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Original Article

Spectrum of morphological changes in the placenta of women with preeclampsia/eclampsia and correlation with neonatal morbidity.

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Abstract

Background: Preeclampsia/eclampsia is a leading cause of maternal and perinatal mortality and morbidity; the prevailing theory is that it is a consequence of disordered placentation, with the resultant underperfusion of the placenta triggering the release of cytokines and vascular factors, which cause widespread endothelial damage. The objective of this study was to compare the gross and microscopic changes in the placentas of women with preeclampsia/eclampsia and neonatal outcome. **Methodology:** One hundred and forty-six (146) pregnant women were recruited; 73 were normotensive, while 73 were diagnosed with preeclampsia/eclampsia. The macroscopic and microscopic placental changes in the two groups were examined. Full-thickness sections were taken from the central areas and grossly abnormal areas. The sections were fixed in 10% neutral-buffered formalin and processed for Haematoxylin/Eosin, Masson's trichrome, and Periodic Acid Schiff staining. The slides were reviewed to evaluate the pathologic changes and correlated with the birth weights and Apgar scores of the newborns. **Results**: 34%, 44%, and 22% of the women had preeclampsia without severe features, preeclampsia with severe features, and eclampsia, respectively. The placental weights were lower in the preeclampsia group compared to the control group. The major pathologic lesions with strong associations with preeclampsia/eclampsia were decidual vasculopathy, infarction, increased syncytial knots, accelerated villi maturity, stromal fibrosis, and microcalcifications. There was also a strong association between disease severity and Apgar scores in the 1st minute. The study group experienced an 11% neonatal mortality rate. **Conclusion**: There were distinct microscopic changes consistent with maternal vascular malperfusion changes in the placentas of mothers with preeclampsia/eclampsia and demonstrable neonatal morbidity depicted by a high incidence of preterm birth and low birth weights

Keywords: *Morphological changes, Neonatal morbidity, Placenta, Preeclampsia*.

Introduction

Pre-eclampsia is a complex pregnancy disorder involving multiple organ systems. It is characterised by varying degrees of placental insufficiency, which leads to the release of soluble factors (proinflammatory cytokines, antiangiogenic molecules) into the bloodstream. These factors result in damage to the maternal vascular endothelium, causing elevated blood pressure and injury to multiple organs.[1-3] The overall prevalence of pre-eclampsia in the United States is 3-8%, with a higher incidence in specific ethnic subpopulations, notably African Americans.[4] The prevalence of preeclampsia in developing countries ranges from 1.8% to 16.7%[4]. Nigeria has recorded varying values ranging from 3 to 8.8%. [5-7.]

Preeclampsia is clinically defined as new-onset hypertension (sustained systolic blood pressure of ≥140 mmHg or diastolic blood pressure of ≥90 mmHg,

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or both) with proteinuria, and/or end-organ dysfunction after 20 weeks of gestation. Pre-eclampsia is a significant cause of maternal and perinatal morbidity and mortality, particularly in low- and middle-income countries. It has been recognised as a significant contributor to maternal and foetal mortality and morbidity for over 100 years.[3]

Despite being one of the leading causes of maternal death and a major contributor to maternal and perinatal morbidity, the pathogenesis of preeclampsia/eclampsia has not been fully unravelled. The primary failure of trophoblast invasion and incomplete remodelling of maternal spiral arteries are recognised key mechanisms that contribute to pre-eclampsia.[1,2] The initiating events triggering the cascade of events leading to the reduced placental perfusion, widespread dysfunction of the maternal vascular endothelium, and hypertension remain undefined [1,2]

Pre-eclampsia is known to be associated with poor foetal/neonatal outcomes, and histopathological and microbiological examination of the placenta can reveal the possible aetiology of conditions such as stillbirth, pre-term delivery, and intrauterine growth restrictions (IUGR).[8] Placental examination also reveals abnormal findings in almost all cases of foetal death, and can diagnose the cause of death in about one-third of the cases in which it could not be determined clinically.[9,10] Furthermore, using placenta examination, medical conditions that can recur can be recognised, providing information to manage or treat subsequent pregnancies.[10-12] The data from a placental examination may also offer essential evidence in medical liability cases. They may provide defence against allegations of malpractice when the findings indicate that foetal or placental disease contributed to adverse pregnancy outcomes, independent of the clinical care provided.[9]

Many studies have established the pathological changes in the placenta of preeclamptic mothers, which include gross findings of smaller placentas with foci of calcification, infarction, and microscopic findings of increased syncytial knot formation, fibrinoid necrosis, stromal fibrosis, hyalinised villi, altered villous vascularity, cytotrophoblast cell proliferation, increased villi vascularisation and branching, intervillous haemorrhage, and basement membrane thickening.[11-17]

However, there is a paucity of such data in this environment; the placenta is handed to the mother or relatives almost immediately after birth, most times without examination, due to social and cultural beliefs that the placenta can be used for diabolical purposes.

