SN:
2181-3906
2026

International scientific journal
«MODERN SCIENCE AND RESEARCH»
VOLUME 5 / ISSUE 6 / UIF:8.2 / MODERNSCIENCE.UZ

THE SIGNIFICANCE OF PLACENTAL MORPHOFUNCTIONAL CHANGES IN OLIGOHYDRAMNIOS

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https://doi.org/10.5281/zenodo.20770599

Annotation. *Oligohydramnios is a significant obstetric complication associated with increased risks of adverse maternal and fetal outcomes. This study examines the significance of placental morphofunctional changes in the development of oligohydramnios. Particular attention is given to structural alterations of the placenta, including villous fibrosis, vascular sclerosis, trophoblastic degeneration, placental infarctions, and impaired microcirculation. These pathological changes contribute to placental insufficiency, resulting in inadequate oxygen and nutrient supply to the fetus. The study also analyzes the relationship between placental dysfunction and reduced amniotic fluid volume, fetal growth restriction, chronic intrauterine hypoxia, and other perinatal complications. Modern diagnostic approaches, including ultrasonography, Doppler velocimetry, and histopathological examination, are discussed as important tools for the early detection of placental abnormalities. Understanding the morphofunctional changes of the placenta in oligohydramnios is essential for improving prenatal diagnosis, preventing adverse pregnancy outcomes, and optimizing maternal and fetal health.*

Keywords: *Oligohydramnios, Placenta, Placental insufficiency, Morphological changes, Functional changes, Fetoplacental insufficiency, Fetal growth restriction, Intrauterine hypoxia, Chorionic villi, Placental circulation.*

Introduction

During pregnancy, the complex interaction between the mother, fetus, and placenta plays a crucial role in ensuring normal fetal growth and development. Amniotic fluid is an essential component of this system, providing a protective and physiological environment for the fetus. It protects the fetus from mechanical injuries, maintains a stable temperature, facilitates fetal movement, and contributes to metabolic exchange. A decrease in amniotic fluid volume, known as oligohydramnios, is a significant obstetric complication associated with adverse perinatal outcomes and impaired fetal development.

The etiology of oligohydramnios is multifactorial and includes maternal diseases, hypertensive disorders of pregnancy, fetal congenital anomalies, placental insufficiency, and impaired fetal renal function. Among these factors, placental dysfunction is considered one of the key mechanisms contributing to the development of oligohydramnios. Structural and functional alterations in the placenta can impair maternal-fetal exchange, resulting in inadequate oxygen and nutrient delivery to the fetus and disturbances in amniotic fluid regulation. Recent studies have demonstrated that oligohydramnios is frequently accompanied by significant morphofunctional changes in placental tissue.

These alterations include villous fibrosis, vascular sclerosis, infarction, intervillous thrombosis, degeneration of trophoblastic cells, and impaired microcirculation. Such pathological changes reduce the efficiency of placental transport functions and contribute to the development of fetoplacental insufficiency. As a consequence, fetal growth restriction, chronic intrauterine hypoxia, and other adverse pregnancy outcomes may occur.

Relevance

Oligohydramnios is one of the most common complications of pregnancy and remains a significant cause of adverse perinatal outcomes worldwide. Reduced amniotic fluid volume is associated with an increased risk of fetal growth restriction, intrauterine hypoxia, preterm birth, fetal distress, and perinatal mortality. Despite advances in obstetric care, the pathogenesis of oligohydramnios is not yet fully understood, and its early diagnosis and prevention continue to be important challenges in modern perinatology.

Aim

The aim of this study is to investigate the significance of placental morphofunctional changes in pregnancies complicated by oligohydramnios and to evaluate their role in the development of fetoplacental insufficiency and adverse fetal outcomes.

Main part

The placenta is a vital organ that ensures the exchange of oxygen, nutrients, and metabolic products between the mother and fetus. In pregnancies complicated by oligohydramnios, significant structural alterations are frequently observed in placental tissue. Histopathological studies have demonstrated the presence of villous fibrosis, syncytial knot formation, trophoblastic degeneration, and placental infarctions. These changes indicate chronic placental insufficiency and impaired maternal-fetal circulation. Fibrosis of chorionic villi reduces the functional surface area available for nutrient and gas exchange. Thickening of placental vessel walls may further compromise blood flow within the fetoplacental circulation. Placental infarctions decrease the amount of functional placental tissue and contribute to fetal hypoxia. Degenerative changes in trophoblastic cells impair the endocrine and transport functions of the placenta. Increased deposition of fibrinoid material is also commonly observed in placentas associated with oligohydramnios. These structural abnormalities disrupt normal placental architecture and reduce its physiological efficiency. As a result, the fetus receives insufficient oxygen and nutrients necessary for normal development. The severity of placental morphological changes often correlates with the degree of amniotic fluid reduction.

In addition to structural abnormalities, significant functional disturbances occur in the placenta during pregnancies complicated by oligohydramnios. The placenta serves as the primary organ responsible for maintaining fetal homeostasis through transport, metabolic, endocrine, and immunological functions. Placental insufficiency results in reduced efficiency of these physiological processes. Impaired uteroplacental blood flow decreases oxygen delivery to fetal tissues and contributes to chronic fetal hypoxia. Reduced transfer of glucose, amino acids, and essential nutrients negatively affects fetal growth and development.

Endocrine dysfunction of the placenta may alter the production of hormones necessary for maintaining normal pregnancy progression.

