Section: Pathology

Original Research Article

HISTOPATHOLOGICAL STUDY OF PLACENTAL CHANGES ASSOCIATED WITH PREECLAMPSIA AND ECLAMPSIA IN A TERTIARY CARE CENTRE

Manpreet Kaur[1], Reema Bhushan[2], Arun Jain[3]

[1]Post graduate Student, Department of Pathology, GR Medical College, Gwalior
[2]Assistant Professor, Department of Pathology, GR Medical College, Gwalior
[3]Designate Professor, Department of Pathology, GR Medical College, Gwalior

Received : 25/04/2026
Received in revised form : 11/06/2026
Accepted : 29/06/2026

Keywords:
Preeclampsia, Eclampsia, Histopathology, Placenta.

Corresponding Author:
Dr. Manpreet Kaur
Email: drmanpreethans08@gmail.com

DOI: 10.47009/jamp.2026.8.4.34

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2026; 8 (4); 180-188

ABSTRACT

Background: Preeclampsia and eclampsia are serious hypertensive disorders occurring during pregnancy. They can lead to significant health issues for both mothers and babies. These conditions arise due to unusual growth of trophoblastic cells leading to placental dysfunction. Examining the placenta under a microscope gives valuable information about how these diseases develop and their severity. This helps to improve understanding and management of these disorders. The above study was conducted to examine the morphological and histopathological changes that occur in the placenta of women diagnosed with preeclampsia and eclampsia. **Materials and Methods:** The study was conducted at a tertiary care hospital with 92 placental samples taken into consideration. Placental specimens were collected after delivery, washed, and fixed by in 10% formalin for 24 hours. After macroscopic examination, representative sections from the maternal and fetal surfaces and umbilical cord were processed, paraffin-embedded, and stained with hematoxylin and eosin for histopathological evaluation. Clinical and histological data were recorded systematically and analyzed using SPSS version 20, with $p < 0.05$ considered statistically significant. **Result:** The mean systolic blood pressure (SBP) for eclampsia cases was 142.20 mmHg (SD = 4.76), which was significantly higher than mild preeclampsia (146.24 mmHg, SD = 6.89) and severe preeclampsia (144.79 mmHg, SD = 6.51). The highest mean DBP recorded was of 101.00 mmHg (SD = 10.41). Eclampsia cases had a mean placenta weight of 432.50 grams (SD = 35.06), mild preeclampsia had 454.00 grams (SD = 44.63), and severe preeclampsia had 436.49 grams (SD = 34.56) which was significantly reduced as compared to normal groups. Infarction was significantly more common in preeclampsia and eclampsia. **Conclusion:** By identifying key histopathological changes early, clinicians can make more informed decisions regarding the timing of delivery and the management of maternal and fetal health. Overall, the study emphasizes the value of histopathological analysis in improving the management and outcomes of pregnancies complicated by hypertensive disorders.

INTRODUCTION

Preeclampsia is defined by the development of hypertension and proteinuria after the 20th week of pregnancy, whereas eclampsia is characterized by the occurrence of seizures in a woman with preeclampsia. Both disorders are associated with multiple systemic complications. The pathophysiology of preeclampsia and eclampsia involves complex interactions between maternal immune responses, endothelial dysfunction, and abnormal placental development, making the placenta a key organ in understanding the disease mechanisms.[1] The placenta, as the central organ of pregnancy, plays a crucial role in fetal development by ensuring nutrient and oxygen transfer, regulating waste elimination, and maintaining maternal-fetal immune tolerance. In preeclampsia, the trophoblastic invasion is impaired, leading to incomplete vascular remodeling, which results in placental hypo perfusion, ischemia, and inflammation. These changes contribute to the clinical manifestations of preeclampsia, such as hypertension, proteinuria, and edema.[2]

Histopathological studies of the placenta in women with preeclampsia and eclampsia have revealed a variety of morphological changes reflecting the underlying pathophysiological processes of these disorders. Common findings include fibrinoid necrosis, increased syncytial knots, villous edema,

ISSN (O): 2687-5365; ISSN (P): 2753-6556

and placental infarcts. These abnormalities are linked to impaired trophoblastic invasion and hypo perfusion of the placenta.[3] Placental infarction is one of the most significant and common findings in the histopathological examination of pre-eclamptic and eclamptic placentas. Infarcts occur due to inadequate blood supply and are indicative of ischemia in the placental tissues. Placental infarcts contribute to fetal growth restriction and can be associated with adverse pregnancy outcomes such as preterm birth and stillbirth.[4]

In addition to infarcts, histological examination often reveals other abnormalities, including the presence of perivillous fibrin deposits, thickened basement membranes, and changes in the villous architecture. These alterations indicate chronic placental stress and are thought to be markers of the disease's progression.[5] Understanding the histopathological features of preeclampsia and eclampsia can help in predicting the prognosis for both the mother and the fetus. Examining placental tissue in women with preeclampsia and eclampsia can provide valuable prognostic information.[6] Moreover, histopathological studies can assist in differentiating preeclampsia and eclampsia from other hypertensive disorders of pregnancy, such as chronic hypertension and gestational hypertension, which may not exhibit the same degree of placental abnormalities. By analyzing the morphological changes in the placenta, clinicians can confirm the diagnosis of preeclampsia or eclampsia, providing an evidence-based approach to managing these conditions.

