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MORPHOMETRIC FEATURES OF PLACENTAL VILLOUS DAMAGE IN PLACENTAL ABRUPTION ASSOCIATED WITH MATERNAL ANEMIA

Gulnoza M. Axmadjonova[1],
Nigina R. Nasimova[2],
Feruzaxon M. Ortikova[2]
[1] Andijan State Medical Institute, Andijan, Republic of Uzbekistan
[2] Samarkand State Medical University, Samarkand, Republic of Uzbekistan
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Introduction

Placental abruption is one of the most severe complications of pregnancy and is associated with a high risk of maternal and perinatal morbidity. Structural and vascular abnormalities of the placenta play an important role in the development of this complication. Maternal anemia may aggravate placental dysfunction through chronic tissue hypoxia, impaired uteroplacental perfusion, endothelial dysfunction, and progressive disturbances of placental microcirculation.

Morphological examination of placental tissue provides an opportunity to assess the structural consequences of these pathological processes. In particular, quantitative morphometric analysis of chorionic villi may reveal objective signs of placental dysfunction and determine the relationship between the severity of maternal anemia and placental tissue damage. The original study demonstrated progressive abnormalities of villous architecture, microcirculation, and stromal components with increasing severity of anemia.

Objective

To evaluate the morphological and morphometric features of placental villous damage in women with placental abruption associated with different degrees of maternal anemia.

Materials and Methods

The study included **157 placentas** obtained after delivery. The main group consisted of **127 women with placental abruption associated with anemia**, while the control group included **30 women with physiological pregnancy, uncomplicated delivery, and normal hemoglobin levels**.

Placental tissue was subjected to comprehensive macroscopic, histological, and morphometric examination. Tissue samples were obtained from the central and peripheral regions of the placenta, including areas adjacent to retroplacental hematomas and ischemic lesions. The specimens were fixed in 10% neutral buffered formalin, embedded in paraffin, sectioned, and stained with hematoxylin and eosin. Computer-assisted morphometry was performed using digital microscopy. At least **400 terminal villi** were evaluated in each specimen. The assessment included syncytial knots, vasculo-syncytial membranes, fibrinoid necrosis, stromal fibrosis, villous dimensions, and syncytiocapillary membrane thickness.

Statistical analysis included Student's *t*-test, χ^2 test or Fisher's exact test, and Pearson correlation analysis. Differences were considered statistically significant at **$p<0.05$**.

Results

Morphological examination demonstrated a progressive increase in placental structural abnormalities with increasing severity of maternal anemia. One of the characteristic findings was an increased number of syncytial knots, reflecting an adaptive response of the syncytiotrophoblast to chronic hypoxia. The proportion of placentas with syncytial knots involving more than 30% of villi reached **70.4% in severe anemia**, compared with **20.0% in the control group** (χ^2=23.16; **$p<0.001$**).

A marked reduction in vasculo-syncytial membranes was also observed. In the control group, vasculo-syncytial membranes involving more than 30% of villi were detected in **86.7%** of placentas,

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whereas this proportion decreased to **25.9% in severe anemia**. The intergroup differences were statistically significant (χ^2=31.12; **p<0.001**), indicating a substantial reduction in functionally active areas responsible for maternal–fetal gas exchange.

Fibrinoid necrosis was another prominent manifestation of placental damage. Extensive fibrinoid necrosis involving more than 5% of villi was identified in only **10.0%** of control placentas but increased to **37.0% in mild anemia, 63.0% in moderate anemia, and 70.4% in severe anemia** (χ^2=33.06; **p<0.001**). These findings indicated progressive microcirculatory impairment and ischemic damage to placental tissue.

Similar changes were observed in villous stromal fibrosis. Pronounced stromal fibrosis involving more than 5% of villi was detected in **13.3% of control placentas**, compared with **37.0% in mild anemia, 78.3% in moderate anemia, and 77.8% in severe anemia** (χ^2=46.03; **p<0.001**). Increasing stromal fibrosis was accompanied by a reduction in functionally active terminal villi and progressive disruption of placental architecture.

Overall, the combination of increased syncytial knot formation, reduced vasculo-syncytial membranes, progressive fibrinoid necrosis, and stromal fibrosis demonstrated substantial impairment of villous structure and placental exchange capacity in women with anemia complicated by placental abruption.

Conclusion

Maternal anemia associated with placental abruption is accompanied by significant morphological and morphometric alterations of placental villi. The severity of placental tissue damage generally increases with the progression of anemia and is characterized by increased syncytial knot formation, reduction of vasculo-syncytial membranes, fibrinoid necrosis, and stromal fibrosis.

Quantitative morphometric assessment provides objective criteria for evaluating the severity of placental structural damage and may improve the diagnostic value of conventional histological examination in placental abruption associated with maternal anemia. The most pronounced abnormalities were observed in women with severe anemia, supporting an important role of chronic hypoxia and impaired uteroplacental circulation in the development of placental dysfunction.