Disturbances in placental transport mechanisms can influence fetal urine production, which is one of the major sources of amniotic fluid during late pregnancy. Consequently, placental dysfunction may directly contribute to the development of oligohydramnios. Alterations in angiogenic factors and endothelial function further impair placental circulation. Chronic hypoperfusion may lead to progressive deterioration of placental performance. These functional abnormalities increase the risk of fetal growth restriction and perinatal complications.

Placental insufficiency is considered one of the most important causes of fetal growth restriction in pregnancies complicated by oligohydramnios. Adequate placental function is essential for providing the fetus with sufficient oxygen and nutrients throughout gestation. When placental circulation becomes compromised, fetal growth may be significantly affected. Reduced blood flow within the uteroplacental and fetoplacental systems limits nutrient availability and impairs cellular metabolism. Chronic hypoxia stimulates adaptive mechanisms that prioritize blood flow to vital organs such as the brain and heart. Although these compensatory responses improve short-term survival, they may negatively affect overall fetal growth. Placental abnormalities associated with oligohydramnios often result in lower birth weight and delayed fetal development. Doppler ultrasonography frequently demonstrates abnormal blood flow patterns in affected pregnancies. These hemodynamic changes reflect increased vascular resistance within the placental circulation. Severe placental insufficiency may lead to intrauterine growth restriction, fetal distress, and adverse neonatal outcomes. Numerous studies have demonstrated a strong association between reduced amniotic fluid volume and impaired fetal growth parameters. Early detection of placental insufficiency may improve pregnancy management and reduce perinatal morbidity.

Placental vascular pathology is one of the most significant factors contributing to the development of oligohydramnios. Normal placental circulation depends on adequate development and function of maternal and fetal blood vessels. In pregnancies affected by oligohydramnios, vascular abnormalities frequently occur within the placental tissue. These changes include narrowing of vascular lumens, endothelial damage, thrombosis, and increased vascular resistance.

Reduced blood flow through placental vessels compromises oxygen and nutrient transport to the fetus. Endothelial dysfunction may further impair vasodilation and promote ischemic injury within placental tissue. Chronic vascular insufficiency often leads to hypoxic damage and progressive placental degeneration. Histological examination commonly reveals vascular sclerosis and obliterative changes in small placental arteries. Such alterations significantly reduce placental perfusion and contribute to fetal compromise. Impaired vascular adaptation during pregnancy may also increase the risk of hypertensive disorders and preeclampsia. These vascular changes are closely associated with the severity of oligohydramnios and adverse perinatal outcomes.

The clinical significance of placental morphofunctional changes in oligohydramnios extends beyond the reduction of amniotic fluid volume. Placental pathology serves as an important indicator of fetal well-being and pregnancy outcome.

Morphological and functional abnormalities may predict the development of serious obstetric complications, including fetal growth restriction, preterm birth, fetal distress, and intrauterine fetal death.

Early identification of placental dysfunction allows healthcare professionals to implement timely interventions aimed at reducing perinatal risks. Ultrasound examination and Doppler studies provide valuable information regarding placental structure and circulation. Laboratory biomarkers may also assist in assessing placental function and fetal condition. Recognition of placental abnormalities is essential for determining the optimal timing and mode of delivery.

Pregnancies complicated by severe placental insufficiency often require close monitoring and specialized obstetric care. Early diagnosis can significantly improve neonatal outcomes and reduce maternal-fetal morbidity.

Early diagnosis and prevention of placental dysfunction remain important priorities in contemporary obstetrics. Advances in prenatal imaging techniques have improved the ability to detect placental abnormalities before the onset of severe clinical manifestations. High-resolution ultrasonography, Doppler velocimetry, and fetal biophysical assessment are widely used for monitoring placental health. Novel biomarkers associated with placental angiogenesis and endothelial function may provide additional diagnostic information. Early identification of women at high risk for placental insufficiency allows implementation of individualized management strategies. Adequate prenatal care, control of maternal diseases, and optimization of maternal nutrition may help reduce the incidence of placental dysfunction. Prevention of smoking, alcohol consumption, and other harmful exposures is also important for maintaining placental health.

Timely recognition of placental abnormalities enables clinicians to prevent severe fetal complications associated with oligohydramnios. Future research focusing on molecular mechanisms of placental pathology may contribute to the development of new therapeutic approaches. Therefore, improving strategies for early diagnosis and prevention of placental dysfunction is essential for enhancing maternal and neonatal outcomes.

Conclusion

In conclusion, oligohydramnios is a significant obstetric condition that is closely associated with placental morphofunctional abnormalities. The findings indicate that structural changes in the placenta, including villous fibrosis, placental infarctions, vascular sclerosis, trophoblastic degeneration, and impaired microcirculation, play a crucial role in the development of fetoplacental insufficiency. These pathological alterations disrupt the normal exchange of oxygen, nutrients, and metabolic products between the mother and fetus, thereby compromising fetal well-being. Placental dysfunction contributes to chronic fetal hypoxia, intrauterine growth restriction, and adverse perinatal outcomes. Furthermore, impaired placental circulation may negatively affect fetal renal function, resulting in decreased fetal urine production and subsequent reduction in amniotic fluid volume. Therefore, placental pathology represents one of the major pathogenetic mechanisms underlying oligohydramnios. The study highlights the importance of evaluating placental morphology and function in pregnancies complicated by oligohydramnios.

Modern diagnostic methods, including ultrasonography, Doppler velocimetry, and histopathological examination, provide valuable information for the early detection of placental abnormalities. Early identification of placental dysfunction may improve risk assessment, facilitate timely clinical intervention, and reduce the incidence of maternal and fetal complications.

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