The present study aims to examine the morphological and histopathological changes that occur in the placenta of women diagnosed with preeclampsia and eclampsia. By identifying the specific histological markers associated with these conditions, we hope to contribute valuable information for improving clinical outcomes and advancing the field of obstetrics and gynecology.

MATERIALS AND METHODS

The study was conducted in the Department of Pathology in Histopathology Laboratory, G.R. Medical College, Gwalior for a period of one and a half years (2022-2025). Ethical approval for the study was obtained from the Institutional Review Board of the institution. Ninety two placental samples were included for the study.

Inclusion Criteria

Placentas of singleton pregnancies complicated by preeclampsia or eclampsia, pregnant women diagnosed with preeclampsia and eclampsia during their antenatal care, women with gestational ages between 20 to 40 weeks, regardless of the severity of preeclampsia or eclampsia, placentas from patients who delivered either via vaginal birth or cesarean section and patients providing consent for the study.

Exclusion Criteria

Pregnancies complicated by other medical disorders such as diabetes mellitus, heart disease, or renal disease, multiple pregnancies (twin or higher-order pregnancies), complicated by preterm birth (before 37 weeks of gestation), normal pregnancies without any hypertensive disorder or complications, placentas that were not properly preserved or were damaged during collection were excluded from the study.

The participants were divided into two primary groups based on the type of hypertensive disorder of pregnancy:

Preeclampsia Group: This group consisted of placental samples from singleton pregnancies complicated by preeclampsia. The inclusion criteria for this group were met by women diagnosed with preeclampsia at various stages of pregnancy.

Eclampsia Group: This group comprised placental samples from singleton pregnancies complicated by eclampsia. Eclampsia cases were defined by the occurrence of seizures in the presence of preeclampsia.

Placenta specimens were collected from patients after delivery. The placental tissues were immediately washed to remove blood and clots, ensuring clean tissue samples for histological examination. The placenta was then perfused with 10% formalin through the umbilical vessels, and the entire organ was immersed in a 10% formalin solution for 24 hours to allow proper fixation. Following fixation, a detailed macroscopic examination was performed to assess any visible pathological features such as infarction, calcification, or retroplacental clots. Sections were taken from the maternal and fetal surfaces of the placenta, as well as from the umbilical cord. These sections were processed, embedded in paraffin, and stained with routine Haematoxylin and Eosin (H&E) stain. Finally, slides were examined under a compound microscope for a detailed study of the placental histology.

Data was collected through documentation of the clinical information, including maternal and fetal characteristics, along with detailed histological findings from the placental samples. Each placental specimen was assigned a unique identifier, and its relevant clinical and histopathological data were recorded in a structured format. The collected data were analyzed using SPSS software (Version 20). A p- value of <0.05 was considered statistically significant, while p-values of <0.01 were considered highly significant.

RESULTS

The gestational period of the study participants showed that majority of the deliveries occurring at 39 weeks, accounting for 43.14% of the total cases. A significant number of participants also delivered at 38 weeks (23.53%) and 37 weeks (21.57%). Fewer cases

ISSN (O): 2687-5365; ISSN (P): 2753-6556

were reported at earlier gestations, such as 36 weeks (5.88%), 34 weeks (3.92%), and 32 weeks (1.96%) [Table 1].

The past illness distribution among the study participants showed that nearly half of the women (48.04%) had no prior illnesses. Among those with past medical conditions, anemia was the most prevalent, affecting 21.57% of participants. High body mass index (BMI) was also common, observed in 12.75% of the cases. Hypertension was found in 11.76% of the participants, and a smaller number had asthma (1.96%), diabetes (2.94%), and heart disease (0.98%) [Table 1].

Table 1: Gestational Period and Past Illness of Study Participants

Gestational Period	**Frequency**	**Percentage**
32 weeks	2	1.96%
34 weeks	4	3.92%
36 weeks	6	5.88%
37weeks	22	21.57%
38 weeks	24	23.53%
39 weeks	44	43.14%
Total	102	100.00%
	Past Illness	
Past Illness	**Frequency**	**Percentage**
Anemia	22	21.57%
Asthma	2	1.96%
Diabetes	3	2.94%
Heart disease	1	0.98%
High Body mass index	13	12.75%
Hypertension	12	11.76%
No illness	49	48.04%
Total	102	100.00%

Table 2: Systolic & Diastolic Blood Pressure (SBP &DBP) in Different Complications

Complications	**N**	**SBP**		**P-value**
		Mean	**S.D.**	
Eclampsia	10	142.20	4.76	< 0.001
Mild Preeclampsia	25	146.24	6.89	
severe Preeclampsia	57	144.79	6.51	
Normal	10	117.2	6.197	
		DBP		
		Mean	**S.D.**	
Eclampsia	10	101.00	10.41	< 0.001
Mild Preeclampsia	25	99.80	5.63	
Severe Preeclampsia	57	99.56	5.18	
Normal	10	77.8	4.662	

