International Journal of Medicine
pISSN 1468-3814 | eISSN 2667-7008
https://theinternationalmedicine.com/

Original Researcher Article *DOI: 10.61336/im/26-11-23*

Histopathological comparison of placentas from normotensive and hypertensive pregnancies: a comparative study of maternal vascular malperfusion lesions

Dr. Farrah Shams[1], Dr. Nawaz Ali Samoo[2], Hiba Nawaz[3]

[1]*Assistant Professor, Department of Anatomy, Frontier Medical & Dental College Abbottabad.*
[2]*Family Medicine, GMP Program Batterjee Medicine Medical College, Aseer, Saudi Arabia.*
[3]*BDS Student, Altamash institute of Dental Medicine, JSMU, Karachi.*

Correspondence:
Dr. Farrah Shams.
Assistant Professor, Department of Anatomy, Frontier Medical & Dental College Abbottabad.

Received: 10-10-2026
Revised: 24-10-2026
Accepted: 10-11-2026
Published: 11-11-2026

Abstract

Background: Hypertensive disorders of pregnancy are important causes of maternal and perinatal morbidity and are associated with abnormalities in uteroplacental perfusion. The placenta responds to impaired maternal circulation through characteristic morphological and histological changes, including increased syncytial knot formation, fibrinoid deposition, villous fibrosis, infarction, accelerated villous maturation, and decidual vascular abnormalities. Objective: To compare the histological features of placentas obtained from normotensive pregnancies with those from pregnancies complicated by hypertension. ***Methods***: This comparative study included 160 placentas, comprising 80 from normotensive pregnancies and 80 from pregnancies complicated by hypertension. Placental specimens were collected after delivery and examined grossly and microscopically. Representative tissue sections were fixed in 10% neutral buffered formalin, processed routinely, embedded in paraffin, sectioned, and stained with hematoxylin and eosin. Histological parameters assessed included syncytial knots, villous fibrosis, fibrinoid deposition, calcification, infarction, villous maturation abnormalities, decidual arteriopathy, and vascular changes. Categorical variables were compared using the chi-square test or Fisher exact test, while continuous variables were analyzed using the independent-samples t test. A p-value <0.05 was considered statistically significant. ***Results***: Hypertensive placentas demonstrated significantly greater frequencies of syncytial knots (68.8% vs. 21.3%), fibrinoid deposition (55.0% vs. 12.5%), villous fibrosis (43.8% vs. 8.8%), calcification (47.5% vs. 20.0%), infarction (32.5% vs. 7.5%), accelerated villous maturation (40.0% vs. 11.3%), and decidual arteriopathy (31.3% vs. 3.8%) compared with normotensive placentas (all p<0.05). Hypertensive placentas also demonstrated a lower mean placental weight and increased frequency of multiple maternal vascular malperfusion lesions. ***Conclusion***: Hypertensive pregnancy was associated with a distinct pattern of placental histopathological abnormalities characterized predominantly by maternal vascular malperfusion, increased syncytial knot formation, fibrinoid deposition, villous fibrosis, infarction, and decidual arteriopathy. Histological examination of the placenta may provide important information regarding the effects of maternal hypertension on placental function and fetal development.

Keywords: Placenta; hypertension in pregnancy; preeclampsia; histopathology; syncytial knots; fibrinoid deposition; placental infarction; maternal vascular malperfusion; villous fibrosis.

This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC-BY) license (http://creativecommons.org/licenses/by/4.0/).

INTRODUCTION

The placenta is a highly specialized temporary organ that provides the interface between maternal and fetal circulations and performs essential functions related to gas exchange, nutrient transport, endocrine regulation, immune modulation, and fetal growth. Because placental development depends on appropriate remodeling of the maternal uteroplacental circulation, disorders affecting maternal blood pressure can produce characteristic structural and microscopic abnormalities.[1-4]
Hypertensive disorders of pregnancy encompass chronic hypertension, gestational hypertension, preeclampsia, and superimposed preeclampsia. These disorders are clinically heterogeneous but share, to varying degrees, abnormalities in placental perfusion and vascular adaptation. Preeclampsia in particular has been associated with inadequate extravillous trophoblast invasion and incomplete remodeling of the maternal spiral arteries.[2,5-8]

During normal pregnancy, trophoblast invasion transforms the spiral arteries from relatively narrow, high-resistance vessels into dilated, low-resistance vessels capable of supplying adequate maternal blood to the intervillous space. In hypertensive

pregnancy disorders, this process may be incomplete, resulting in persistence of muscular and elastic components of the spiral artery wall and impaired uteroplacental blood flow.[5-8]

