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# LIPID PROFILE AND HISTOPATHOLOGICAL CHANGES IN PLACENTAE OF WOMEN WITH PREECLAMPSIA

**Dr Sadia Nasir[1], Dr Sumbul Aqeeq[2*], Dr Shabana Bano Soomro[3], Dr Muhammad Razaq[4], Dr Farhan Abbas Baloch[5], Dr Tania Khattak[6]**

[1]Assistant Professor, Department of Obstetrics and Gynaecology, Khyber Teaching Hospital, Peshawar, Pakistan
[2*]Senior Lecturer, Department of Biochemistry, Kabir Medical College, Peshawar, Pakistan
[3]Assistant Professor, OBGYN Unit 3, Shaheed Muhtarma Benazir Bhutto Medical University, Chandka Medical College, Shaikh Zayed Women Hospital, Larkana, Pakistan
[4]Assistant Professor, Department of Biochemistry, Jinnah Medical College, Peshawar, Pakistan
[5]Associate Professor, Department of Pathology, Pak International Medical College, Peshawar, Pakistan
[6]Assistant Professor, Department of Histopathology, Nowshera Medical College, Nowshera, Pakistan

***Corresponding Author:** Dr Sumbul Aqeeq,
*Senior Lecturer, Department of Biochemistry, Kabir Medical College, Peshawar, Pakistan
Email: drsumbulaqeeq@gmail.com

## ABSTRACT

### Background

To assess and compare the lipid profiles and histopathological placental changes in women with preeclampsia and normotensive pregnancies.

### Methods

This cross-sectional study was conducted at Jinnah Medical College, Peshawar, from January 2022 to January 2023. A total of 71 pregnant women were enrolled 36 diagnosed with preeclampsia and 35 normotensive controls. Blood samples were taken for lipid profile analysis, including total cholesterol, triglycerides, HDL, LDL, and VLDL. Placentae collected after delivery were examined histologically for infarction, fibrinoid necrosis, syncytial knotting, thrombotic lesions, and other changes. Statistical comparisons were made using SPSS, with significance set at $p<0.05$.

### Results

'Women with preeclampsia had significantly elevated levels of total cholesterol, triglycerides, LDL, and VLDL, along with reduced HDL ($p<0.001$)'. Placental histology showed higher incidences of infarction (66.7%), fibrinoid necrosis (58.3%), thrombotic lesions (52.8%), and decidual vasculopathy (47.2%) in preeclamptic women, all statistically significant ($p<0.001$).

### Conclusion

Preeclampsia is associated with a distinct atherogenic lipid profile and substantial histopathological damage to the placenta. Routine lipid monitoring during antenatal care and pathological assessment of placentae in hypertensive pregnancies can aid in early detection and management strategies.

**Keywords**
Preeclampsia, Lipid Profile, Placental Histopathology, Triglycerides, Syncytial Knotting, Decidual Vasculopathy, Pregnancy Complications

## INTRODUCTION

Preeclampsia remains one of the leading complications of pregnancy worldwide, accounting for a significant proportion of maternal and neonatal morbidity and mortality. It typically arises after the 20th week of gestation and is characterized by elevated blood pressure, proteinuria, and multisystem involvement. Although the precise etiology remains unclear, the central role of endothelial dysfunction and abnormal placental development is well established [1-3].

Recent studies have highlighted the contribution of metabolic factors, particularly maternal dyslipidemia, in the pathogenesis of preeclampsia. Alterations in lipid metabolism not only exacerbate oxidative stress and inflammation but also influence vascular tone and placental perfusion. Elevated triglycerides and low levels of protective lipoproteins such as HDL are consistently reported in women with hypertensive pregnancies [4-6].

The placenta plays a crucial role in fetal development, and its structure reflects the intrauterine environment. In preeclampsia, placental changes such as infarction, fibrinoid necrosis, thrombotic vasculopathy, and abnormal syncytial knotting have been documented, indicating impaired maternal-fetal circulation and hypoxia [7-9].