In 1997, the College of American Pathologists published practical guidelines for placental pathological examination following delivery, [10] and in recent times, institutions have published modifications based on the original guidelines. The recommendations for the pathological examination are underlying maternal disease, pregnancy complications, foetal/ neonatal conditions, or placental indications.[8,18]

This research aimed to study the pathological changes in the placenta of preeclamptic/eclamptic mothers and their impact on newborn morbidity and mortality.

Materials and methods

One hundred and forty-six (146) pregnant women diagnosed at the clinic, labour ward, or at the Emergency Room of the University of Ilorin Teaching Hospital, Nigeria, were recruited for this study.

The sample size was calculated using the formula for cross-sectional studies.[19]

$$N = \frac{Z^2 pq}{d^2}$$

Where.

N = the minimum sample size

Z (1-α)/2 = 1.96 =value of the standard normal distribution corresponding to a significance level of α (0.05 at a confidence level of 95%).

p = proportion of the population estimated to have a particular condition, i.e., the prevalence of preeclampsia, 5%[4] = 0.05

q = 1.0- p = 1-0.05=0.95

d = degree of accuracy desired, which is 5% (0.05)

Applying the formula, the minimum sample size,

$$N = \frac{1.96^2 X\ 0.05\ X\ (1-0.05)}{(0.05)^2} = 72.9$$

N = 72.9, which is approximately 73.

Hence, a minimum sample size of 73 participants for the study group and an equal number for the control group was taken. Informed consents were taken from the recruited patients.

Inclusion criteria:

Study group

1. Maternal age 18-40 years.
2. Gestational age 28 weeks to term.
3. Diagnosis of preeclampsia.
4. Willingness to participate and signing of the informed consent form

Control group

1. Maternal age 18-40 years.
2. Gestational age 28 weeks to expected date of delivery.
3. Normal clinical and laboratory parameters

3. Willingness to participate and signing of the informed consent form

The diagnosis of preeclampsia was based on findings of new-onset hypertension and proteinuria or end-organ dysfunction occurring after 20 weeks of gestation.[20,21]

The study group participants were further stratified into:

1. Preeclampsia without severe features: Patients with systolic BP >140mmHg but <160mmHg and Diastolic<110mmHg, proteinuria >0.3g/24 hours.
2. Preeclampsia with severe features: Patients with any of these:
   (a)Systolic BP ≥160mmHg or diastolic BP >110mmHg on two occasions at least 4 hours apart, proteinuria >5g/24 hours.
   (b) Thrombocytopenia
   (c) Impaired liver function
   (d) New development of renal insufficiency.
   (e) Pulmonary oedema.
   (f) New-onset cerebral or visual disturbances.
3. Eclampsia: A preeclamptic patient with onset of seizures before, during, or after delivery.

Immediately after delivery, the newborns' weights and Apgar scores were recorded. The placentas were washed, weighed, and examined for completeness and gross abnormalities.

Full-thickness sections were taken from the central areas, grossly abnormal areas, and the membrane roll. The sections were fixed in 10% neutral-buffered formalin and processed for paraffin sections using an automatic tissue processor. Masson's Trichrome and Periodic Acid Schiff were used to demonstrate the presence of fibrosis and the basement membranes of placental/villous vessels, respectively. The stained slides were reviewed to evaluate the pathologic changes, and the pathologic findings were correlated with the birth weights and Apgar scores of the newborns. The data was collected using a proforma designed.

Statistical analysis was done with the Statistical Package for the Social Sciences version 22.0 (SPSS Inc., Chicago, IL, USA). Descriptive analysis was reported as means and standard deviations. Pearson's chi-squared test was used to analyse the observed differences in the study and control groups. The level of significance was considered at p p-value of ≤ 0.05.