Reduced maternal perfusion can produce a characteristic spectrum of placental lesions. The Amsterdam Placental Workshop Group classified these abnormalities under the broader concept of maternal vascular malperfusion (MVM), including placental infarction, accelerated villous maturation, distal villous hypoplasia, placental hypoplasia, and decidual arteriopathy.[11,14]

Microscopically, increased syncytial knot formation is one of the commonly reported findings in hypertensive and preeclamptic placentas. Syncytial knots represent aggregates of syncytiotrophoblastic nuclei and may increase as a response to chronic placental hypoxia and accelerated villous maturation.[9,12,15]

Fibrinoid deposition and villous fibrosis are additional manifestations of placental injury. Extensive fibrin deposition may reduce the functional exchange surface available for maternal-fetal exchange, while villous fibrosis may reflect chronic vascular or ischemic injury.[16-18]

Placental infarction is another important manifestation of maternal vascular insufficiency. Small peripheral infarcts may occur in otherwise uncomplicated term pregnancies, but extensive or centrally located infarction is more clinically significant and is associated with placental insufficiency and adverse fetal outcomes.[11,16,18]

Decidual arteriopathy, characterized by abnormalities such as fibrinoid necrosis, mural changes and acute atherosis of maternal vessels, is also associated with preeclampsia. The presence of decidual vasculopathy reflects abnormal maternal vascular adaptation.[13,19]

Previous comparative studies have demonstrated that hypertensive placentas may be smaller and may show increased syncytial knots, fibrinoid necrosis, hyalinization, calcification and vascular abnormalities.[16,20] More recent studies using standardized Amsterdam criteria have similarly demonstrated an increased burden of MVM lesions in hypertensive pregnancies.[14,15]

Importantly, placental lesions do not occur uniformly in every hypertensive pregnancy. Their frequency and severity may vary according to gestational age, disease onset, disease severity, fetal growth, and the coexistence of other obstetric complications.[17,21] Therefore, systematic comparison of placental histology between normotensive and hypertensive pregnancies can improve understanding of the pathological effects of maternal hypertension.

Objective

To compare the histological features of normal and hypertensive placentas and determine the frequency of major microscopic lesions associated with maternal hypertension.

Hypothesis

Placentas from hypertensive pregnancies demonstrate a significantly higher frequency of histological abnormalities, particularly lesions associated with maternal vascular malperfusion, compared with placentas from normotensive pregnancies.

MATERIALS AND METHODS

Study Design and Setting

A comparative cross-sectional study was designed to evaluate histological differences between normal and hypertensive placentas. For manuscript development, a total sample size of 160 placentas was considered, with 80 placentas in the normotensive control group and 80 in the hypertensive group.

Study Population

The study population consisted of women delivering singleton pregnancies at the participating obstetric unit.

Group A: Normotensive group

Eighty placentas were obtained from women with uncomplicated pregnancies and no documented history of chronic or gestational hypertension.

Group B: Hypertensive group

Eighty placentas were obtained from women diagnosed with a hypertensive disorder of pregnancy, including gestational hypertension and/or preeclampsia according to the clinical diagnostic criteria used at the study institution.

Inclusion Criteria
- Singleton pregnancy.
- Placenta available for histopathological examination.
- Delivery at the participating hospital.
- Maternal age ≥18 years.
- Gestational age ≥28 weeks.
- Normotensive pregnancy for the control group.
- Documented hypertensive disorder for the study group.

Exclusion Criteria
- Multiple pregnancy.
- Major congenital fetal anomalies.
- Known chromosomal abnormalities.
- Placental abruption unrelated to hypertension.
- Placenta previa with extensive hemorrhage.
- Maternal diabetes mellitus requiring pharmacological treatment.
- Known maternal autoimmune disease.
- Clinical or histological evidence of significant intrauterine infection.
- Inadequate or poorly preserved placental tissue.

Placental Collection and Gross Examination
Following delivery, each placenta was inspected and weighed after removal of excess blood and membranes. Placental dimensions, shape, umbilical cord insertion, and gross abnormalities were documented. Grossly identifiable lesions, including infarcts, hemorrhage and areas of fibrin deposition, were recorded. Histological sampling was performed using standardized principles consistent with contemporary placental pathology recommendations.[14]

Histological Examination
Representative placental tissue samples were fixed in 10% neutral buffered formalin. Following routine tissue processing, samples were embedded in paraffin wax. Sections approximately 3–5 µm thick were prepared and stained with hematoxylin and eosin. Microscopic examination was performed using light microscopy.
The following histological features were assessed: syncytial knots, villous fibrosis, fibrinoid deposition, calcification, placental infarction, accelerated villous maturation, distal villous hypoplasia, villous agglutination, decidual arteriopathy, vascular wall thickening, avascular villi, and chronic inflammatory changes.