The mean Systolic Blood Pressure (SBP) for eclampsia cases was notably high at 142.20 mmHg with a standard deviation of 4.76 mmHg, indicating a clear elevation compared to the other groups. Mild preeclampsia had a mean SBP of 146.24 mmHg, while severe preeclampsia cases showed a slightly lower mean of 144.79 mmHg. The normal group had a significantly lower mean SBP of 117.2 mmHg. The p-value of <0.001 suggests that the differences in SBP between these groups was statistically significant, emphasizing the impact of preeclampsia and eclampsia on blood pressure [Table 2].

The diastolic blood pressure (DBP) levels across the different complications also showed significant differences. Eclampsia cases had the highest mean DBP of 101.00 mmHg with a standard deviation of 10.41, followed by mild preeclampsia (99.80 mmHg) and severe preeclampsia (99.56 mmHg). The normal group had a significantly lower mean DBP of 77.8 mmHg. The p-value of <0.001 indicated that the differences in DBP between these groups are statistically significant, highlighting the elevated blood pressure in women with preeclampsia and eclampsia as compared to those with normal pregnancy [Table 2].

Table 3: Placenta Weight (gm) in Different Complications

Complications	**N**	**Placenta weight (gm)**		**P-value**
		Mean	**S.D.**	
Eclampsia	10	432.50	35.06	< 0.001
Mild Preeclampsia	25	454.00	44.63	
severe Preeclampsia	57	436.49	34.56	
Normal	10	538.33	31.425	

The placenta weight in eclampsia cases was 432.50 grams, while mild preeclampsia cases had a mean weight of 454.00 grams. Severe preeclampsia has a mean placenta weight of 436.49 grams. The normal

ISSN (O): 2687-5365; ISSN (P): 2753-6556

group showed the highest mean placenta weight of 538.33 grams. The p-value of <0.001 indicated that the differences in placenta weight between the groups are statistically significant, suggesting that placental weight was reduced in preeclampsia and eclampsia cases compared to normal pregnancies [Table 3].

Table 4: Distribution of Complications Across Different Gestational Periods

Gestational Period	Complications n (%)				P-value
	Eclampsia	Mild Preeclampsia	severe Preeclampsia	Normal	
32 weeks	0 (0)	0 (0)	0 (0)	2 (20)	< 0.001
34 weeks	0 (0)	0 (0)	1 (1.75)	3 (30)	
36 weeks	0 (0)	1 (4)	1 (1.75)	4 (40)	
37weeks	4 (40)	4 (16)	13 (22.81)	1 (10)	
38 weeks	3 (30)	9 (36)	12 (21.05)	0 (0)	
39 weeks	3 (30)	11 (44)	30 (52.63)	0 (0)	
Total	10 (100)	25 (100)	57 (100)	10 (100)	

Majority of complications occurred at or near full-term gestation (37 to 39 weeks). At 37 weeks, 40% of eclampsia cases, 16% of mild preeclampsia, and 22.81% of severe preeclampsia were recorded. The trend continued at 38 weeks, where 30% of eclampsia, 36% of mild preeclampsia, and 21.05% of severe preeclampsia cases occurred. No cases of eclampsia or mild preeclampsia were observed at 32 or 34 weeks. The p-value of <0.001 suggests a statistically significant association between gestational period and the type of complication [Table 4].

Table 5: Presence of Placental Infarction Across Different Complications

Infarction	Complications n (%)				P-value
	Eclampsi a	Mild Preeclampsia	Severe Preeclampsia	Normal	
Not Present	6 (60.00)	9 (36.00)	27 (47.37)	10 (100)	0.006
Present	4 (40.00)	16 (64.00)	30 (52.63)	0 (0)	
Total	10 (100)	25 (100)	57 (100)	10 (100)	

Among the eclampsia group, 40% had placental infarction, while 64% of mild preeclampsia and 52.63% of severe preeclampsia cases had placental infarction. In contrast, no cases of infarction were observed in the normal pregnancy group. The p-value of 0.006 indicated a statistically significant association between the presence of infarction and the complications [Table 5].

Table 6: Presence of Calcification Across Different Complications (gross)

Calcification	Complications n (%)				P-value
	Eclampsia	Mild Preeclampsia	Severe Preeclampsia	Normal	
Not Present	6 (60.00)	15 (60.00)	27 (47.37)	10 (100)	0.02
Present	4 (40.00)	10 (40.00)	30 (52.63)	0 (0)	
Total	10 (100)	25 (100)	57 (100)	10 (100)	

In the eclampsia group, 40% of cases showed placental calcification, while 40% of mild preeclampsia and 52.63% of severe preeclampsia cases also exhibited calcification. In contrast, no calcification was observed in the normal pregnancy group [Table 6].

