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## Histopathological Examination of Placenta in Patients with Pre-Eclampsia/Eclampsia and Aberrant Thyroid Profiles

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### Abstract

**Background:** Pre-eclampsia and eclampsia are leading causes of maternal and perinatal morbidity and mortality. Abnormal thyroid hormone levels during pregnancy have been implicated in worsening placental pathology and fetal outcomes. Histopathological examination of the placenta offers valuable insights into the combined effects of these conditions.

**Aim:** To assess the histomorphological changes of the placenta in association with abnormal thyroid hormone levels in patients with pre-eclampsia/eclampsia and their relationship with neonatal birth weight.

**Material and Methods:** This prospective observational study included 50 pregnant women (33 controls and 17 cases) at a tertiary care hospital. Controls were normotensive women with normal thyroid profiles, while cases were women with pre-eclampsia/eclampsia and deranged thyroid function. Placental samples were evaluated for infarction, syncytial knots, chorangiosis,

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haemorrhage, and calcification. Birth weights were recorded and correlated with placental findings.

**Results:** Cases showed a higher frequency of infarction (52.9%), advanced syncytial knots (type 2a), and haemorrhage (82.4%) compared to controls. Calcification was more common in controls (51.5%). Most babies in the case group had birth weights between 1.5–2.5 kg, while controls had a higher proportion of normal birth weights (2.5–3.5 kg). These findings highlight the compounded placental injury and fetal compromise associated with thyroid dysfunction and hypertensive disorders.

**Conclusion:** Deranged thyroid profiles in pre-eclamptic and eclamptic pregnancies are associated with significant placental histopathological changes and lower neonatal birth weights. Early detection and management of thyroid abnormalities may improve maternal and fetal outcomes.

**Keywords:** Pre-eclampsia, eclampsia, placenta, histopathology, thyroid dysfunction, birth weight, pregnancy outcome

## Introduction

Pre-eclampsia and eclampsia remain among the most significant contributors to maternal and perinatal morbidity and mortality worldwide, especially in developing countries like India. These hypertensive disorders of pregnancy are characterized by

multisystem involvement, with the placenta playing a central role in their pathophysiology [1]. The placenta is a dynamic organ responsible for fetal growth, nutrient exchange, and hormonal regulation,



and its structural integrity is vital for a healthy pregnancy outcome.

One important hormonal system implicated in pregnancy outcomes is the thyroid axis. Maternal thyroid hormones play an essential role in fetal neurodevelopment, growth, and placental development, particularly in the first and second trimesters [2]. Derangements in maternal thyroid function, such as hypothyroidism and subclinical hypothyroidism, have been associated with increased risk of gestational hypertension, pre-eclampsia, fetal growth restriction (FGR), and low birth weight (LBW) [3,4].

Histopathological examination of the placenta provides crucial insights into the adaptive and pathological changes in response to maternal diseases. Studies have demonstrated that pre-eclamptic placentas often show features like infarcts, increased

syncytial knots, fibrinoid necrosis, villous stromal fibrosis, and abnormal vascularity, reflecting uteroplacental insufficiency [5]. Thyroid dysfunction, through its influence on placental metabolism and vascular remodeling, can further exacerbate these placental abnormalities [6].

Recent evidence suggests that thyroid hormones modulate placental angiogenesis, trophoblast proliferation, and apoptotic processes, thereby influencing placental development and fetal outcomes [7]. The interplay between thyroid abnormalities and hypertensive disorders of pregnancy could lead to compounded placental injury, worsening fetal growth restriction and low birth weight [8,9]. Despite this, limited research has explored the combined impact of thyroid dysfunction and pre-eclampsia/eclampsia on placental



histopathology, particularly in the Indian context where nutritional and environmental factors further complicate maternal-fetal health [10].

This study aims to assess the histomorphological changes in the placenta in relation to abnormal maternal thyroid profiles in patients with pre-eclampsia/eclampsia and evaluate their association with neonatal birth weight. This research will contribute to better understanding of the placental mechanisms underlying adverse pregnancy outcomes and may guide antenatal surveillance and management strategies.

### Material and Methods

This was a prospective, observational, comparative study conducted at the Department of Pathology and Obstetrics & Gynaecology at tertiary care hospital in India.