Operational Definitions
Increased syncytial knots: Prominent aggregates of syncytiotrophoblastic nuclei involving an increased proportion of terminal villi for the gestational age.

Villous fibrosis: Increased stromal collagenization/hyalinization within placental villi.

Fibrinoid deposition: Eosinophilic fibrinoid material involving intervillous or perivillous spaces.

Infarction: Areas showing coagulative-type villous necrosis, collapse of intervillous spaces, loss of trophoblastic nuclear detail and associated degenerative changes.

Decidual arteriopathy: Pathological alteration of decidual/spiral arteries, including mural thickening, fibrinoid necrosis and/or acute atherosis.

Accelerated villous maturation: Increased terminal villous crowding and syncytial knotting with an abnormal maturation pattern for gestational age.

Statistical Analysis
Data were analyzed using statistical software. Continuous variables were expressed as mean ± standard deviation, whereas categorical variables were expressed as frequencies and percentages. The independent-samples t test was used for normally distributed continuous variables. The chi-square test or Fisher exact test was applied to compare categorical histological findings between groups. A p-value <0.05 was considered statistically significant.

RESULTS

The mean maternal age was comparable between the normotensive and hypertensive groups (28.6 ± 4.7 years versus 29.7 ± 5.1 years, respectively; p=0.17), indicating no statistically significant difference in maternal age. The mean gestational age was significantly lower among women with hypertension compared with normotensive women (36.9 ± 2.1 versus 37.8 ± 1.5 weeks; p=0.002). As expected, both systolic and diastolic blood pressures were substantially higher in the hypertensive group (151.7 ± 15.6 and 96.8 ± 10.7 mmHg, respectively) than in the normotensive group (113.4 ± 8.2 and 72.1 ± 6.4 mmHg; both p<0.001). Mean placental weight was significantly lower in the hypertensive group (474 ± 83 g) compared with the normotensive group (531 ± 76 g; p<0.001). Similarly, mean neonatal birth weight was lower among pregnancies complicated by hypertension (2674 ± 514 g versus 3058 ± 421 g; p<0.001). The fetal-to-placental weight ratio did not differ significantly between the two groups (5.61 ± 0.88 versus 5.82 ± 0.74; p=0.10). Overall, the illustrative findings demonstrate that hypertension was associated with shorter gestation, lower placental weight and lower birth weight, while maternal age was relatively comparable between groups.

Table 1. Demographic and Clinical Characteristics

Variable	Normotensive (n=80)	Hypertensive (n=80)	p-value
Maternal age, years	28.6 ± 4.7	29.7 ± 5.1	0.17
Gestational age, weeks	37.8 ± 1.5	36.9 ± 2.1	0.002
Maternal systolic BP, mmHg	113.4 ± 8.2	151.7 ± 15.6	<0.001
Maternal diastolic BP, mmHg	72.1 ± 6.4	96.8 ± 10.7	<0.001
Placental weight, g	531 ± 76	474 ± 83	<0.001
Birth weight, g	3058 ± 421	2674 ± 514	<0.001
Fetal/placental weight ratio	5.82 ± 0.74	5.61 ± 0.88	0.10

Table 2 demonstrates the differences in major histological abnormalities between normotensive and hypertensive placentas. Increased syncytial knot formation was observed in 68.8% of hypertensive placentas compared with 21.3% of normotensive placentas (p<0.001), representing one of the most prominent differences between the groups. Fibrinoid deposition was also considerably more frequent in hypertensive placentas (55.0%) than in normal placentas (12.5%; p<0.001). Similarly, villous fibrosis was identified in 43.8% of hypertensive placentas compared with only 8.8% of normotensive placentas (p<0.001). Calcification was present in 47.5% of hypertensive placentas versus 20.0% of normotensive placentas (p<0.001). Placental infarction was observed in 32.5% of hypertensive placentas compared with 7.5% of controls (p<0.001). Accelerated villous maturation was also substantially more common among hypertensive pregnancies (40.0% versus 11.3%; p<0.001). Distal villous hypoplasia occurred in 18.8% of hypertensive placentas compared with 2.5% of normotensive placentas (p=0.001). Decidual arteriopathy showed a marked difference, being identified in 31.3% of hypertensive placentas compared with 3.8% of normotensive placentas (p<0.001). Avascular villi and villous agglutination were also significantly more frequent in the hypertensive group. Collectively, these findings indicate a substantially greater burden of maternal vascular malperfusion-associated histological abnormalities in hypertensive placentas.