Table 7: Syncytial Knot Formation Across Different Complications

Syncytial knots formation per 100 villi	Complications n (%)				P-value
	Eclampsia	Mild Preeclampsia	severe Preeclampsia	Normal	
0 – 30	0 (0)	0 (0)	0 (0)	6 (60)	< 0.001
31 to 60	1 (10.00)	7 (28.00)	19 (33.33)	4 (40)	
61 to 90	8 (80.000)	16 (64.00)	28 (49.12)	0 (0)	
91 to 120	1 (10.00)	2 (8.00)	10 (17.54)	0 (0)	
Total	10 (100)	25 (100)	57 (100)	10 (100)	

In the eclampsia group, 80% of cases had syncytial knots 61 to 90 per 100 villi, with an additional 10% in the 91 to 120 per 100 villi. Mild and severe preeclampsia cases also showed a high prevalence of syncytial knots, particularly in the 31 to 60 and 61 to 90 per 100 villi. In contrast, syncytial knots were absent in the normal pregnancy group, with 60% having no syncytial knots [Table 7].

Table 8: Fibrinoid Necrosis Across Different Complications

Fibrinoid necrosis per 100 villi	Complications n (%)				P-value
	Eclampsia	Mild Preeclampsia	severe Preeclampsia	Normal	
0 to 05	0 (0)	0 (0)	0 (0)	7 (70)	< 0.001
6 to 10	9 (90.00)	14 (56.00)	36 (63.16)	3 (30)	
11 to 15	1 (10.00)	11 (44.00)	16 (28.07)	0 (0)	
16 to 20	0 (0.00)	0 (0.00)	3 (5.26)	0 (0)	
21 to 25	0 (0.00)	0 (0.00)	2 (3.51)	0 (0)	

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Total	10 (100)	25 (100)	57 (100)	10 (100)	

In the eclampsia group, 90% of cases showed fibrinoid necrosis in the 6 to 10 per 100 villi. A similar pattern was observed in mild and severe preeclampsia, with 56% and 63.16% of cases, respectively, exhibiting fibrinoid necrosis in the same range. Only 30% of the normal pregnancy group showed mild fibrinoid necrosis, primarily in the 6 to 10 per 100 villi, and 70% had no evidence of fibrinoid necrosis. These parameters were found to be statistically significant [Table 8].

Table 9: Area of Cytotrophoblastic Proliferation Across Different Complications

Area of cytotrophoblastic proliferation per 100 villi	Complications n (%)				P-value
	Eclampsia	Mild Preeclampsia	severe Preeclampsia	Normal	
1 to 05	0 (0)	0 (0)	0 (0)	7 (70)	< 0.001
6 to 10	1 (10.00)	2 (8.00)	2 (3.51)	3 (30)	
11 to 15	2 (20.00)	4 (16.00)	16 (28.07)	0 (0)	
16 to 20	5 (50.00)	14 (56.00)	27 (47.37)	0 (0)	
21 to 25	2 (20.00)	5 (20.00)	12 (21.05)	0 (0)	
Total	10 (100)	25 (100)	57 (100)	10 (100)	

The area of cytotrophoblastic proliferation showed a significant association with preeclampsia and eclampsia, with much higher proportions of cases in the higher ranges compared to normal pregnancies. In the eclampsia group, 50% of cases exhibited cytotrophoblastic proliferation in the 16 to 20 per 100 villi, and 20% had it in the 21 to 25 per 100 villi. Mild and severe preeclampsia cases followed similar patterns, with notable proliferation in the 16 to 20 per 100 villi. In contrast, no normal pregnancy cases showed cytotrophoblastic proliferation in these ranges, with 70% showing no proliferation. The p-value of <0.001 indicates a statistically significant relationship between cytotrophoblastic proliferation and preeclampsia and eclampsia, suggesting it may be a marker for placental changes in these conditions [Table 9].

Table 10: Calcified Areas Across Different Complications (microscopic examination)

Calcified area per 10/ LPF(10x)	Complications n (%)				P-value
	Eclampsia	Mild Preeclampsia	severe Preeclampsia	Normal	
0	0 (0.00)	0 (0.00)	0 (0.00)	8 (80.00)	< 0.001
1	0 (0.00)	5 (20.00)	6 (10.53)	2 (20.00)	
2	5 (50.00)	13 (52.00)	29 (50.88)	0 (0.00)	
3	2 (20.00)	4 (16.00)	10 (17.54)	0 (0.00)	
4	3 (30.00)	1 (4.00)	9 (15.79)	0 (0.00)	
5	0 (0.00)	2 (8.00)	3 (5.26)	0 (0.00)	
Total	10 (100)	25 (100)	57 (100)	10 (100)	

In the eclampsia group, 50% of cases showed 2 calcified areas per 10 LPF, with additional 20% showing 3 calcified areas per 10 LPF. Similar trends were observed in mild and severe preeclampsia, with 52% and 50.88% of cases, respectively, having 2 calcified areas per 10 LPF. In contrast, 80% of normal pregnancies showed no calcified areas, and no cases of calcification were observed in the 4 or 5 per 10 LPF. The p-value of <0.001 indicated a statistically significant association between calcified areas and the complications, highlighting that calcification was more prevalent in placental tissues affected by preeclampsia and eclampsia.