Results

Demographic characteristics of the study group

A total of 1,575 women had their deliveries at the Department of Obstetrics and Gynaecology of the University of Ilorin Teaching Hospital, Ilorin, during the period of study. 5.14% of the women had preeclampsia-eclampsia. 60.3%, 17.8%, and 21.9% of the preeclampsia/eclampsia patients had preeclampsia without severe features, preeclampsia with severe features, and eclampsia, respectively. Twenty-three per cent (23%) had a history of hypertension in pregnancy without proteinuria, and 16.4% had a history of chronic hypertension (Fig.1). The mean gestational age at which the diagnosis of preeclampsia-eclampsia was made was 35.21 ± 3.85 weeks. The mean gestational age at delivery for the study and control groups was 37.04 ± 2.32 weeks and 38.48 ± 1.28 weeks, respectively. There was an increase in the rate of caesarean section in those with preeclampsia/eclampsia (41.1%) compared to the control group (2.7%).

Foetal weight, Apgar scores, and disease severity.

The mean birth weights of the study and control groups were 2.66 ± 0.77kg and 3.21 ± 0.43kg, respectively. 41.1% and 5.5% of the neonates in the study group had low birth weights and very low birth weights, respectively, compared to 9.6% in the control group who had low birth weights (Table 1).

Most (41.7%) of the newborns with low Apgar scores in the 1st minute were from mothers with preeclampsia with severe features, while 36.1% from mothers with eclampsia. However, half (50%) of neonates with low Apgar scores in the 5th minute were those from eclamptic mothers. The relationship between the 1st Apgar scores was significant (ρ = 0.008) (Table 1).

Table 1: Comparison of severity of disease and Apgar scores in the study group using chi-square.

	Severity of disease			
Variables	**Pre-eclampsia without severe features (%)**	**Pre-eclampsia with severe features (%)**	**Eclampsia (%)**	***P-value***
Birth weight				0.369
Very low birth weight	0 (0.0)	3 (75.0)	1 (25.0)	
Low birth weight	7 (23.3)	14 (46.7)	9 (30.0)	
Normal weight	18 (46.2)	15 (38.5)	6 (15.4)	
1st minute Apgar score				**0.008**
Low (<7)	8 (22.2)	15 (41.7)	13 (36.1)	
Normal (>7)	17 (45.9)	17 (45.9)	3 (8.2)	
5th minute Apgar score				0.127
Low (0-3)	2 (25.00)	2 (25.0)	4 (50.0)	
Abnormal (4-6)	2 (28.60)	1 (14.3)	4 (57.1)	
Reassuring (>7)	21 (36.2)	29 (50.0)	8 (13.8)	

*p< 0.050

Placental parameters in the participants (Study vs. Control).

Gross findings of infarction were seen in 16.4% of the placentas in the study group (66.7% were seen in mothers with preeclampsia with severe features, 25% eclampsia, and 8.3% preeclampsia without severe features), while none were observed in the placentas of the control group. The mean placental weights in the study vs. control group were 556.82g ± 169.72g vs. 649.93g ± 116.38g. All three 3 placentas that weighed less than 300g were from mothers with preeclampsia with severe features, and 71.4% of those weighing between 300-399g were from women with preeclampsia with severe features, and 28.6% preeclampsia without severe features. Twenty-nine (29) placentas weighed above 600g, out of which 51.7% were from mothers with mild-moderate preeclampsia, 31% severe preeclampsia, and 17.2% eclampsia.

Microscopic changes:

The pathologic findings seen include decidual arteriopathy, infarction, increased syncytial knots, abnormal vascular remodelling, terminal villous hypoplasia, accelerated villi maturation, villi immaturity, stromal fibrosis, acute chorioamnionitis, and chorangiosis.

Comparison of microscopic abnormalities in the study and control groups demonstrated statistical significance (Table 2, in all the microscopic features except for chorangiosis (Fig.2A) and acute chorioamnionitis (Fig.2B), in which there were no significant differences between the two groups.

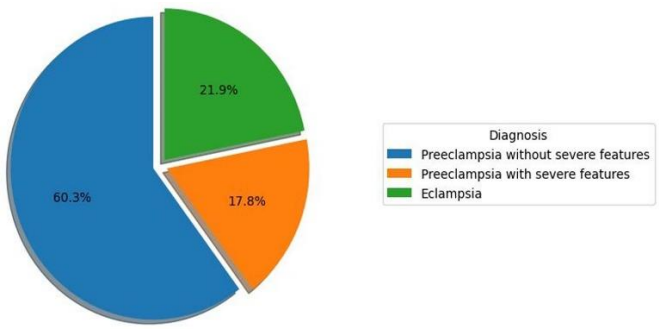

Figure 1: Pie chart showing the distribution of preeclampsia cases

Table 2: Comparison of microscopic placental parameters (Study vs. Control) using chi-square.