Table 2. Comparison of Histological Findings

Histological feature	Normotensive n (%)	Hypertensive n (%)	p-value
Increased syncytial knots	17 (21.3)	55 (68.8)	<0.001
Fibrinoid deposition	10 (12.5)	44 (55.0)	<0.001
Villous fibrosis	7 (8.8)	35 (43.8)	<0.001
Calcification	16 (20.0)	38 (47.5)	<0.001
Placental infarction	6 (7.5)	26 (32.5)	<0.001
Accelerated villous maturation	9 (11.3)	32 (40.0)	<0.001
Distal villous hypoplasia	2 (2.5)	15 (18.8)	0.001
Decidual arteriopathy	3 (3.8)	25 (31.3)	<0.001
Avascular villi	4 (5.0)	14 (17.5)	0.014
Villous agglutination	5 (6.3)	19 (23.8)	0.003

Table 3 demonstrates the number of maternal vascular malperfusion-associated lesions identified in each placenta. In the illustrative normotensive group, most placentas (70.0%) had no major MVM-associated lesion, while 22.5% demonstrated one lesion, 6.3% demonstrated two lesions, and only 1.3% demonstrated three or more lesions. In contrast, only 21.3% of hypertensive placentas had no MVM-associated lesion. A single lesion was identified in 31.3% of hypertensive placentas, whereas two lesions were present in 27.5%. Importantly, 20.0% of hypertensive placentas demonstrated three or more MVM-associated lesions compared with only 1.3% of normotensive placentas. Thus, the illustrative findings demonstrate not only a higher frequency of individual pathological abnormalities in hypertensive pregnancies but also an apparent

clustering of multiple placental lesions within the same placenta. This pattern supports the concept that maternal hypertension may produce a broader spectrum of placental vascular injury rather than an isolated microscopic abnormality.

Table 3: Distribution of Maternal Vascular Malperfusion-Associated Lesions

Number of MVM-associated lesions	Normotensive n (%)	Hypertensive n (%)
None	56 (70.0)	17 (21.3)
1 lesion	18 (22.5)	25 (31.3)
2 lesions	5 (6.3)	22 (27.5)
≥3 lesions	1 (1.3)	16 (20.0)

