American Journal of Medicine and Medical Sciences 2026, 16(1): 166-169
DOI: 10.5923/j.ajmms.20261601.37

Association Between Clinical Factors and Morphological Placental Damage in Premature Placental Abruption in High-Risk Pregnant Women

Ortiqova Feruzakhon

Research Fellow, Samarkand Medical University, Samarkand, Uzbekistan

Abstract Premature abruption of a normally implanted placenta (placental abruption) was investigated as one of the most severe and prognostically unfavorable obstetric conditions in pregnant women. The aim of the study was to evaluate clinical and morphological factors associated with the development of placental abruption in women at high risk. The material included the results of a comprehensive clinico-morphological analysis of 107 women diagnosed with placental abruption at 28–36 weeks of gestation; the control group consisted of women with a physiological course of pregnancy and delivery. Morphological examination of the placentas revealed characteristic changes, including extensive intervillous hemorrhages, trophoblastic destruction with fibrinoid necrosis, as well as signs of vascular spasm and endothelial dystrophy. The findings indicate that placental abruption is a polyetiological process primarily associated with impaired vascular adaptation and structural alterations of placental tissue, highlighting the future relevance of an integrated clinico-morphological approach for early identification of high-risk patients and prevention of obstetric complications.

Keywords Premature placental abruption, Risk group, Placental morphology, Placental insufficiency, Ischemia, Vascular disorders

1. Introduction

Premature abruption of a normally implanted placenta (PANIP) remains one of the most severe and prognostically unfavorable obstetric complications, accounting for a high level of maternal and perinatal morbidity and mortality [1,2,3]. Despite improvements in antenatal surveillance, intensive care, and obstetric management, the incidence of PANIP shows no consistent tendency toward reduction, while its clinical course is often characterized by sudden onset, rapid progression of complications, and limited possibilities for early diagnosis. A key pathogenetic mechanism of PANIP is an acute disturbance of uteroplacental circulation, leading to the formation of a retroplacental hematoma, pronounced ischemia of placental tissue, and severe fetoplacental insufficiency. However, clinical manifestations frequently do not correlate with the depth and extent of morphological placental damage, which complicates objective assessment of disease severity and prediction of perinatal outcomes [4,5,6]. Complete placental formation, including growth and functional maturation, is achieved by the end of the third month of gestation. During placentation, the functional layer of the endometrium transforms into the decidual membrane, which consists of three parts: decidua basalis, forming the maternal component of the placenta; decidua parietalis, lining the uterine wall free of the embryo and preventing bleeding from lacunae; and decidua capsularis, separating the embryo from the uterine cavity. The basal decidua and capsular layer surround the chorion. In the capsular region facing the uterine cavity, chorionic villi regress and the chorion becomes smooth, whereas in the basal region facing the endometrium, villi undergo intensive branching and development. These villi are immersed in maternal blood-filled lacunae, with some forming anchoring villi that fuse with the basal endometrial layer. Accordingly, the placenta consists of a maternal part (part uterina), represented by the basal decidua and maternal blood lacunae, and a fetal part (part fetalis), represented by the villous chorion. Under modern conditions, morphological examination of the placenta acquires particular importance as an objective method for retrospective verification of PANIP, enabling the identification of both acute and chronic disturbances of placental circulation. Histological analysis allows determination of the temporal characteristics of vascular injury, assessment of ischemic and hemorrhagic changes, and evaluation of the role of vascular, thrombotic, and decidual factors in the development of placental abruption [7,8,9]. Despite numerous publications addressing clinical aspects of PANIP, morphological criteria of this complication remain insufficiently systematized, and their qualitative and

Received: Dec. 26, 2025; Accepted: Jan. 16, 2026; Published: Jan. 20, 2026
Published online at http://journal.sapub.org/ajmms

quantitative assessment is often fragmentary. Studies based on representative case series with comprehensive morphological characterization of placental tissue are particularly limited, reducing the reproducibility and practical value of available data [10,11,12]. In this context, conducting an in-depth histological investigation of placentas from pregnant women with PANIP based on a substantial clinical dataset represents a highly relevant scientific and practical task. Systematization of morphological features and determination of their diagnostic and prognostic significance will expand current understanding of the pathogenesis of this obstetric complication, improve the accuracy of morphological diagnosis, and provide a foundation for developing objective criteria for assessing the severity of placental insufficiency. The obtained results have future-oriented significance for enhancing clinico-morphological correlations, improving the quality of pathological and forensic medical conclusions, and integrating morphological criteria of PANIP into obstetric and perinatal practice in order to improve maternal and perinatal outcomes.