Given these observations, this study aimed to compare the lipid profiles and histopathological features of placentae in women with and without preeclampsia. Understanding this association may provide insight into early diagnostic indicators and the underlying mechanisms of placental injury in preeclamptic pregnancies.

## METHODOLOGY

This analytical cross-sectional study was conducted at the Department of Obstetrics and Gynecology, Jinnah Medical College, Peshawar, over a one-year period from January 2022 to January 2023. The research aimed to assess the alterations in lipid profiles and placental histopathological changes among women diagnosed with preeclampsia and compare them with normotensive pregnant women. Ethical approval for the study was obtained from the Institutional Review Board of Jinnah Medical College, Peshawar, prior to commencement.

A total of 71 pregnant women admitted for delivery were enrolled through non-probability consecutive sampling. Participants were divided into two groups: 36 women with clinically diagnosed preeclampsia formed the study group, while 35 normotensive women served as the control group. ‘The diagnosis of preeclampsia was based on criteria established by the American College of Obstetricians and Gynecologists (ACOG), which include blood pressure ≥140/90 mmHg on two occasions at least four hours apart after 20 weeks of gestation, accompanied by proteinuria or signs of end-organ dysfunction’.

Women with chronic hypertension, gestational diabetes, renal disorders, or other systemic illnesses were excluded to minimize confounding factors. After obtaining informed written consent, relevant demographic and clinical data were recorded using a structured proforma.

Prior to delivery, fasting blood samples were collected ‘to assess lipid parameters including total cholesterol, triglycerides, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very-low-density lipoprotein (VLDL)’. The atherogenic index was calculated as the logarithmic ratio of triglycerides to HDL (log[TG/HDL]). All biochemical analyses were performed in the hospital laboratory using standardized enzymatic methods.

Following delivery, the placentae were collected immediately, cleaned with saline, and fixed in 10% formalin. Histopathological examination was conducted by a qualified pathologist blinded to the clinical grouping. Sections from maternal and fetal surfaces were processed, stained with hematoxylin and eosin (H&E), and evaluated under a light microscope. Features such as villous infarction, fibrinoid necrosis, syncytial knotting, thrombotic lesions, intervillous fibrin deposition, decidual vasculopathy, and calcification were systematically assessed.

All data were entered and analyzed using SPSS version 25.0. 'Continuous variables such as lipid levels were presented as mean ± standard deviation and compared using the independent sample t-test'. 'Categorical variables like histopathological findings were expressed as frequencies and percentages, and analyzed using the Chi-square test'. A p-value of less than 0.05 was considered statistically significant.

## RESULTS

The study included a total of 71 participants, with 36 women in the preeclamptic group and 35 in the normotensive group. The majority of participants in both groups were over 25 years of age, although the difference was not statistically significant (p = 0.66). Primigravida status was slightly more common among women with preeclampsia (55.6%) compared to the normotensive group (48.6%), but this was not significant (p = 0.55). Notably, gestational age at delivery was significantly lower in the preeclamptic group, with 50% delivering before 37 weeks compared to only 22.9% in the normotensive group (p = 0.01). Similarly, the proportion of women with a BMI of 25 or higher was significantly greater in the preeclampsia group (61.1%) than in the control group (37.1%) (p = 0.04). Cesarean deliveries were significantly more frequent among preeclamptic women (66.7%) compared to normotensive women (34.3%) (p = 0.006).

**Table 1: Demographic Characteristics of Study Participants (n = 71)**

| Variable | Preeclampsia (n=36) | Normotensive (n=35) | p-value |
|---|---|---|---|
| Age ≤25 years | 12 (33.3%) | 10 (28.6%) | 0.66 |
| Age >25 years | 24 (66.7%) | 25 (71.4%) | |
| Primigravida | 20 (55.6%) | 17 (48.6%) | 0.55 |
| Multigravida | 16 (44.4%) | 18 (51.4%) | |
| Gestational age <37 weeks | 18 (50.0%) | 8 (22.9%) | 0.01* |
| Gestational age ≥37 weeks | 18 (50.0%) | 27 (77.1%) | |
| BMI ≥25 | 22 (61.1%) | 13 (37.1%) | 0.04* |
| Cesarean delivery | 24 (66.7%) | 12 (34.3%) | 0.006* |