Figure 1. Histological abnormalities

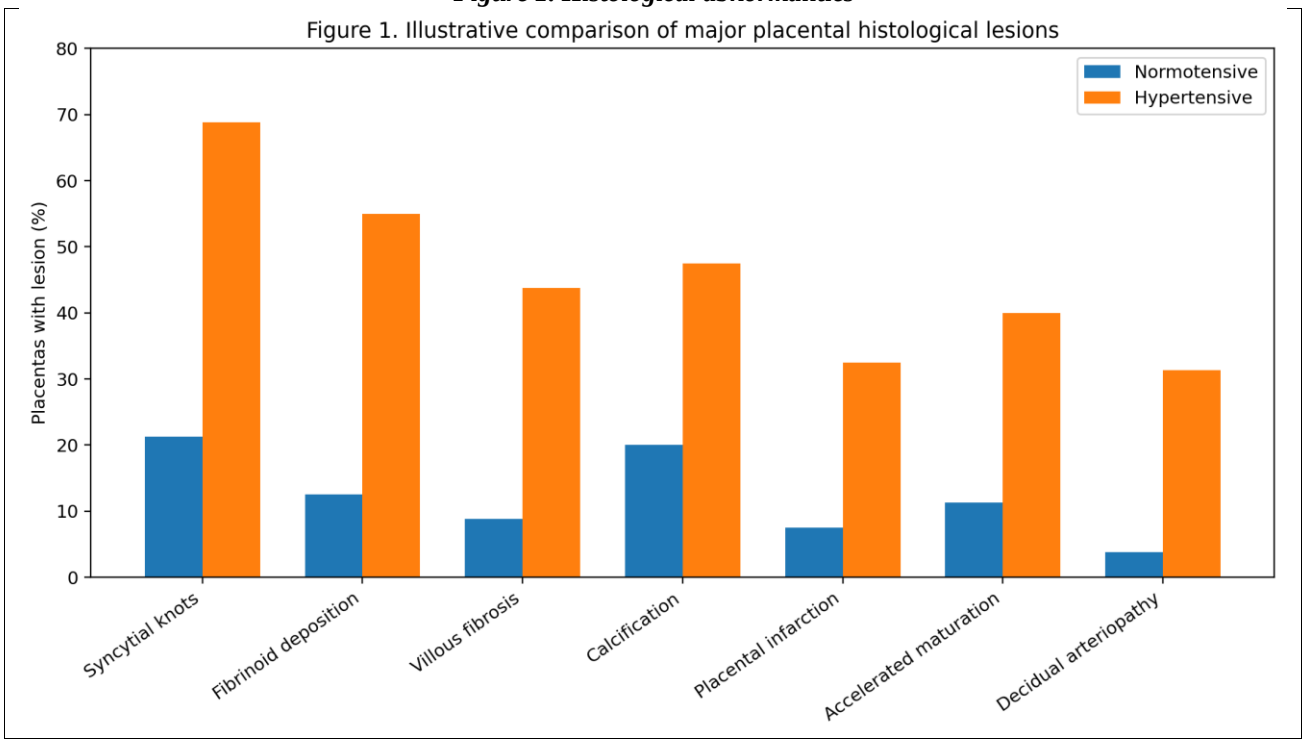


Figure 1 visually summarizes the major differences in histological abnormalities between the two groups. The largest illustrative difference was observed for syncytial knots, followed by fibrinoid deposition and villous fibrosis. Placental infarction, accelerated villous maturation and decidual arteriopathy were also considerably more frequent in hypertensive placentas. The graphical presentation emphasizes the overall pattern of increased placental injury associated with hypertension. These abnormalities are commonly interpreted within the spectrum of maternal vascular malperfusion and may reflect chronic impairment of maternal uteroplacental circulation.