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A total of 50 pregnant women were enrolled, divided into two groups:

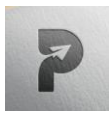
- Controls: 33 normotensive pregnant women with normal thyroid profiles.
- Cases: 17 pregnant women diagnosed with pre-eclampsia or eclampsia with deranged thyroid profiles.

#### Inclusion Criteria:

- Singleton pregnancy.
- Gestational age  $\geq 28$  weeks at delivery.
- Known thyroid hormone levels (TSH, FT3, FT4) during pregnancy.
- Pre-eclampsia/eclampsia diagnosed as per the American College of Obstetricians and Gynecologists (ACOG) criteria.

#### Exclusion Criteria:

- Multiple pregnancies.
- Gestational diabetes mellitus.



- Known fetal congenital anomalies.
  - Chronic hypertension or chronic kidney disease.
  - Women with pre-existing thyroid disease on treatment.
  - Sections (4–5  $\mu$ m) were stained with hematoxylin and eosin (H&E) for histopathological examination.
- The following parameters were assessed:
- Syncytial knots, cytotrophoblastic proliferation.

#### Data Collection and Sample Processing:

- Detailed maternal history, clinical examination, blood pressure records, thyroid profile results, and neonatal birth weight were documented.
  - Villous stromal fibrosis, fibrinoid necrosis.
  - Infarction, intervillous fibrin deposition.
  - Maternal and fetal vascular changes.
  - Calcification, villous edema.
- After delivery, placentas were collected fresh and examined grossly for weight, shape, size, infarcts, calcification, and retroplacental clots.
- Tissue samples were taken from standardized sites (central, peripheral, and any abnormal area), fixed in 10% formalin, processed, and embedded in paraffin blocks.
- Outcome Measures:
- Correlation of placental histopathological features with maternal thyroid hormone levels.
  - Association between histopathological findings and birth weight of the baby.

#### Statistical Analysis:



Data was analyzed using SPSS software. Continuous variables were expressed as mean  $\pm$  SD and compared using Student's t-test or ANOVA. Categorical variables were compared using the chi-square test or Fisher's exact test. Pearson correlation was used to assess the relationship between thyroid profile, histopathological changes, and birth weight. A p-value  $<0.05$  was considered statistically significant.

## Results

Table 1 shows the distribution of study participants based on their TSH levels. Out of the total 50 participants, 33 (66%) were categorized as controls with normal thyroid-stimulating hormone (TSH) levels ranging between 0.3–3.1 mIU/L, while 17 (34%) were classified as cases with elevated TSH levels ( $>3.1$  mIU/L). This distribution highlights that about one-third of the

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participants with pre-eclampsia/eclampsia were associated with deranged thyroid profiles.

Table 2 presents the final clinical diagnosis of the mothers. Among the controls, the most common conditions were previous LSCS (4), post-term delivery (5), and oligohydramnios (5), while pre-eclampsia/eclampsia was seen in 7 controls (21.2%). In contrast, among the cases, pre-eclampsia/eclampsia predominated, affecting 13 mothers (76.5%). Other conditions like anemia, IUGR, and placenta previa were also observed in both groups, though at lower frequencies.

Table 3 displays the distribution of placental weight among controls and cases. In the control group, most placentas weighed between 300–400 gms (39.4%) and 400–500 gms (18.2%), while the cases showed a slightly lower proportion in the 300–400 gms



range (35.3%) and higher proportions in the 400–500 gms (29.4%) and 500–600 gms (17.6%) ranges. Lower placental weights (<300 gms) were less common in both groups but slightly more observed in the controls.

Table 4 shows the histomorphological features of the placenta. Among controls, infarction was seen in 36.4%, chorangiosis in 42.4%, haemorrhage in 48.5%, and calcification in 51.5%. In contrast, the cases showed higher rates of infarction (52.9%), more advanced syncytial knots (notably type-2a), and a markedly higher incidence of

haemorrhage (82.4%) but lower calcification (29.4%). These findings reflect greater placental compromise in the cases.

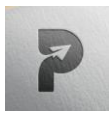
Table 5 presents the distribution of birth weights. Among cases, most babies weighed between 1.5–2.5 kg (10 babies), with 2 babies under 1.5 kg. In the control group, most babies were between 2.5–3.5 kg (17 babies), with only 3 babies below 1.5 kg. These results show a clear trend toward lower birth weights among babies born to mothers with deranged thyroid profiles and hypertensive disorders.