*Statistically significant at p<0.05

Women diagnosed with preeclampsia showed significantly altered lipid profiles compared to the normotensive group. Mean total cholesterol levels were markedly higher in the preeclamptic group (224.3 ± 32.1 mg/dL) versus the normotensive group (188.6 ± 28.9 mg/dL), with a p-value <0.001. Similarly, triglycerides and LDL were elevated in preeclamptic women, while HDL was significantly lower (38.5 ± 6.8 mg/dL vs. 47.1 ± 7.2 mg/dL, p <0.001). The atherogenic index, which provides insight into cardiovascular risk, was also significantly higher in the preeclampsia group (p <0.001). These findings reflect a pronounced dyslipidemic pattern associated with preeclampsia.

**Table 2: Comparison of Lipid Profile Between Preeclampsia and Normotensive Groups**

| Lipid Parameter | Preeclampsia (Mean ± SD) | Normotensive (Mean ± SD) | p-value |
|---|---|---|---|
| Total Cholesterol (mg/dL) | 224.3 ± 32.1 | 188.6 ± 28.9 | <0.001* |
| Triglycerides (mg/dL) | 196.4 ± 35.5 | 151.8 ± 30.7 | <0.001* |
| HDL (mg/dL) | 38.5 ± 6.8 | 47.1 ± 7.2 | <0.001* |
| LDL (mg/dL) | 135.6 ± 27.4 | 110.2 ± 24.9 | 0.002* |
| VLDL (mg/dL) | 39.2 ± 7.1 | 30.4 ± 6.2 | <0.001* |
| Atherogenic Index (log(TG/HDL)) | 0.71 ± 0.15 | 0.50 ± 0.12 | <0.001* |

*Statistically significant at p<0.05

Histological examination of the placentae revealed significantly more abnormalities in the preeclamptic group. Villous infarctions were observed in 66.7% of preeclamptic placentae compared to only 17.1% in the normotensive group (p <0.001). Similarly, fibrinoid necrosis, syncytial knotting, and thrombotic lesions were all significantly more prevalent in women with preeclampsia (p <0.001 for each). Increased fibrin deposition and decidual vasculopathy were also more common in the preeclampsia group, indicating severe placental ischemia and vascular compromise. These pathological changes highlight the extent of placental damage in hypertensive pregnancies.

**Table 3: Histopathological Placental Changes in Preeclampsia vs. Normotensive Group**

| Histopathological Feature | Preeclampsia (n=36) | Normotensive (n=35) | p-value |
|---|---|---|---|
| Villous Infarction | 24 (66.7%) | 6 (17.1%) | <0.001* |
| Fibrinoid Necrosis | 21 (58.3%) | 5 (14.3%) | <0.001* |
| Syncytial Knotting (Moderate/Severe) | 27 (75.0%) | 10 (28.6%) | <0.001* |
| Thrombotic Lesions | 19 (52.8%) | 4 (11.4%) | <0.001* |
| Increased Intervillous Fibrin | 23 (63.9%) | 8 (22.9%) | <0.001* |
| Decidual Vasculopathy | 17 (47.2%) | 3 (8.6%) | <0.001* |
| Placental Calcification (Mod-Severe) | 20 (55.6%) | 6 (17.1%) | <0.001* |