Microscopic parameters	Study group (%)	Control (%)	ρ
	Yes	Yes	
Decidual arteriopathy	11 (15.1)	0 (0.0)	**0.001***
Infarction	22 (30.1)	0 (0.0)	**< 0.001***
Increased syncytial knots	48 (65.8)	0 (0.0)	**< 0.001***
Incomplete vascular remodelling	9 (12.3)	0 (0.0)	**0.002***
Chorangiosis	7 (9.6)	2 (2.7)	0.085
Terminal villous hypoplasia	4 (5.5)	0 (0.0)	**0.043***
Accelerated villi maturity	11 (15.1)	0 (0.0)	**0.001***
Villi immaturity	11 (15.1)	3 (4.1)	**0.025***
Stromal fibrosis	50 (68.5)	0 (0.0)	**< 0.001***
Acute chorioamnionitis	3 (4.1)	4 (5.5)	0.698
Microcalcifications	54 (74.0)	24 (32.9)	**< 0.001***

*p< 0.050

Table 3: Association between microscopic findings and severity of disease using chi-square.

Microscopy	Severity of disease			ρ
	Pre-eclampsia without severe features (%)	Pre-eclampsia with severe features (%)	Eclampsia (%)	
Presence of decidual vasculopathy	1 (9.1)	6 (54.5)	4 (36.4)	0.138
Infarction	6 (27.3)	9 (40.9)	7 (31.8)	0.383
Increased syncytial knots	16 (33.3)	21 (43.8)	11 (22.9)	0.952
Incomplete vascular remodelling	0 (0.0)	5 (55.6)	4 (44.4)	**0.045***
Chorangiosis	2 (28.6)	5 (71.4)	0 (0.0)	0.211
Terminal villous hypoplasia	2 (50.0)	2 (50.0)	0 (0.0)	0.530
Accelerated villi maturity in preterm	2 (18.2)	4 (36.4)	5 (45.5)	0.110
Villi immaturity	0 (0.0)	6 (54.5)	5 (45.5)	**0.018***
Stromal fibrosis	15 (30.0)	22 (44.00)	13 (26.0)	0.360
Acute chorioamnionitis	3 (100.0)	0 (0.0)	0 (0.0)	0.050
Microcalcifications	14 (25.9)	27 (50.0)	13 (24.1)	**0.040***

*p< 0.050

Decidual arteriopathy (Fig.2C), infarction (Fig.2D), and increased syncytial knotting (Fig.3A) were seen in 15.1%, 30.1%, and 65.8%, respectively, of the placentas in the study group, as against none in the control group. Also, 12.3%, 5.5%, 15.1% and 68.5% of the placentas in the study group showed incomplete vascular remodelling (Fig.3B), terminal villous hypoplasia (Fig.3D), accelerated maturity (Fig.3E), and stromal fibrosis (Fig.3F) respectively was seen in of the placentas while none was seen in the control

group. However, 74% in the study group showed microcalcifications (Fig.3G) while 32.9% placentas in the control group showed microcalcifications.

Microscopic placental findings, severity of preeclampsia/eclampsia, and foetal outcome.
About half of mothers with preeclampsia with severe features showed incomplete vascular modelling, villi immaturity, and microcalcifications; they were seen in 44.4%, 45.5%, and 24.1%, respectively, compared to those with eclampsia (Table 3). Infarction was seen in 40.9% of those with preeclampsia with severe features, 31.8% of eclampsia, and 27 of preeclampsia without severe features. Microscopic findings of accelerated villi maturity had no statistical significance but were observed to be more common in the placentas of patients with severe disease, with 36.4% in preeclampsia with severe features, 45.5% in eclampsia, and 18.2% in preeclampsia without severe features.
In 63.6% of the neonates who survived and 36.4% of the neonates who died, there was a decidual arteriopathy in the placentas. Incomplete vascular remodelling, accelerated villi maturity, and microcalcifications were seen in 55.6%, 63.6%, and 94.4% of the placentas of the neonates who survived, respectively. Microscopic findings of infarction increased syncytial knots, chorangiosis, terminal villous hypoplasia, villi immaturity, and stromal fibrosis, were seen more predominantly in neonates who survived.