2. Materials and Methods

The study was based on the analysis of 107 cases of premature abruption of a normally implanted placenta observed in high-risk women aged 21 to 39 years. The control group consisted of 20 women with a physiological course of pregnancy and delivery. Clinical data included obstetric history as well as the results of laboratory and instrumental examinations (ultrasound, Doppler velocimetry, and coagulation profile). Morphological examination was performed on placental samples obtained after delivery using standard histological techniques with hematoxylin and eosin staining.

3. Results of the Study

Among the examined women, patients with a complicated course of pregnancy predominated. Arterial hypertension was identified in 20 cases (42.5%), preeclampsia in 17 (36.2%), anemia of pregnancy in 13 (27.6%), and chronic inflammatory diseases of the pelvic organs in 14 women (29.7%). Age over 30 years was noted in 15 patients (31.9%). Signs of placental insufficiency were registered in 18 women (38.3%), manifested by Doppler findings of impaired uteroplacental blood flow, including altered resistance indices of the uterine arteries. In 11 cases (23.4%), premature abruption of a normally implanted placenta was associated with hemostatic disorders and a tendency toward hypercoagulation.

Morphological examination revealed characteristic placental changes. Macroscopically, placentas often demonstrated reduced mass and thickness, with areas of hemorrhage and the presence of retroplacental hematomas. In a number of cases, placentas exceeded normal dimensions. On the maternal surface, retroplacental hematomas of varying size, dense or loose consistency, and dark cherry coloration were identified in 43 cases. Tension of the chorionic plate was observed in 15 cases. In 34 cases, placental tissue on section exhibited foci of dark red, brown, or gray-yellow coloration, reflecting ischemic and hemorrhagic alterations.

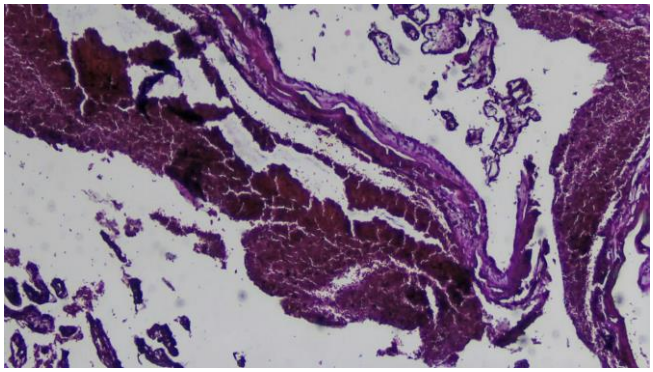

Figure 1. In the intervillous space, there are extensive accumulations of coagulated and organizing blood, fresh fibrin clots, and areas of organization. Stain: H&E. Magnification: 10x40

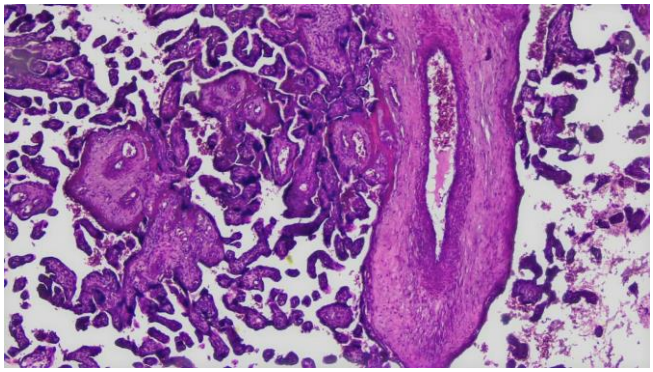

Figure 2. Formation of a retroplacental hematoma with signs of varying ages, foci of villous compression with deformation of the placental architecture, capillary congestion. Stain: H&E. Mag: 10x40