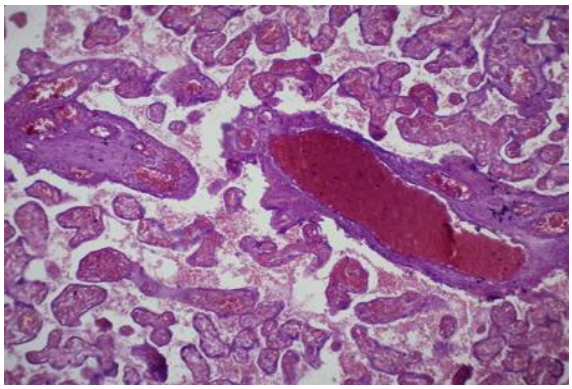

Figure 1: H & E image of Intra-villous Heamorrhage (100X)

ISSN (O): 2687-5365; ISSN (P): 2753-6556

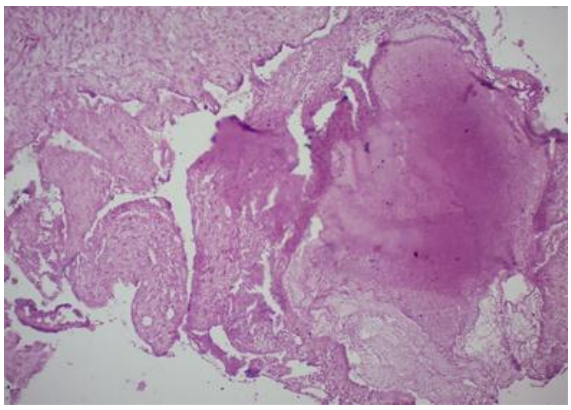

Figure 2: H & E image of Fibrinoid Necrosis (100X)

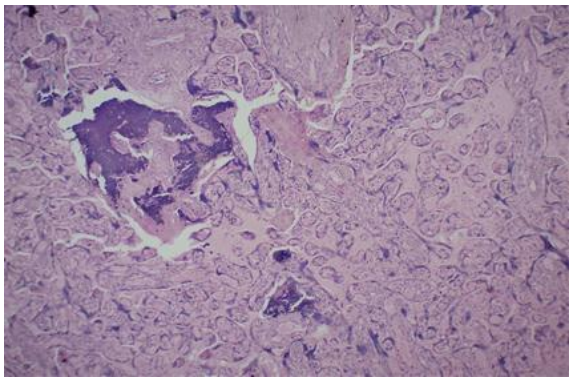

Figure 3: H & E image of Calcification (100X)

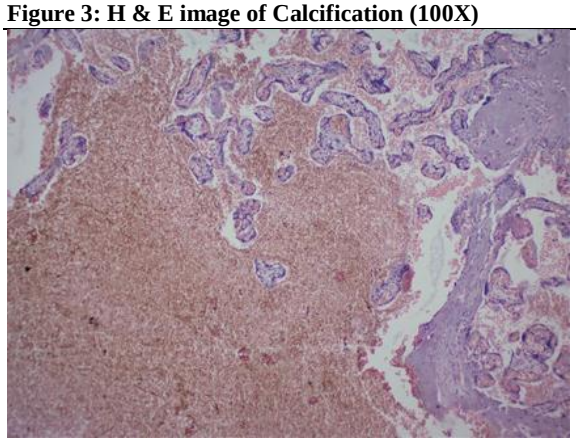

Figure 4: H & E image of Inter-villous Haemorrhage (100X)

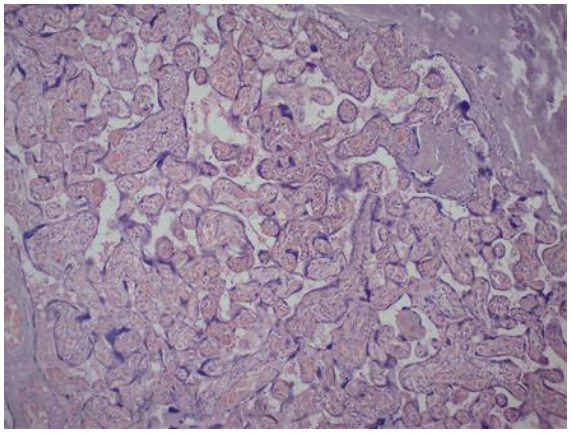

Figure 5: H & E image of Syncytial Knot formation (100X)

DISCUSSION

The results indicated that a majority of preeclampsia and eclampsia cases occurred at or near full-term gestation, with the largest group delivering at 39 weeks (43.14%), followed by those at 38 weeks (23.53%) and 37 weeks of geststion (21.57%). This aligns with the general understanding that hypertensive disorders such as preeclampsia and eclampsia are more commonly diagnosed closer to term.[7] Similarly, studies conducted by Askar et al,[8] have shown that the onset of these conditions tends to occur in the third trimester, particularly in the late stages of pregnancy, which may be linked to the increased placental demand and insufficient placental remodeling. In contrast, cases at earlier gestations (such as 32 weeks and 34 weeks) were less frequent, which suggests that while preeclampsia and eclampsia can occasionally arise earlier, these conditions are more commonly associated with later gestation periods.

