonclaves C, pregnancy outcome and placental pathology in plasmodium bergheiAnka infected mice reproduce the pathogenesis 2008;doi :10. 1371/journal. Ponem0001608.
18. Brabin BJ, kalanda JF, verhoeff FH, Chimsukha LH, broadhead RL. Risk factor for foetal anaemia in a malarious area of Malawi . Ann trop paediatric.2004;24:311-321.
19. Premoli ACG, Sala MA, Matheus M , Lopes RA: Influence of ethinicity on the term placenta . The internet journal of gynaecology and obstetrics. 2006. Volume 6 number 1.
20. Lo YF, Jeng M.J,LEE Y-S, Soong .W.J, Hwang B. Placenta weight nd birth characteristics of healthy singleton newborns Acta paediatrica Taiwania,2002, Vol 43, no 1, pp21-25 .
21. Mardi K,Sharma histopathological evaluation of placentas in iugr pregnancies . Indian J. Pathol microbial 2003 : 46(4):551-4
22. Meejun G, 2005, influence of placental size on Gross abnormalities on IUGR in high risk pregnancies ; Acta medica lithanica, 2005 vol12 no 2 p 14-19.
23. Barker DJP, Bull AR, Osmond C, Simmons SJ. Foetal and placental size and the risk of hypertension in adult life . BM J 1990 ; 301: 259-62.
24. Williams LA, Evans SF, Newnham JP. Prospective cohort study of factors influencing the relative weights of the placenta and the newborn infant. BMJ 1997;314:1854-68.
25. LittleaRE,Zadorozhnagab TD, HulchiyeOP,Mendel NA, Shkyryak-NyzhnkbZA,ChyslorskabN,GladeneBC. Placental weight and its ratio in a Ukranian city, early Hum.Dev. vol 71 issue 2, pages 117-127,2003.
26. Adebami O.J, OwaJ.A ,Oyedeji G.A . factors associated with placental weight and placental weight /birth weight percent (placental ratio) among mothers in Ilesha, southwestern Nigeria international journal of tropical medicine 2(2): 68-73, 2007.
27. Risnes KR, Romundstad PR, WilsenTIL,Esklid A, Vatten J placental

weight relative to birthweight and longterm cardiovascular mortality: findings from a cohort study of 31,307 men and women. Am.j.epidermiol 2009 170 (5) :622-651 doi :10.1093/aje/kwp/182
28. Ebeigbe PN, Igberase GO, Aziken ME. Hypertensive disorders in pregnancy experience with 442 recent consecutive cases in Benin city Nigeria. Nig. Med J 2007;48:4.
29. Dahiru T, Aliyu A, Kene TS. Statistics in Medical Research: Misuse of sampling and sample size determination. Ann Afr Med. 2006; 5(3): 158-161.
30. Bernirschke K. The placenta : how to examine it and what you can learn –contempobst and gynaecol, 1981:17:117-119
31. Subramamyam MA, Ackerson LK, Subramanian SV. Patterning in birthweight in India: analysis of maternal recall and health data card. Plos one 5(7):e11424.doi:10:1371/journal pone.0011424.
32. Perry I.J,D.G.Beevers, P.H. Whincup and D. Barefora, 1995. Predictors of ratio of placenta weight to foetal weight in multiethnic community. BMJ., 310:436-439.
33. Becker V, RockleinG . pathology der weiblichengenitalorgone I. Pathologie der plazentz und des abortes . springer –verlag ,Heidelberg,1989 . pp476-477.
34. MayhewT.M ,Wadrys E, Simpson RA : the proliferative vessels hypertrophic growth in tissue subcompartments of human placental villi during gestation J.anat. 184:535-543,1994.
35. Tochos T, Sciamme A, Piccoli E, Guiot c, Kaufmann P,Kingdom J. Umbilical Doppler waveforms and placental villous angiogenesis in pregnancies complicated by foetal growth restriction obstegynaecol 1999;93:499-503
36. Campbe.l DM, Hall MH, Barker DJP.CrossJ,Shell AW, Godfrey KM(1996) diet in pregnancy and the offspring's blood pressure 40 years later. Brit J. Obstet. Gynaecol103:273-280
37. Erikson JG,Forsen TJ, TuomichetoJ,OsmondJC,Barker DJP (2000). Foetal and childhood growth and hypertension in adult life . hypertension 36:790-794.
38. Godfrey KM, and Barker DJP. Fetal nutrition and adult disease. Am J Clin Nutr 2000;71:13488-13528
39. Levine RJ, Lam C, Quian C, .Yu KF, Maynard SE, Schch BP. Soluble endoglins and other circulating antiaging factors in pre-ecclampsia. N Engl J Med 2006;355(10):992-1005.
40. Redline RW,2008. Placental pathology : a systematic approach with clinical correlations. Placenta vol 29 supplemen, pgs86-91
41. Matteeli A, Caligaris S, Castelli F, Carosi G. The placenta and malaria. Ann Trop Med Parasitol 1997;91(7):803-10
42. Habib FA. Prediction of low birth weight infants from ultrasound measurements of placental diameter and placental thickness. Ann Saudi Med 2002;22(5-6):312-4.
43. Mital P, Hooja N, Mehndiratta K. Placental thickness: a sonogra hic parameter for estimating gestational age of the foetus. Indian J Radiol Imaging 2002;12(4):553-4 .
44. Baptiste – Roberts K, Salafia (m,Nicholson WK, Duggan A, Wang N, Brancati FL: maternal risk factors for abnormal placental growth: The national collaborative perinatal project Bmc pregnancy & childbirth 2008 ,8:44 doi 10.1186/1471-2393-8-44 .
45. Wallace JM ,Aitken RP, Milne JS,HayJ.Ww:, nutritionally – mediated placental growth restriction in the growing adolescent : consequentces for the foetus Biol repos .2004 ,71 (4) : 1062.
46. Majumdar S, Dasgwa H, Bhattachaya K, Bhattacharya A. A study of placenta in normal and hypertensive pregnancies ,J. Anat. Soc. India 54 (2) 1-9 (2005).
47.Barker theory (2010) : the surface area of the placenta and hypertension in the offspring in later life. The international journal of developmental biology ,vol 54 nos : 2/3 ,2010 ;doi : 10,1387/ ildb.082760db.
48. Naeye RL. Do placental weight have clinical significance? Hum pathol 1987: 18: 387-391,680-691, in medical algorithms project.

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