*Statistically significant at p<0.05

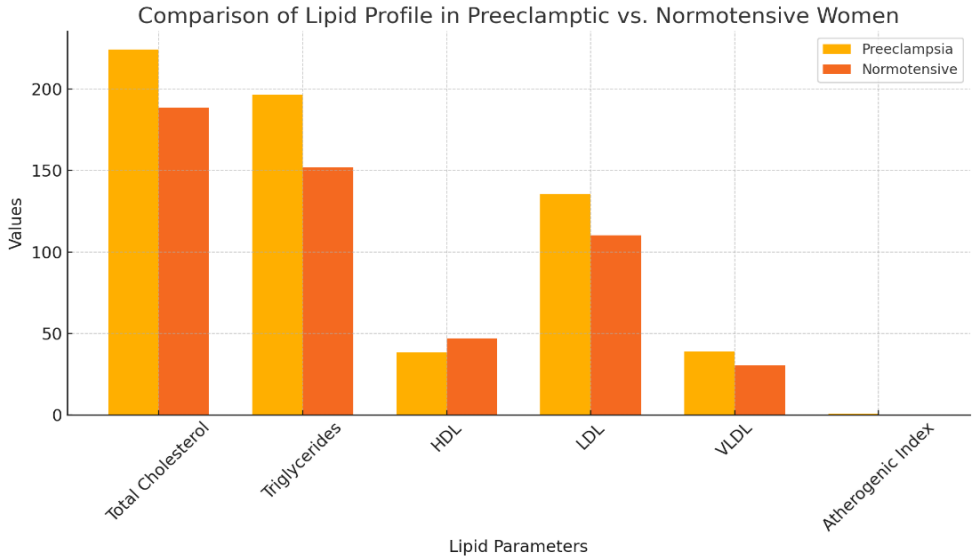


**Figure 1**

bar graph comparing lipid profile parameters between preeclamptic and normotensive women.

## DISCUSSION

This study explored the association between preeclampsia, dyslipidemia, and placental histopathological changes in pregnant women. The findings demonstrate a significant alteration in lipid profile and a marked increase in pathological changes within the placentae of women affected by preeclampsia compared to normotensive controls.

'Elevated levels of total cholesterol, triglycerides, LDL, and VLDL along with reduced HDL were significantly associated with preeclamptic women'. These lipid abnormalities mirror a pattern of atherogenic dyslipidemia, which is increasingly recognized as a contributing factor in the pathogenesis of preeclampsia. Studies confirmed that dyslipidemia, particularly hypertriglyceridemia, contributes to endothelial dysfunction and oxidative stress, leading to the vascular abnormalities seen in preeclampsia [10-12]. Similarly, studies found elevated triglycerides and lower HDL levels in preeclamptic women, suggesting their potential as early biomarkers [13, 14].

Our results also align with the findings of study, who reported increased lipid peroxidation and oxidative stress markers in preeclampsia, indicating the biochemical burden that promotes vascular injury in placental tissue [15].

In terms of placental histology, the study revealed a significantly higher frequency of villous infarction, fibrinoid necrosis, syncytial knotting, thrombotic lesions, and decidual vasculopathy in preeclamptic placentae. These features are indicative of uteroplacental insufficiency and impaired maternal-fetal exchange. Study reported similar findings, highlighting that placental ischemia and infarction are hallmarks of hypertensive disorders of pregnancy [16, 17]. In addition, study emphasized the role of maternal vascular malperfusion in promoting syncytial knot formation and intervillous fibrin deposition [18].

Calcification and extensive fibrin deposition in our study further support the concept of placental aging and chronic hypoxic injury in preeclampsia. This corresponds with the work of study, who noted a higher incidence of calcific deposits and necrotic areas in the placentae of women with hypertensive pregnancies [19].

Our findings support the hypothesis that preeclampsia is a systemic syndrome involving endothelial dysfunction, pro-inflammatory states, and poor placental perfusion, as described by study. This makes both lipid profile and placental histology valuable tools for identifying and understanding the extent of maternal and fetal compromise in preeclamptic cases [20].

While this study adds to the growing evidence, its limitation lies in its single-center design and moderate sample size, which may affect generalizability. Future research involving larger, multi-center cohorts and follow-up on neonatal outcomes is recommended.

## CONCLUSION

This study found a significant association between abnormal lipid profiles and histopathological changes in the placentae of women with preeclampsia. Elevated total cholesterol, triglycerides, and LDL, along with decreased HDL levels, were prominently seen in preeclamptic patients. Histologically, their placentae demonstrated signs of ischemic damage such as infarction, necrosis, thrombi, and calcification.