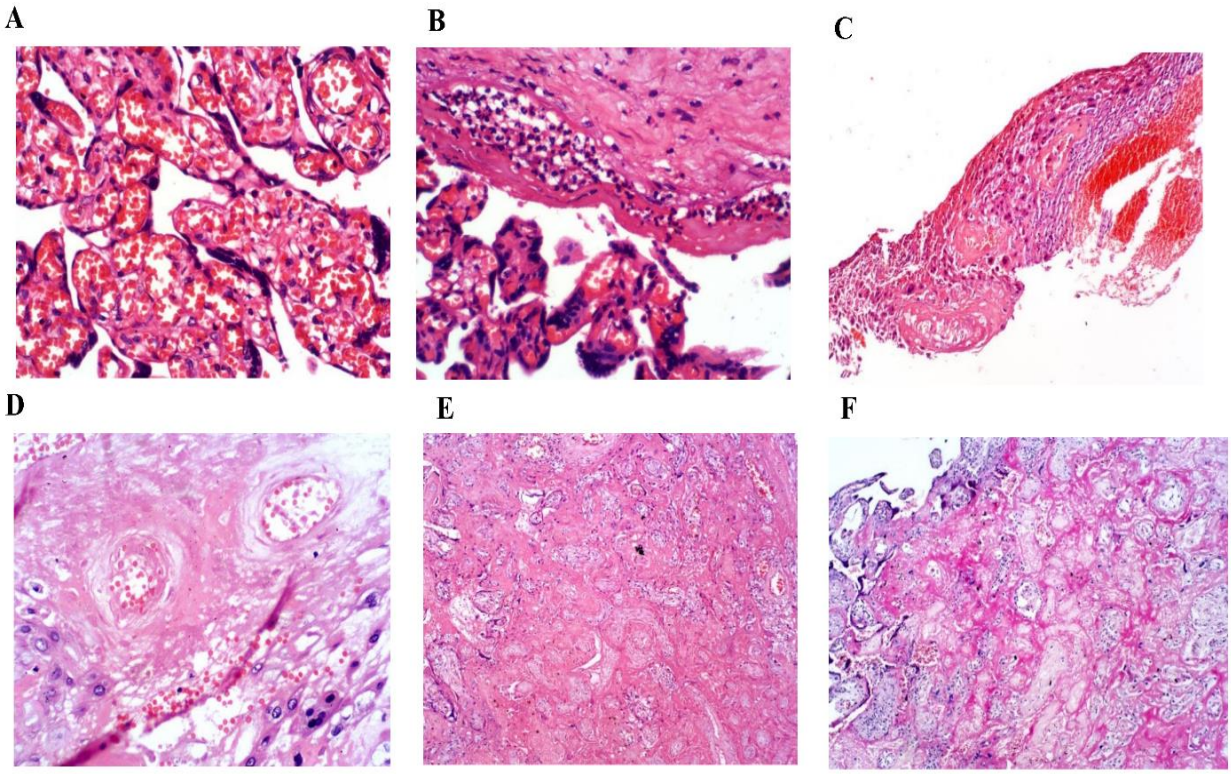

Figure 2. Photomicrograph showing A: Chorangiosis; increased villous capillaries and congestion, H&E x400. B: Acute chorioamnionitis showing neutrophilic infiltrates in the chorionic plate, H&E x400. C: Advanced decidual arteriopathy with acute atherosis and fibronoid necrosis, H&E x100. D: Advanced decidual arteriopathy with marked fibrinoid necrosis of decidual vessels, H&E x400. E: Placental infarction with collapse of the intervillous space, loss of nuclear basophilia in the villi (few pockets of viable villi in the left upper part), H&E x100. F: Fibrin deposition (intense pink) around necrotic villi (pale pink), loss of basophilia and loss of intervillous space, PAS x100