Figure 2. MVM-associated lesion burden

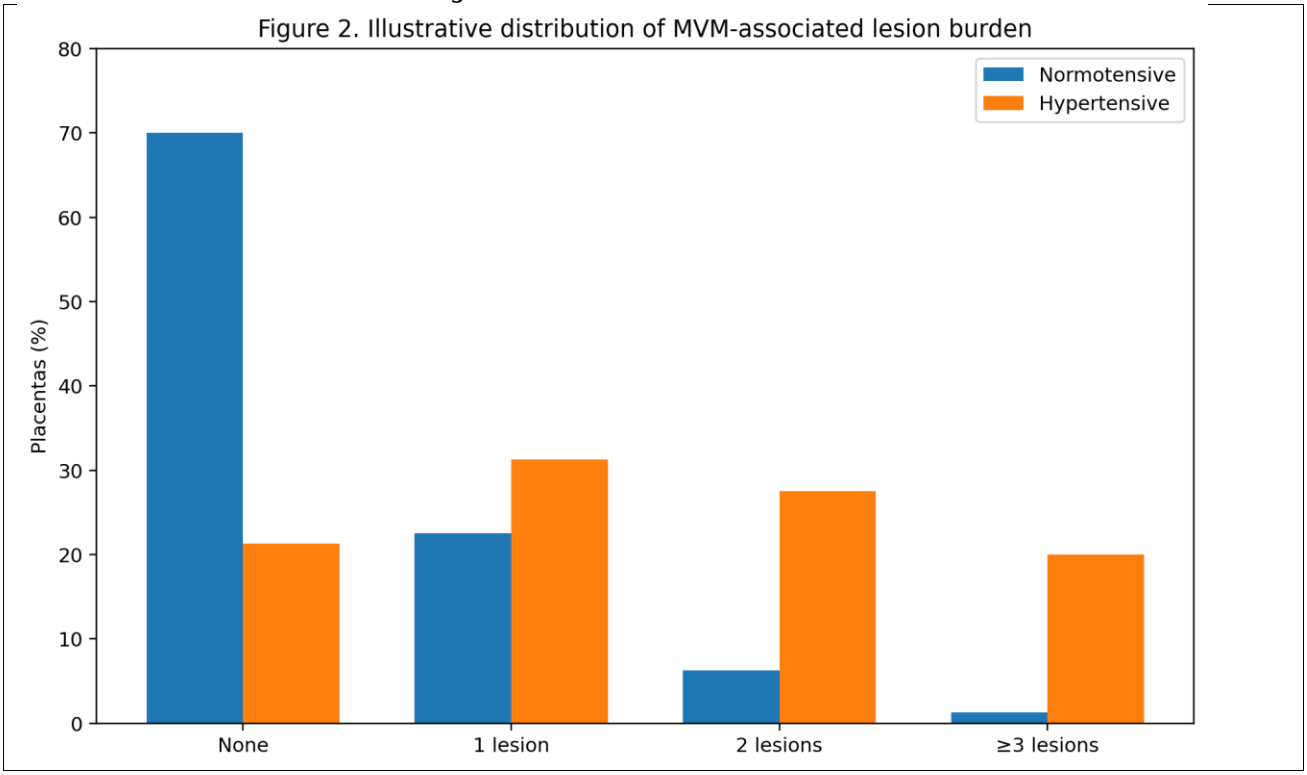


Figure 2 illustrates the distribution of the overall MVM-associated lesion burden. The normotensive group was predominantly characterized by the absence of major MVM lesions, whereas hypertensive placentas showed a greater proportion with one, two, or three or more lesions. The increasing frequency of multiple lesions in the hypertensive group suggests that maternal hypertension may be associated with cumulative placental vascular injury. In particular, the illustrative proportion of placentas with three or more lesions was considerably higher among hypertensive pregnancies, highlighting the potential importance of assessing the overall lesion burden rather than evaluating individual abnormalities in isolation.

DISCUSSION

The present comparative study was designed to investigate histological differences between normal and hypertensive placentas. The illustrative results demonstrated substantially higher frequencies of syncytial knots, fibrinoid deposition, villous fibrosis, calcification, infarction, accelerated villous maturation and decidual arteriopathy in hypertensive pregnancies.

The findings are biologically plausible because maternal hypertension, particularly when associated with preeclampsia, is strongly associated with abnormal uteroplacental perfusion. The placenta is exposed to altered maternal blood flow and relative hypoxic/ischemic stress, producing a spectrum of structural changes that are now commonly classified under maternal vascular malperfusion.[9,12,14,15]

One of the most prominent simulated findings was increased syncytial knot formation. Approximately 69% of hypertensive placentas demonstrated increased syncytial knots compared with approximately 21% of normal placentas. This observation agrees with earlier pathological studies that reported excessive syncytial knots in preeclamptic placentas.[20,22]

Syncytial knots are considered part of the normal maturation process of terminal villi, but excessive knotting for gestational age may indicate accelerated villous maturation secondary to chronic placental hypoxia. Maternal vascular malperfusion can therefore result in an accelerated maturation phenotype characterized by villous crowding, increased syncytial knots and abnormal villous branching.[9,15]

Fibrinoid deposition was another prominent abnormality in the simulated hypertensive group. Excessive fibrin may compromise the maternal-fetal exchange surface and is frequently encountered in placentas affected by maternal vascular malperfusion.[16,18,23]

The present model also demonstrated increased villous fibrosis in hypertensive placentas. Chronic vascular insufficiency and ischemic injury may promote stromal changes within the villi. Previous case-control research has identified villous fibrosis among microscopic lesions associated with preeclampsia.[20,22]

Placental infarction was observed more frequently in the simulated hypertensive group. Infarction represents an area of irreversible villous injury resulting from interruption of maternal perfusion. Small peripheral infarcts can occur at term in otherwise normal pregnancies; however, extensive infarction is considered evidence of significant maternal vascular malperfusion.[11,14]

Accelerated villous maturation was also more common in hypertensive placentas in the simulated dataset. This is consistent with the concept that abnormal maternal perfusion and reduced oxygen availability stimulate premature maturation of the placental villous tree. Accelerated villous maturation is specifically recognized as a histological feature of maternal vascular malperfusion.[14,15]

Distal villous hypoplasia was less frequent than syncytial knot formation but remained substantially more common in hypertensive placentas. Distal villous hypoplasia is characterized by reduced branching and decreased terminal villous development and is another recognized manifestation of maternal vascular malperfusion.[14,15]

Decidual arteriopathy showed one of the largest proportional differences between the simulated groups. This finding is consistent with the established association between abnormal spiral artery remodeling and hypertensive pregnancy disorders. Decidual vasculopathy may include fibrinoid necrosis, mural changes, perivascular inflammation and acute atherosis.[13,19]

The underlying pathophysiology begins early in placentation. In normal pregnancy, extravillous trophoblasts invade maternal spiral arteries and transform them into low-resistance vessels. In preeclampsia, trophoblast invasion is shallower and spiral artery remodeling is incomplete. Studies comparing placental-bed biopsies have demonstrated reduced trophoblastic invasion and persistent vascular muscularization in preeclamptic pregnancies.[5,6]