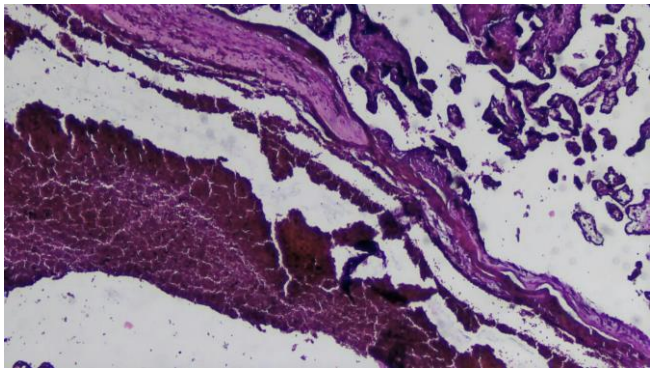

Figure 3. Extensive hemorrhage in the basal plate, decidual necrosis, damage and ruptures of decidual vessels. Stain: H&E. Mag: 10x20

In placental tissue affected by abruption, pronounced signs of acute placental ischemia were identified, including fibrinoid necrosis of vascular walls, multiple thrombi within villous vessels, and areas of placental infarction. These findings reflect severe impairment of placental perfusion. The combination of systemic clinical factors—arterial hypertension, preeclampsia, and anemia—with local

morphological alterations such as vascular spasm, villous destruction, and fibrinoid degeneration creates conditions for microcirculatory failure and retroplacental hemorrhage, ultimately leading to placental abruption.

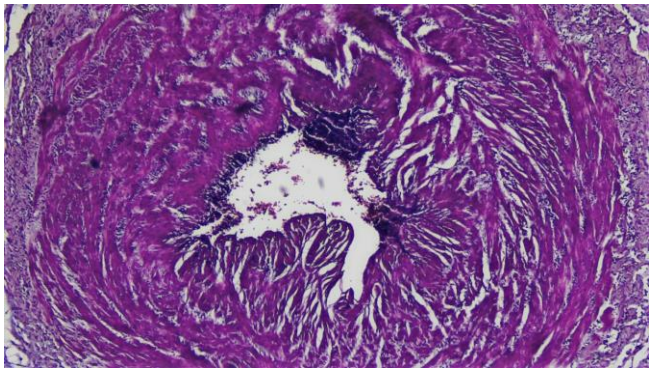

Figure 4. Extensive intervillous hemorrhages in the placental umbilical cord and pronounced spasm with dystrophic changes of vessels. Stain: H&E. Mag: 10x20

The overall morphological pattern of the placenta corresponded to premature abruption of a normally implanted placenta complicated by retroplacental hematoma formation, marked ischemia, necrosis of chorionic villi, thrombosis of decidual and villous vessels, and disruption of uteroplacental blood flow (Figs. 2 and 3).

Microscopically, extensive intervillous hemorrhages and ruptures of chorionic villi were observed, along with pronounced vascular spasm and dystrophic changes, fibrinoid degeneration of the trophoblast, hyalinization of the villous stroma, and foci of ischemia and necrosis within placental tissue. An increased number of macrophages and lymphoid infiltrates in the intervillous space was also noted (Fig. 4). These findings confirm the polyetiological nature of premature placental abruption, in which vascular and trophoblastic disturbances developing against a background of chronic hypoxia and inflammation play a central role.

Histological examination of placentas obtained from 107 pregnant women diagnosed with premature abruption of a normally implanted placenta revealed, in all cases, a complex of characteristic morphological changes reflecting acute and chronic impairment of uteroplacental circulation of varying severity. Retroplacental hematoma, as the leading morphological feature of this condition, was identified in 86 cases and was localized between the basal plate and the myometrium. Massive hemorrhages with fresh fibrin clots as well as areas of organization were detected in the intervillous space, indicating non-simultaneous vascular injury and a progressive course of placental separation.