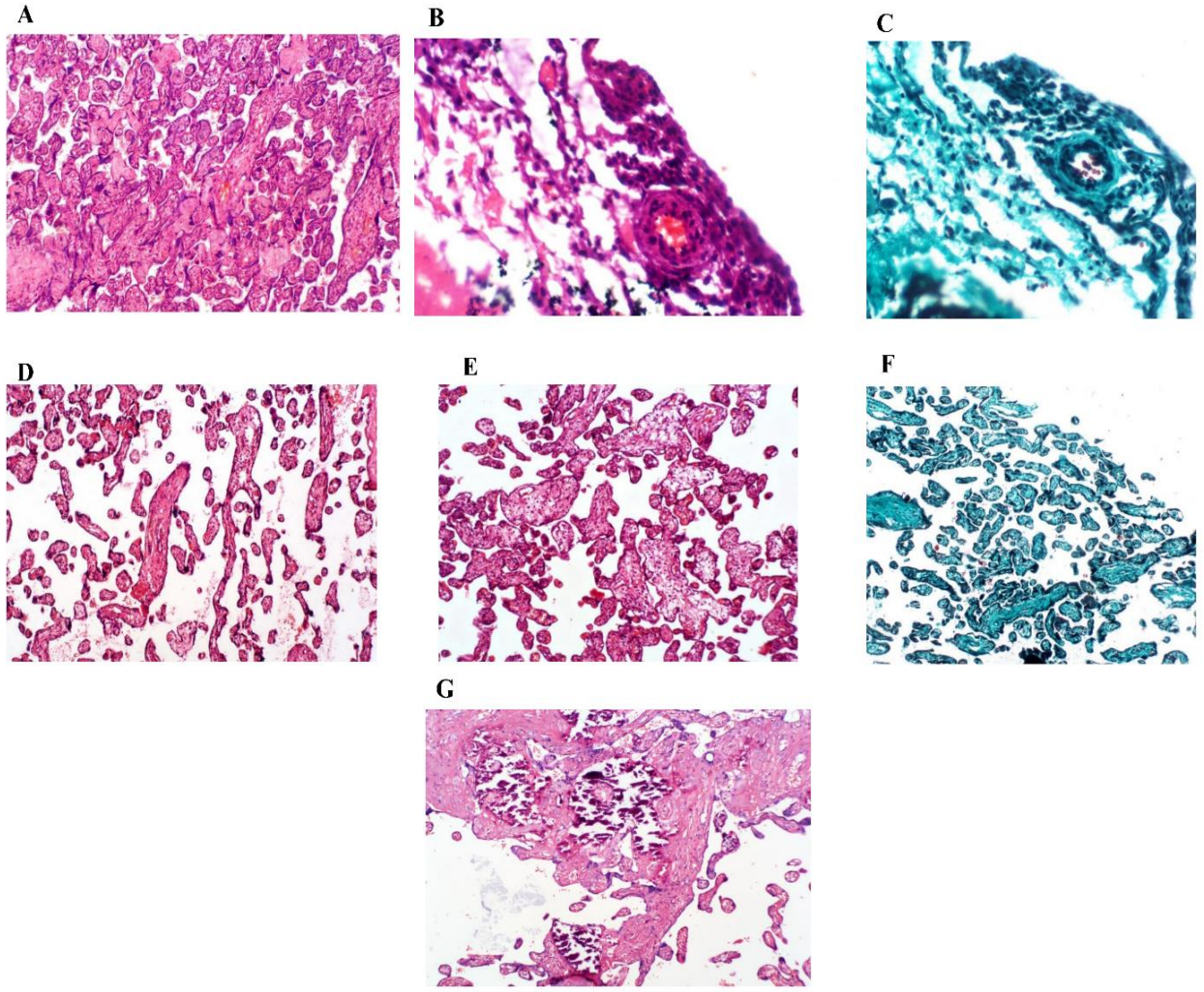

Figure 3. Photomicrograph showing A: Tenney Parker" changes: Increased syncytial knots in the intermediate and terminal villi, H&E x100. B: Basal plate showing an arteriole with persistent muscularised arterioles (abnormal vascular remodelling) H&E x100. C: Basal plate, Masson's Trichrome x100. D: Villous hypoplasia in a term placenta characterised by long, slender, minimal branching terminal villi and unusually wide intervillous spaces, H&E x100. E: Villi immaturity immature intermediate villi (larger), few mature intermediate villi and few terminal villi, H&E x100. F: Stromal fibrosis, terminal villi showing increased collagen and reduced vasculature, MT x100. G: Microcalcifications in a term placenta, H&E x40

Discussion

Preeclampsia-eclampsia is a pregnancy-specific disease, hence its historic name "Toxaemia of pregnancy".[22] It is characterised by new-onset hypertension, with proteinuria and/or end-organ dysfunction, differentiating it from chronic hypertension and gestational hypertension. The placenta has been identified as the root cause of preeclampsia, resulting from disordered implantation and poor perfusion of the placenta, leading to the release of molecules that cause widespread endothelial injury.[1,23,24] A common pattern of placental injury associated with altered intervillous blood flow is maternal vascular malperfusion of the placenta. Maternal vascular malperfusion is defined by a group of pathological findings observed in the maternal decidual vessels, which reflect unusual spiral artery remodelling and villous parenchyma abnormalities relating to oxygenation and flow dynamics within the intervillous space of the placenta.[25]

The prevalence of preeclampsia in this study is 5%. The prevalence of the disease varies widely within Nigeria. Singh et al[7] reported a prevalence of 6% in Sokoto. A prevalence of 8% was reported in Kano[26]

and Jos[5], 5.44% in Nnewi[27,] and 5.6% in Benin[28]. However, a lower prevalence rate of 1.2% was reported in Calabar[29] and 3.3% in Ogun[30]. The reasons for these interregional differences are not known, but could be due to sociocultural practices and geographical factors.