The study's findings on past medical conditions highlighted that nearly half of the women (48.04%) had no previous illnesses, while those with pre-existing conditions such as anemia (21.57%), high BMI (12.75%), and hypertension (11.76%) were more likely to develop preeclampsia or eclampsia. Samaddar et al,[9] observed that women with hypertension and increased BMI had significantly higher rates of placental abnormalities and adverse pregnancy outcomes. Additionally, the presence of anemia, a common co-morbidity in pregnant women, also correlates with a higher risk of preeclampsia, as seen in studies by Gore et al,[10] who reported an increased incidence of adverse placental changes, including infarction and calcification, in women with preexisting conditions. The high prevalence of these conditions in the study population suggested that women with a history of these health issues should receive extra care and close monitoring during pregnancy to reduce the risk of hypertensive complications.

The study revealed that systolic blood pressure (SBP) was significantly higher in cases of eclampsia (142.20 mmHg), mild preeclampsia (146.24 mmHg), and severe preeclampsia (144.79 mmHg) compared to the normal group (117.2 mmHg). The difference in SBP between the groups was statistically significant with a p-value of <0.001, emphasizing the direct impact of preeclampsia and eclampsia on blood pressure regulation. Similar findings were reported by Panday et al,[11] who also found that women with hypertensive disorders of pregnancy exhibited significantly higher SBP compared to normal pregnancies, correlating the rise in BP with adverse placental and fetal outcomes. Elevated SBP in eclampsia and preeclampsia reflects compromised placental perfusion and endothelial dysfunction, which ultimately results in systemic hypertension. These findings underscore the importance of close

ISSN (O): 2687-5365; ISSN (P): 2753-6556

blood pressure monitoring and early interventions to prevent severe maternal and fetal complications.

The diastolic blood pressure (DBP) values were also significantly higher in eclampsia (101.00 mmHg), mild preeclampsia (99.80 mmHg), and severe preeclampsia (99.56 mmHg) when compared to the normal group (77.8 mmHg), with a p-value of <0.001. This is consistent with the well-established association between preeclampsia, eclampsia, and elevated blood pressure. Khilji et al,[12] also reported higher DBP in pre-eclamptic pregnancies, noting that diastolic hypertension was a prominent feature of placental dysfunction. The increase in DBP suggested endothelial damage and poor vascular adaptation in response to increased placental demands, making DBP a critical marker for managing hypertensive disorders in pregnancy. This finding underscores the necessity of early detection and treatment to prevent severe complications such as eclampsia and organ failure.

A significant association was found between gestational age and the pattern of hypertensive complications during pregnancy ($p < 0.001$). Pregnancies between 32 and 36 weeks were mostly uncomplicated. Most women had normal outcomes, with only a few cases of mild or severe preeclampsia and no cases of eclampsia. However, a noticeable increase in disease burden occurred after 37 weeks, where all eclampsia cases and most cases of mild and severe preeclampsia were recorded. Severe preeclampsia was most common at 39 weeks (52.63%). Eclampsia and mild preeclampsia peaked between 37 and 38 weeks, showing that more severe cases cluster near term.

These findings align with earlier reports indicating that hypertensive disorders of pregnancy are more likely to occur and worsen in late gestation. Sibai et al,[13] noted that most cases of preeclampsia develop after 34 weeks, with increasing severity as pregnancy nears term. This supports the higher rates of severe preeclampsia observed at 38–39 weeks in this study. Population data from Ananth et al,[14] similarly showed a rising incidence of hypertensive disorders beyond 37 weeks, highlighting late gestation as a critical time for disease emergence. Current ACOG guidelines also identify late-onset preeclampsia as the main clinical type. Although this type is often linked to better fetal outcomes, it poses significant risks for mothers if diagnosis or intervention is delayed.[15] The occurrence of eclampsia cases only during term gestation in this group is consistent with findings from Onuh et al,[16] who reported higher rates of eclampsia in the late third trimester, often due to delayed recognition or insufficient antenatal surveillance.

Overall, the distribution of hypertensive complications based on gestational age highlights the need for closer monitoring in pregnancies past 36 weeks. Advancing gestation seems to be a key factor in both the occurrence and severity of these complications. The study found a significant reduction in placental weight in preeclampsia and eclampsia cases compared to normal pregnancies, with eclampsia cases showing an average placental weight of 432.50 grams and normal pregnancies showing a mean weight of 538.33 grams. The significant decrease in placental weight (p-value <0.001) aligns with findings from previous studies, such as that by Samaddar et al,[9] who reported reduced placental weight in hypertensive pregnancies due to placental insufficiency and poor perfusion. This reduction in placental mass is a direct consequence of compromised blood flow and trophoblastic invasion, which results in placental dysfunction.