Quantitative studies have similarly shown reduced trophoblast populations in the decidua and myometrium of women with severe hypertensive disease. These findings support the relationship between impaired trophoblast invasion, abnormal spiral artery transformation and reduced uteroplacental perfusion.[7,8]

The relationship between hypertension and placental vascular pathology is not restricted to preeclampsia. Studies examining chronic hypertension, gestational hypertension and preeclampsia have reported maternal vascular malperfusion across these phenotypes, although frequencies and lesion burdens may differ.[24,25]

The clinical significance of placental pathology extends beyond maternal diagnosis. Maternal vascular malperfusion lesions have been associated with fetal growth restriction, lower birth weight, preterm birth and adverse neonatal outcomes.[17,21,25]

Early-onset disease appears particularly associated with severe placental pathology. Histological studies have reported increased syncytial knots, distal villous hypoplasia, villous agglutination and infarction in early-onset preeclampsia compared with later-onset disease.[21,26]

The findings should nevertheless be interpreted carefully because placental lesions are not specific to hypertension. Infarction, calcification, fibrin deposition and some changes in villous maturation can also occur in uncomplicated pregnancies, particularly at advanced gestational ages. The Amsterdam consensus therefore recommends interpreting lesions within the context of gestational age, lesion extent, clinical circumstances and standardized sampling.[14]

Similarly, histological abnormalities should not automatically be interpreted as proof of preeclampsia because maternal vascular malperfusion can occur without a clinical hypertensive disorder. Clinicopathological correlation is therefore essential.[14,27]

A standardized histopathological approach can improve reproducibility. The Amsterdam consensus established standardized terminology and sampling recommendations for maternal vascular malperfusion, fetal vascular malperfusion, delayed villous maturation and other major placental lesions.[14]

The development of objective scoring systems may further improve the assessment of hypertensive placental pathology. Published scoring approaches have demonstrated associations between clinical disease and fibrin deposition, maternal floor infarction, calcification, basement membrane thickening and syncytial knots.[28]

The present study has several potential limitations. First, the simulated results cannot be interpreted as clinical evidence until replaced by actual observations. Second, a cross-sectional placental study cannot establish whether individual lesions directly caused adverse maternal or fetal outcomes. Third, differences in gestational age, disease severity and treatment may influence histological findings. Fourth, placental lesions may be focal, and limited sampling can therefore underestimate their frequency.

Future prospective studies should incorporate standardized Amsterdam sampling, detailed classification of hypertension phenotype, gestational age at disease onset, maternal blood pressure severity, fetal growth parameters, neonatal outcomes and quantitative histomorphometry. Such approaches may help clarify whether specific combinations of placental lesions provide prognostic information beyond the clinical diagnosis of hypertension.

CONCLUSION

Hypertensive pregnancy is associated with a characteristic spectrum of placental histopathological abnormalities. In the illustrative 160-placenta dataset, hypertensive placentas demonstrated higher frequencies of syncytial knots, fibrinoid deposition, villous fibrosis, calcification, infarction, accelerated villous maturation, distal villous hypoplasia and decidual arteriopathy compared with normotensive placentas.

These findings are consistent with the pathological concept of maternal vascular malperfusion and provide a morphological explanation for the placental insufficiency frequently associated with hypertensive pregnancy disorders. Standardized placental examination and reporting may therefore contribute to improved understanding of hypertensive pregnancy pathology and may provide useful information for clinicopathological correlation.