The percentages of women with a past obstetric history of preeclampsia/eclampsia and/or chronic hypertension in previous pregnancies in this study were 17.8% and 16.4%, respectively. This implies recurrence in this group of patients. A large study done in the UK noted higher risks of recurrence in Asians and Blacks and highlighted chronic hypertension as an established risk factor for preeclampsia, with data showing up to 22% of women with chronic hypertension having subsequent preeclampsia.[31] The recurrence rate of preeclampsia was noticed to be 17.1% in a study by Wainstock and Sheiner. Maternal age, caesarean, and preterm deliveries were the noted risk factors that contributed significantly to the recurrence.[32] Obesity and women of colour also contributed significantly to the risk of pre-eclampsia recurrence [33,34]

In this study, the gestational age at delivery in the study group was significantly lower than in the control group; hence, prematurity was one of the major perinatal complications seen. The increased rate of caesarean delivery seen in the study group showed that preeclamptic mothers had over 13 times the risk of having surgical obstetric intervention than normotensive patients due to the precarious states the mothers present in. The high prevalence of low-birth-weight newborns seen in the study group implied that preeclampsia imposes a 5x risk of low birth weight in preeclamptic mothers compared to the control group. The high incidence of low birth weight in the neonates born to preeclamptic mothers in this study reflects the high incidence of prematurity (49.3%) seen, as prematurity was the major adverse perinatal outcome.

The overall mean placental weights in those with preeclampsia in this study were significantly lower than those of the normotensive control group. Previous studies also reported similar findings[13,16,27,31.] A patient with preeclampsia and intrauterine growth restriction had a significant reduction in gestational age-adjusted placental weight when compared with the control.[36] A previous work in our centre recorded a mean placental weight of 580.8 grams.[37] The mean feto-placental weight ratios in the study and the control group were 4.87 and 5.01. The feto-placental weight was slightly lower in the study group, but there were no statistical differences between these groups. A higher incidence of abnormal cord insertion was recorded in the study group. The incidence of marginal cord insertion was 10 times higher in the study group compared to the control group. However, abnormal cord insertions are not considered specific for preeclampsia. Gross infarcts were seen in 16% of the placentas from the preeclamptic group compared to none seen in the normotensive group. Other studies have recorded higher incidences. Placental infarcts are common, but when they are multiple or occupy more than 5% of the placental volume, they are considered pathologic.

The microscopic placental lesions exclusively seen in the preeclampsia/eclampsia group (in decreasing frequency) in this study were stromal fibrosis, increased syncytial knotting, infarction, decidual arteriopathy, accelerated villi maturity, and incomplete vascular remodelling. The incidence of villous/vascular lesions was 4-7-fold higher in the placentas of the preeclampsia group compared to the normotensive groups. These findings were comparable to early work by Moldenhauer et al. ([38] who reported that decidual arteriolopathy, villi hypermaturity, intervillous thrombi, and central infarction were significantly higher in the placentas of women with preeclampsia.[38] Similar findings were reported by Falcon et al[39]. The study revealed villous lesions in 48.2% of preeclamptic pregnancies and 11.6% of normal pregnancies, while vascular lesions were recorded in 37.3% of preeclamptic pregnancies and 8.1% of normal pregnancies. This review concluded that the incidence of villous/vascular lesions was 4-7-fold higher in the placentas of the preeclampsia group compared to the normotensive groups.[39] The significant microscopic findings in the placentas of preeclamptic women in the study by Ezeigwe et al[27] were decidual arteriopathy, accelerated villous maturation, and cytotrophoblastic proliferation. These findings were partly comparable to our own findings, as stromal fibrosis and increased syncytial knots were prominent findings in our study, although Ezeigwe et al employed only Haematoxylin/Eosin staining in their histological assessment of the placenta, with no additional histochemical staining. This could explain the low incidence of stromal fibrosis recorded in that study. Donthi et al. ([40] reported a significant association between fibrin deposition, maternal floor infarction,

calcification, villous basement membrane thickening, and syncytial knots in patients with pre-eclampsia and eclampsia.[40] Konkov et al[41] noted widespread fibroid necrosis and compensatory angiomatosis of the terminal villi.