These findings suggest that routine lipid profiling and placental evaluation can aid in the early detection and risk stratification of preeclampsia. A better understanding of these changes may also help clinicians adopt preventive and targeted management strategies to improve maternal and fetal outcomes.

## REFERENCES

1. Franchuk, U., et al., Histological analysis of the placenta in patients with preeclampsia, taking into account the proposed treatment. 2022. **29**(2): p. 207-213.
2. Obiri, D., et al., Histopathological lesions and exposure to Plasmodium falciparum infections in the placenta increases the risk of preeclampsia among pregnant women. 2020. **10**(1): p. 8280.
3. Bhojwani, K., A. Agrawal, and A.K.J.C. Agrawal, Study of histopathological changes in the placenta in preeclampsia. 2022. **14**(10).
4. Khaire, A.A., et al., Placental lipid metabolism in preeclampsia. 2021. **39**(1): p. 127-134.
5. Özgökçe, Ç., et al., Histopathological, ultrastructural, and immunohistochemical examination of changes in the placenta as a result of severe preeclampsia. 2023. **38**: p. e382023.
6. Dahabiyeh, L.A., et al., Investigating the molecular structure of placenta and plasma in pre-eclampsia by infrared microspectroscopy. 2020. **184**: p. 113186.

7. Usta, A., et al., Placental fractalkine immunoreactivity in preeclampsia and its correlation with histopathological changes in the placenta and adverse pregnancy outcomes. 2020. **33**(5): p. 806-815.
8. Franchuk, U.Y., S. Khmil, and Y.M.J.R.o.V.N.M.U. Orel, Histological aspects of remodeling of the placental complex in pregnancy complicated by late preeclampsia with different options for its correction. 2021. **25**(3): p. 394-397.
9. Lombó, M., et al., Preeclampsia correlates with an increase in cannabinoid receptor 1 levels leading to macromolecular alterations in chorionic villi of term placenta. 2022. **23**(21): p. 12931.
10. Ali, H. and L.J.A.o.t.C.o.M.M. Al-Allaf, Evaluation of the histology and the oxidative stress status in the placentas of obese mothers. 2021. **43**(1): p. 60.0-69.0.
11. Brosens, I., et al., Acute atherosis and diffuse lipid infiltration of the placental bed: A review of historical lipid studies. 2020. **97**: p. 36-41.
12. Zhang, L., et al., Integrated metabolomic and lipidomic analysis in the placenta of preeclampsia. 2022. **13**: p. 807583.
13. Essiben, F., et al., Histopathological Aspects of Placental Lesions in Mild and Severe Pre-Eclampsia in a Population of Cameroonian Women. 2022. **12**(2): p. 154-168.
14. de Moreuil, C., et al., Correlation of biological parameters with placental parameters and pregnancy outcomes in pre-eclamptic women. 2020. **19**: p. 61-66.
15. Kashish, B. and A.J.C. Anil, Study of Histopathological Changes in the Placenta in Preeclampsia. 2022. **14**(10).
16. Konrad, E., et al., Correlation of elevated levels of lipoprotein (a), high-density lipoprotein and low-density lipoprotein with severity of preeclampsia: a prospective longitudinal study. 2020. **40**(1): p. 53-58.
17. Pietro, L., et al., Placental findings in preterm and term preeclampsia: an integrative review of the literature. 2021. **43**(07): p. 560-569.
18. Degryse, M., THE ASSOCIATION BETWEEN ANATOMOPATHOLOGICAL ABNORMALITIES OF THE PLACENTA AND PRE-ECLAMPSIA. 2023, Ghent University.
19. Balachandiran, M., et al., Placental accumulation of triacylglycerols in gestational diabetes mellitus and its association with altered fetal growth are related to the differential expressions of proteins of lipid metabolism. 2021. **129**(11): p. 803-812.
20. Rani, A., et al., Region-specific changes in the mRNA and protein expression of LCPUFA biosynthesis enzymes and transporters in the placentae of women with preeclampsia. 2020. **95**: p. 33-43.