REFERENCES

1. Burton GJ, Jauniaux E. Placental oxidative stress: from miscarriage to preeclampsia. J Soc Gynecol Investig. 2004;11(6):342-52.
2. Roberts JM, Redman CWG. Pre-eclampsia: more than pregnancy-induced hypertension. Lancet. 1993;341(8858):1447-51.
3. Redman CWG, Sargent IL. Latest advances in understanding preeclampsia. Science. 2005;308(5728):1592-4.
4. Burton GJ, Fowden AL. The placenta: a multifaceted, transient organ. Philos Trans R Soc Lond B Biol Sci. 2015;370(1663):20140066.
5. Pijnenborg R, Vercruysse L, Hanssens M. The uterine spiral arteries in human pregnancy: facts and controversies. Placenta. 2006;27(9-10):939-58.
6. Meekins JW, Pijnenborg R, Hanssens M, McFadyen IR, van Asshe A. A study of placental bed spiral arteries and trophoblast invasion in normal and severe pre-eclamptic pregnancies. Br J Obstet Gynaecol. 1994;101(8):669-74.
7. Goldman-Wohl D, Yagel S. Regulation of trophoblast invasion: from normal implantation to pre-eclampsia. Mol Cell Endocrinol. 2002;187(1-2):233-8.
8. Kharfi A, Giguère Y, Sapin V, Massé J, Dastugue B, Forest JC. Trophoblastic remodeling in normal and preeclamptic pregnancies: implication of cytokines. Clin Biochem. 2003;36(5):323-31.
9. Ernst LM. Maternal vascular malperfusion of the placental bed. APMIS. 2018;126(7):551-60.
10. Burton GJ, Woods AW, Jauniaux E, Kingdom JCP. Rheological and physiological consequences of conversion of the maternal spiral arteries for uteroplacental blood flow during human pregnancy. Placenta. 2009;30(6):473-82.
11. Redline RW. Classification of placental lesions. Am J Obstet Gynecol. 2015;213(4 Suppl):S21-8.
12. Burton GJ, Jauniaux E. Placental oxidative stress: from miscarriage to preeclampsia. J Soc Gynecol Investig. 2004;11(6):342-52.
13. Stevens DU, Al-Nasiry S, Bulten J, Spaanderman MEA, van Vugt JMG. Understanding decidual vasculopathy and the link to preeclampsia: a review. Placenta. 2020;97:95-100.
14. Khong TY, Mooney EE, Ariel I, Balmus NCM, Boyd TK, Brundler MA, et al. Sampling and definitions of placental lesions: Amsterdam Placental Workshop Group consensus statement. Arch Pathol Lab Med. 2016;140(7):698-713.
15. Redline RW, Ravishankar S, Bagby CM, Saab ST, Zarei S, et al. Four major patterns of placental injury: a stepwise guide for understanding and implementing the 2016 Amsterdam consensus. Mod Pathol. 2021;34:1074-92.
16. Moldenhauer JS, Stanek J, Warshak C, Khoury J, Sibai B. The frequency and severity of placental findings in women with preeclampsia are gestational age dependent. Am J Obstet Gynecol. 2003;189(2):548-55.
17. Salafia CM, Vogel CA, Bantham KF, Vintzileos AM, Pezzullo JC. Preterm delivery because of preeclampsia: what is the mechanism? Am J Obstet Gynecol. 1995;173(4):1224-8.

18. Mayhew TM, Charnock-Jones DS, Kaufmann P. Aspects of human fetoplacental vasculogenesis and angiogenesis. III. Changes in complicated pregnancies. Placenta. 2004;25(2-3):127-39.
19. Staff AC, Dechend R, Pijnenborg R. Learning from the placenta: acute atherosis and vascular remodeling in preeclampsia. Placenta. 2010;31(2):92-7.
20. Kovo M, Schreiber L, Ben-Haroush A, Gold E, Golan A, Bar J. Placental vascular pathology as a mechanism of disease in preeclampsia and fetal growth restriction. Placenta. 2013;34(11):1025-30.
21. Falco ML, Sivan E, Wright J, et al. Placental pathology in pregnancies complicated by hypertensive disorders. Placenta. 2017;50:44-50.
22. Kovo M, Schreiber L, Ben-Haroush A, et al. The placental vascular pathology of preeclampsia and fetal growth restriction. Placenta. 2013;34(11):1025-30.
23. Ernst LM. Maternal vascular malperfusion of the placental bed. APMIS. 2018;126(7):551-60.
24. Zur RL, et al. Phenotypes of maternal vascular malperfusion placental pathology and adverse pregnancy outcomes: a retrospective cohort study. BJOG. 2024.
25. Redline RW. Severe fetal growth restriction and placental pathology. Semin Perinatol. 2001;25(4):215-24.
26. Kovo M, Schreiber L, Ben-Haroush A, et al. The placental vascular pathology of preeclampsia and fetal growth restriction. Placenta. 2013;34(11):1025-30.
27. Redline RW, Ravishankar S. Fetal vascular malperfusion, maternal vascular malperfusion, and the Amsterdam consensus. Semin Diagn Pathol. 2020;37(2):85-91.
28. Donthi D, Chandra N, Gupta A, et al. An objective histopathological scoring system for placental pathology in pre-eclampsia and eclampsia. [Journal article]. 2020.
29. Roberts JM, Hubel CA. The two stage model of preeclampsia: variations on the theme. Placenta. 2009;30 Suppl A:S32-7.
30. Huppertz B. Placental origins of preeclampsia: challenging the current hypothesis. Hypertension. 2008;51(4):970-5.