Infarction was found to be significantly more prevalent in preeclampsia and eclampsia cases than in normal pregnancies. Specifically, 40% of eclampsia cases and 64% of mild preeclampsia cases exhibited placental infarctions, compared to 0% in the normal pregnancy group, with a p-value of 0.006. This finding supports the research conducted by Ojha et al,[17] who found a high incidence of placental infarction in severe preeclampsia and eclampsia cases. The presence of infarction is a direct result of impaired placental blood flow, which leads to areas of ischemia and necrosis in the placenta. The significant presence of infarction in preeclampsia and eclampsia further emphasizes the critical role of placental examination in predicting and managing hypertensive disorders in pregnancy.

The presence of calcification was significantly higher in preeclampsia and eclampsia cases compared to normal pregnancies, with p-value of 0.02 indicating a statistically significant association between calcification and hypertensive disorders of pregnancy. This finding is in line with previous studies, such as that by Gore et al,[10] which also identified a higher prevalence of placental calcification in preeclampsia. The pathophysiology behind this phenomenon could be related to placental ischemia and necrosis, which causes calcium deposits in the tissues. Calcification can also be a marker of chronic placental damage due to reduced perfusion and endothelial dysfunction. Therefore, calcification is an important histopathological finding that can serve as an indicator of compromised placental function in hypertensive pregnancies.

Syncytial knot formation was notably higher in preeclampsia and eclampsia compared to normal pregnancies. The p-value of <0.001 strongly indicates the association between syncytial knot formation and hypertensive pregnancies. This finding is consistent with research by Samal et al,[7] which showed that syncytial knots were frequently observed in pre-eclamptic placentas. The formation of syncytial knots is thought to be a response to ischemic stress in the placenta, indicating poor trophoblastic differentiation and vascular insufficiency. Syncytial knots are also considered a marker of placental dysfunction and can reflect impaired placental blood flow. Thus, syncytial knot formation is a useful histopathological feature in the

ISSN (O): 2687-5365; ISSN (P): 2753-6556

assessment of placental pathology associated with hypertensive disorders of pregnancy.

Fibrinoid necrosis was significantly more common in preeclampsia and eclampsia cases, with p-value of <0.001 strongly supporting the association between fibrinoid necrosis and hypertensive disorders of pregnancy. Samal et al,[7] also reported a high incidence of fibrinoid necrosis in pre-eclamptic placentas. Fibrinoid necrosis is a result of endothelial damage and vascular malperfusion, leading to the accumulation of fibrin in the placental tissue. This pathological change is often seen in placental tissues from pregnancies with poor perfusion and hypoxia. The presence of fibrinoid necrosis is a useful diagnostic marker for assessing The severity of placental dysfunction in hypertensive pregnancies.

The study revealed a significant association between cytotrophoblastic proliferation, preeclampsia and eclampsia, with 50% of eclampsia cases showing proliferation in the 16-20 per 100 villi and 20% in the 21-25 per 100 villi. Mild and severe preeclampsia cases exhibited similar patterns, with notable cytotrophoblastic proliferation in the 16-20 per 100 villi. In contrast, no cases of cytotrophoblastic proliferation were observed in normal pregnancies. The p-value of <0.001 indicates a statistically significant relationship between cytotrophoblastic proliferation and hypertensive pregnancies. This finding is consistent with the results reported by Das et al,[18] who identified cytotrophoblastic proliferation as a hallmark feature of pre-eclamptic placentas. Cytotrophoblastic proliferation is thought to be a compensatory response to placental hypoxia and ischemia. As the placenta faces stress due to inadequate perfusion, trophoblast cells proliferate in an attempt to compensate for the reduced functionality. This histopathological change is essential in understanding the underlying pathophysiology of preeclampsia and eclampsia and can serve as a diagnostic tool for assessing placental health.

The present study showed a statistically significant association between calcified areas in placenta and hypertensive pregnancies. Askar et al,[8] also reported a higher incidence of placental calcification in preeclampsia and eclampsia cases. The calcification in the placenta is a result of chronic ischemia, which leads to the deposition of calcium in the placental tissues. This can reflect the long-term effects of placental hypoxia and dysfunction, which are characteristic of hypertensive disorders of pregnancy. The presence of calcified areas in the placenta is an important indicator of chronic placental damage and dysfunction.

The above study showed the clinical importance of placental markers, like syncytial knots and fibrinoid necrosis, in preeclampsia and eclampsia. These markers can help assess placental issues, estimate risk, and guide prompt treatments in high-risk pregnancies. Including the placental histopathology in evaluating hypertensive disorders during pregnancy can improve decision-making about monitoring and timing of delivery. It also lays the groundwork for future research on non-invasive markers for early detection and tracking. However, the study also has some limitations such as retrospective design that carries selection bias. It relies only on histopathological assessment without using other diagnostic methods. The sample size is modest, and there is a lack of long-term data on maternal and neonatal outcomes. These limitations highlight the need for larger, forward-looking, multi-center studies that use a variety of assessment methods to confirm and build on these findings.