Although chorangiosis was observed more frequently in placentas of individuals with preeclampsia with severe features, this study found no strong association between chorangiosis and maternal disease severity. However, some studies have linked chorangiosis to preeclampsia and diabetes, suggesting that it may be a consequence of placental insufficiency.[17,42] Hargitai et al[43] and Roberts[44] pointed out that chorangiosis is an adaptive feature developing as a result of maternal hypoxaemia, which could be due to cardiopulmonary diseases or high altitude, and not necessarily placental insufficiency or ischaemia. On the other hand, villous immaturity was unexpectedly a significant finding in our study, although it is not usually associated with hypertensive diseases; however, some studies have reported this finding in cases of maternal diabetes, some cases of foetal growth restriction, and chronic cord obstruction.[43,45]

This study found associations between vascular and villous abnormalities in the placentas of preeclamptic mothers and Apgar scores at the 5th minute. These same observations were made in the association between the vascular and villi abnormalities and foetal outcome, but with much stronger statistical significance. Stevens et al[46] examined the association between decidual arteriopathy and worse clinical outcomes and found a striking linear correlation between the numbers of vessels showing fibrinoid necrosis and/or thrombosis in the placental bed (decidua basalis) and increased perinatal mortality [46]

There were strong associations between disease severity and microscopic findings of incomplete vascular remodelling, villi immaturity, and calcifications in the placenta. These lesions were seen mostly in patients with preeclampsia with severe features and eclampsia. The absence of incomplete vascular remodelling in the placentas of normotensive women and those with mild diseases implied the specificity of this finding in the severe spectrum of preeclampsia. It was seen in 11% of the preeclamptic group and 4.1% of the normotensive group, with a strong association with severe disease.

Unlike the 1st minute Apgar scores, this study found that scores at the 5th minute had no significant association with disease severity. This suggests that standard resuscitative measures initiated immediately after birth are often successful in reversing depressed circulation. The 1st minute score remains a vital tool. It provides a "quick peek" into the true intrauterine perfusion status and the immediate success of the transition to extrauterine life, while the scores at the 5th minute measure the efficacy of the resuscitative efforts rather than the initial fetal state.[47,48]
In clinical practice, resuscitation is extended for infants with low 5th-minute scores. Occasionally, what is recorded as a "5th-minute score" may reflect an extended Apgar score (allotted every 5 minutes up to 20 minutes). It is often impractical to pause life-saving resuscitation at exactly the 5-minute mark simply to update a chart.[47,48]

We recorded 11% neonatal mortality in the study group, but none in the control group. This is corroborated by a similar finding by Randriamahavonjy et al[49,] which reported intrauterine mortality and perinatal mortality of 11.34%.[49] Placental insufficiency is often described as the cause of perinatal mortality in preeclampsia. It is defined as the inability of the poorly adaptive placental tissue to deliver adequate perfusion to the growing foetus in utero. A secondary analysis by the World Health Organisation on the adverse outcomes of preeclampsia/eclampsia in low- and medium-income countries found that preeclampsia was a predominant risk factor for maternal/perinatal mortality, preterm birth, and low birth weight.[50] Also, there was an inverse linear relationship between perinatal mortality and increasing gestation age up to 40 weeks

Conclusion

The major pathologic lesions in the placenta with a strong association with preeclampsia/eclampsia were acute atherosis, infarction, increased syncytial knots (Tenney-Parker changes), accelerated villi maturity, stromal fibrosis, and microcalcifications. There is a strong association between disease severity and Apgar scores in the 1st minute. There were distinct pathological changes in the placentas of mothers with preeclampsia and demonstrable neonatal morbidity depicted by a high incidence of preterm birth, low birth weights, and low Apgar scores in the 1st minute. The vascular lesions are more of a consequence of the initiating placental pathology in

preeclampsia/eclampsia rather than the direct cause of morbidity.

Declaration

Ethics approval and consent to participate.

The University of Ilorin Teaching Hospital Ethical Research Committee approved the study, Approval number ERC PAN/2017/01/1625. Both the study and control group participants showed willingness to participate and signed the informed consent after explaining the aim of the study. The signing of the consent form was part of the inclusion criteria.

Consent for publication

Not applicable

Availability of data and materials

The datasets used and analysed during this current study are available from the corresponding author on reasonable request.

Competing interest

The authors declare that they have no competing interests.

Funding

Not applicable

Authors contribution

FOO, BMO, and IOK conceptualised and designed this project; FOO collected and analysed the data.

FOO, IKM, FIO, IAS, and AL wrote the manuscript. All authors proofread the manuscript.

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