Chorionic villi demonstrated pronounced dystrophic and necrobiotic changes. In 24 cases, ischemic coagulative necrosis of villi was observed, accompanied by destruction of the trophoblastic covering, vacuolization of the syncytiotrophoblast, and loss of clear cellular contours. In 18 cases, a marked reduction in villous vascularization, stromal sclerosis, and collapse of the vascular lumen were identified. Perivillous fibrinoid deposition was widespread and associated with villous compression, further aggravating fetoplacental circulatory impairment. In addition, areas of acute and subacute placental infarction of varying size were detected, indicating prolonged placental ischemia in a subset of patients.

Significant pathological changes were also observed in the placental vascular bed. Multiple fibrin thrombi, vascular stasis, and congestion were identified in spiral arteries and villous vessels. In several cases, fibrinoid necrosis of vascular walls with endothelial destruction was noted, reflecting severe disturbances of maternal hemodynamics and endothelial dysfunction. The decidual membrane was characterized by focal and diffuse necrosis of decidual cells, vascular ruptures, and hemorrhages of varying duration. In 19 cases, moderate lympho-macrophage infiltration was observed, suggesting the possible contribution of an inflammatory component in addition to vascular pathology.

Overall, the constellation of histological findings in all 107 cases of premature abruption of a normally implanted placenta indicates the predominance of an ischemic-hemorrhagic mechanism of placental injury with retroplacental hematoma formation, severe placental insufficiency, and profound impairment of uteroplacental and fetoplacental circulation. These morphological data provide a structural basis for adverse perinatal outcomes and fully support the clinical diagnosis of premature placental abruption.

4. Conclusions

Premature detachment of a normally located placenta in women of the risk group is caused by a combination of systemic vascular, inflammatory, and metabolic disorders. The morphological structure of the placenta in PONRP is characterized by ischemic, necrotic, and fibrinoid changes in the chorionic villi, as well as signs of chronic inflammation. Clinical-morphological evaluation of the placenta allows identifying women at high risk and applying differentiated preventive measures to reduce the frequency of obstetric complications.

REFERENCES

[1] Abduganieva D.F., Jabbarova L.A. Pregravid and prenatal risk factors for fetal growth restriction syndrome. *Tadqiqot Journal*. Tashkent, 2024.

[2] Uzokova M.K. Premature abruption of a normally implanted placenta. *Medical Journal of Uzbekistan*. 2025.

[3] Komilova M. Pathogenesis, diagnosis, and management tactics of premature abruption of a normally implanted placenta in primiparous women. *Inlibrary.uz*, 2016.

[4] Niyazmetov R.E. Features of management of massive obstetric hemorrhage (a retrospective analysis). Tashkent, 2020.

[5] Zenodo. Premature abruption of a normally implanted placenta: clinical and morphological aspects. Tashkent, 2024.

[6] Anderson E., Raja E.A., Shetty A. et al. Changing risk factors for placental abruption: a case-crossover study using routinely collected data from Finland, Malta and Aberdeen. PLOS ONE. 2020; 15(6): e0233641. DOI: 10.1371/journal.pone. 0233641.

[7] Bączkowska M., Dadarowska J., et al. Epidemiology, Risk Factors, and Perinatal Outcomes of Placental Abruption: Systematic Data Review. International Journal of Environmental Research and Public Health. 2022; 19(9): 5148. DOI: 10.3390/ijerph19095148.

[8] Chen D., et al. Independent risk factors for placental abruption: a systematic review and meta-analysis. BMC Pregnancy and Childbirth. 2025. DOI: 10.1186/s12884-025-07482-7.

[9] Zamudio S., Postigo L., et al. Human placental hypoxia-inducible factor-1α expression correlates with clinical outcomes in chronic hypoxia in vivo. Placenta. 2007; 28(8–9): 1005–1010.

[10] Rath G., et al. Expression of HIF-1 Alpha and Placental Growth Factor in Pregnancies Complicated by Hypoxia. PubMed. 2016.

[11] Tsubokura H., et al. Significance of placental CD200 expression in patients with early-onset preeclampsia. Molecular Medicine Reports. 2022; DOI: 10.3892/mmr.2022.12905.

[12] Castro P.R., et al. Cellular and molecular heterogeneity associated with placental microvasculature and angiogenesis. Wiley. 2018.