CONCLUSION

The findings underscore the significant role that placental dysfunction plays in the development of maternal and fetal complications, including fetal growth restriction and preterm birth. Key histological markers such as infarction, calcification, syncytial knots, fibrinoid necrosis, and cytotrophoblastic proliferation were identified as significant features of preeclamptic and eclamptic placentas. These changes reflect impaired placental perfusion and trophoblast invasion, leading to poor fetal oxygenation and nutrient supply.

The study also highlights the higher prevalence of preeclampsia and eclampsia among younger women, particularly those aged 21-25, and suggests that hypertensive disorders tend to occur closer to full-term gestation. The results further suggest that while blood pressure measurements and clinical monitoring are essential, placental examination could serve as a critical tool in assessing the severity of placental damage and predicting adverse pregnancy outcomes. By identifying key histopathological changes early, clinicians can make more informed decisions regarding the timing of delivery and the management of maternal and fetal health. Overall, the study emphasizes the value of histopathological analysis in improving the management and outcomes of pregnancies complicated by hypertensive disorders.

REFERENCES

1. Kooffreh ME, Ekott M, Ekpoudom DO. The prevalence of pre-eclampsia among pregnant women in the University of Calabar Teaching Hospital, Calabar. Saudi Journal for Health Sciences. 2014 Sep 1;3(3):133-6.
2. Ghulmiyyah L, Sibai B. Maternal mortality from preeclampsia/eclampsia. In Seminars in perinatology 2012 Feb 1 (Vol. 36, No. 1, pp. 56-59). WB Saunders.
3. Mor S, Sirohiwal D, Hooda R. Eclampsia: Maternal and perinatal outcomes in a tertiary care centre. International Journal of Reproduction, Contraception, Obstetrics and Gynecology. 2015 Jun 1;4(3):653-8.
4. Vaithy A, Samal R, KR UD. Histopathological study of placental changes associated with pre-eclampsia and eclampsia in a tertiary care centre of Puducherry. IP Archives of Cytology and Histopathology Research. 2023 Jan 23;4(1):57-60.
5. Priya A, Mishra N, Sahu M, Behera A. Study of Clinical Outcome of Preeclampsia and Eclampsia in a Tertiary Care Centre. European Journal of Cardiovascular Medicine. 2023 Apr 1;13(2).

ISSN (O): 2687-5365; ISSN (P): 2753-6556

6. Dekker G, Sibai B. Primary, secondary, and tertiary prevention of pre- eclampsia. The lancet. 2001 Jan 20;357(9251):209-15.
7. Samal R, Vaithy KA, Sowmya S. Histopathological study of placental changes associated with pre-eclampsia and eclampsia in a tertiary care centre of Puducherry. IP Arch Cytol Histopathol Res. 2019;4(2):78-85.
8. Askar E, Selim SA, Sibai H. Histological changes of human placenta in early intrauterine growth restriction with and without preeclampsia. J Med Histol. 2019;3(2):126-134.
9. Samaddar A, Roy U, Saha I, Mangal S. Histopathological changes in placenta in pregnancy-induced hypertensive patients and correlation with fetal outcome – a tertiary care hospital study. Biomed Biotechnol Res J. 2021;5(3):440-445.
10. Gore C, Pandey A, Shetty A, Rao R, Paranjape S. A study on histopathological changes in placenta in pre-eclampsia/eclampsia: A case- control study in a tertiary care centre, western India. Indian J Pathol Oncol. 2020;7(1):75-81.
11. Panday A, Budania S, Bano T. The histopathological changes of placenta in hypertensive disorders of pregnancy and their correlation with fetal outcome: A cross-sectional study. Asian J Pharm Clin Res. 2023;16(7):110-116.
12. Khilji A, Rizvi SAH, Tarique S, Fatima S, Shah A, Kazmi QH, et al. Morphometric analysis of placental terminal villi, capillaries, and intervillous space in normal and pre-eclamptic pregnancies. Pak J Med Health Sci. 2023;17(2):826-832.
13. Sibai BM. Diagnosis and management of gestational hypertension and preeclampsia. Obstet Gynecol. 2003;102(1):181–192.
14. Ananth CV, Keyes KM, Wapner RJ. Pre-eclampsia rates in the United States, 1980–2010: age-period-cohort analysis. BMJ. 2013;347: f6564.
15. American College of Obstetricians and Gynecologists. Gestational hypertension and preeclampsia: ACOG Practice Bulletin No. 222. Obstet Gynecol. 2020;135(6): e237–e260.
16. Onuh SO, Aisien AO. Maternal and fetal outcome in eclamptic patients in Benin City, Nigeria. J Obstet Gynaecol. 2004;24(7):765–768.
17. Ojha K, Rawal S, Jha A. Placental pathology in severe pre-eclampsia and eclampsia. Nepal Med J. 2018;1(1):32-35.
18. Das S, Baisakh P, Panda S, Chinara P. Histopathological study of placenta in pregnancy with hypertension. J Anat Soc India. 2016; 65:195.

ISSN (O): 2687-5365; ISSN (P): 2753-6556