**Table 1: Distribution of Controls & Cases**

TSH Level	Controls (TSH = 0.3–3.1 mIU/L)	Cases (TSH > 3.1 mIU/L)	Total (n = 50)
Number (%)	33 (66%)	17 (34%)	50 (100%)

**Table 2: Final Clinical Diagnosis of the Mother**

Final Diagnosis of Mother	Controls (n = 33)	Cases (n = 17)
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Pre-eclampsia/Eclampsia	7 (21.2%)	13 (76.5%)
Oligohydramnios	5	2
IUGR	3	2
Previous LSCS	4	3
Contracted Pelvis/CPD	3	0
Anemia	3	2
Placenta Previa	0	1
HELLP Syndrome	1	0
Post-Term Delivery	5	0
IUD	1	0
Others	1	2

**Table 3: Placental Weight**

Weight (gms)	Controls (n = 33)	Cases (n = 17)
<100	1	0
100–200	2	1
200–300	7	2
300–400	13 (39.4%)	6 (35.3%)
400–500	6 (18.2%)	5 (29.4%)





500–600	4 (12.1%)	3 (17.6%)
Total	33	17

**Table 4: Histomorphology of Placenta**

Feature	Controls (n = 33)	Cases (n = 17)
Infarction	12 (36.4%)	9 (52.9%)
Syncytial Knots	Type-1: 18	Type-1: 3
	Type-2a: 6	Type-2a: 10
	Type-2b: 2	Type-2b: 1
Chorangiomas	14 (42.4%)	7 (41.2%)
Haemorrhage	16 (48.5%)	14 (82.4%)
Calcification	17 (51.5%)	5 (29.4%)

**Table 5: Birth Weight of Baby**

Birth Weight (Kg)	Cases (n = 17)	Controls (n = 33)
<1.5	2	3
1.5–2.5	10	9
2.5–3.5	4	17
>3.5	1	4



Total	17	33
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## Discussion

This study explored the histomorphological changes of the placenta in association with deranged thyroid profiles among patients with pre-eclampsia/eclampsia, correlating these findings with neonatal birth weight. Our results highlight a significant relationship between abnormal thyroid function, placental pathology, and adverse fetal outcomes.

We found a higher frequency of infarction, syncytial knot formation (especially type-2a), and haemorrhage in the placentas of cases compared to controls. These histopathological changes reflect impaired utero-placental blood flow and maternal vascular malperfusion, consistent with the known pathophysiology of pre-eclampsia

and eclampsia [11]. Similar observations were reported by Aggarwal et al., who noted increased placental infarcts and villous changes in hypertensive disorders of pregnancy [12].

The addition of thyroid dysfunction further aggravated placental pathology. Thyroid hormones are critical regulators of placental development, influencing angiogenesis, trophoblast invasion, and nutrient transport [13]. Abnormal maternal thyroid status, particularly hypothyroidism, has been linked to defective placentation, increased oxidative stress, and impaired remodeling of spiral arteries, exacerbating placental insufficiency [14]. Our findings align with studies by Karakosta et al. and Korevaar et al., who



reported significant associations between hypothyroidism, pre-eclampsia, and low birth weight [15,16].

Regarding fetal outcomes, our study showed that birth weights were notably lower in the case group, with most babies weighing 1.5–2.5 kg or less. This is consistent with prior studies demonstrating that maternal thyroid dysfunction and hypertensive states compromise fetal growth through combined effects on placental perfusion and metabolism [17]. Monitoring and correcting thyroid abnormalities during pregnancy may therefore represent an important intervention to improve maternal and neonatal outcomes. Importantly, placental histopathology serves as a window into the maternal-fetal environment and provides valuable insights into the mechanisms underlying adverse pregnancy outcomes. Incorporating placental

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examination into routine evaluation, especially in high-risk pregnancies, can aid in refining perinatal care and guide preventive strategies.

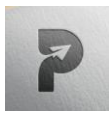
### **Conclusion**

In conclusion, our study demonstrates that deranged thyroid profiles in patients with pre-eclampsia/eclampsia are associated with more severe placental histopathological changes, including increased infarction, haemorrhage, and advanced syncytial knot formation. These changes are closely linked to lower neonatal birth weights, highlighting the compounded effect of thyroid dysfunction and hypertensive disorders on pregnancy outcomes. Early detection and management of thyroid abnormalities during pregnancy may help mitigate placental injury and improve fetal health.



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