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# DECREASED VEGF EXPRESSION AS AN IMMUNOHISTOCHEMICAL MARKER OF IMPAIRED PLACENTAL ANGIOGENESIS IN SEVERE MATERNAL ANEMIA COMPLICATED BY PLACENTAL ABRUPTION

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**Introduction**

Placental abruption is a severe obstetric complication associated with acute disruption of uteroplacental circulation and adverse maternal and perinatal outcomes. Current concepts suggest that placental abruption may represent the final manifestation of a prolonged pathological process involving placental ischemia, endothelial dysfunction, impaired remodeling of spiral arteries, and structural abnormalities of the villous vascular network.

Severe maternal anemia may aggravate these mechanisms by reducing the oxygen-carrying capacity of blood and promoting chronic tissue hypoxia, microcirculatory disturbances, and endothelial injury. Under these conditions, disruption of placental angiogenesis may play an important role in the progression of placental dysfunction.

Vascular endothelial growth factor (VEGF) is involved in the regulation of placental angiogenesis, vascular endothelial function, and development of the placental capillary network. Therefore, assessment of VEGF expression in placental structures may provide additional information about the molecular mechanisms underlying placental vascular damage.

**Objective**

To evaluate immunohistochemical expression of VEGF in the major structural components of placental tissue in women with severe anemia complicated by placental abruption and to determine its relationship with impaired placental angiogenesis.

**Materials and Methods**

A comparative immunohistochemical study was performed on placental tissue obtained after delivery from **52 pregnant women**. The main group included **42 women with clinically confirmed placental abruption associated with severe iron-deficiency anemia**, defined by a hemoglobin concentration below 70 g/L. The control group consisted of **10 women with physiological singleton pregnancies, uncomplicated deliveries, hemoglobin levels ≥110 g/L, and no evidence of placental insufficiency**.

Placental specimens were collected from the central and peripheral regions and from areas adjacent to the site of abruption. Tissue samples were fixed in 10% neutral buffered formalin, paraffin-embedded, and sectioned at a thickness of 4–5 μm.

Immunohistochemical VEGF expression was assessed in three principal placental structures: **syncytiotrophoblast of terminal villi, stromal villous capillaries, and decidual cells of the basal plate**. The intensity of immunohistochemical staining was evaluated semiquantitatively. At least 10 microscopic fields were analyzed at ×400 magnification for each specimen, and mean expression values were calculated.

**Results**

Immunohistochemical analysis demonstrated pronounced differences in VEGF expression between placentas from women with severe anemia and placental abruption and those from women with physiological pregnancies.

In the control group, VEGF showed relatively strong and uniform positive expression in the cytoplasm of syncytiotrophoblasts of terminal and intermediate villi as well as in the endothelium of



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stromal capillaries, indicating preserved angiogenic activity. In contrast, placentas from the main group demonstrated markedly reduced or weak VEGF immunoreactivity.

The mean VEGF expression score in the **syncytiotrophoblast** decreased from **3.82±0.21 in the control group to 1.64±0.12 in the main group ($p<0.01$)**.

The most pronounced reduction was observed in the **stromal capillaries of placental villi**, where VEGF expression decreased from **3.94±0.15 to 1.12±0.08 ($p<0.001$)**.

VEGF expression in **decidual cells** was also substantially reduced, from **3.12±0.18 in physiological pregnancies to 0.95±0.05 in women with severe anemia and placental abruption ($p<0.01$)**.

Thus, the reduction in VEGF expression involved both the trophoblastic and vascular components of the placenta. The particularly marked decrease in villous stromal capillaries indicates pronounced impairment of the angiogenic activity of the placental vascular bed.

These immunohistochemical abnormalities were accompanied by morphological signs of placental damage, including thrombosis of chorionic villous vessels, fibrinoid necrosis of vascular walls, disruption of villous architecture, reduction in functionally active villi, and increased thickness of the syncytiocapillary membrane.

The combination of reduced VEGF expression and structural vascular abnormalities supports an association between severe maternal anemia, impaired placental angiogenesis, chronic hypoxic injury, and placental dysfunction.

**Conclusion**

Placental abruption associated with severe maternal anemia is characterized by a significant reduction in **VEGF expression in the syncytiotrophoblast, villous stromal capillaries, and decidual cells**. The greatest reduction was observed in the vascular component of chorionic villi, suggesting pronounced impairment of placental angiogenesis and microcirculation.

Reduced VEGF expression may therefore be considered a potentially informative **immunohistochemical indicator of structural and functional placental dysfunction** in severe maternal anemia complicated by placental abruption.

The integration of immunohistochemical assessment with conventional morphological examination provides a more comprehensive characterization of placental injury and contributes to a better understanding of the molecular and vascular mechanisms involved in placental abruption.



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