



MATERNAL COMORBIDITIES AND CHRONIC PLACENTAL STRUCTURAL DAMAGE IN HIGH-RISK PREGNANCIES

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ABSTRACT

High-risk pregnancies frequently develop on the background of long-standing maternal disorders. Gynecological and endocrine diseases may interfere with placental development from early gestation, leading to chronic structural damage that compromises placental efficiency. These alterations may persist throughout pregnancy and ultimately affect fetal growth and survival. Understanding the relationship between maternal comorbidities and placental structural changes is essential for improving risk stratification and preventive strategies in obstetric care.

Introduction

High-risk pregnancies frequently develop on the background of long-standing maternal disorders. Gynecological and endocrine diseases may interfere with placental development from early gestation, leading to chronic structural damage that compromises placental efficiency. These alterations may persist throughout pregnancy and ultimately affect fetal growth and survival. Understanding the relationship between maternal comorbidities and placental structural changes is essential for improving risk stratification and preventive strategies in obstetric care.

Materials and Methods

Placental tissues obtained after delivery were analyzed from a total of 250 women of reproductive age. The study group included women with gynecological and/or endocrine disorders and a complicated obstetric history, while placentas from low-risk pregnancies served as controls. All samples underwent routine histological processing. Structural alterations, including villous degeneration, stromal remodeling, and vascular insufficiency, were evaluated using standard morphological criteria. Maternal clinical data were analyzed in relation to placental findings. Statistical analysis was performed, and differences were considered significant at $p < 0.05$.

Results

A total of 250 placental samples were examined, including 150 samples from high-risk pregnancies and 100 samples from low-risk controls. Gynecological disorders were identified in 98 of 150 high-risk cases (65.3%), while endocrine abnormalities were present in 82 cases (54.7%). Combined gynecological and endocrine comorbidities were observed in 61 women

(40.7%). In the control group, comparable conditions were detected in only 9% of cases, and the difference between groups was statistically significant ($p < 0.001$).

Histological examination revealed placental structural damage in 112 high-risk cases (74.7%), compared to 14 cases (14.0%) in the control group ($p < 0.001$). Villous degeneration was observed in 79 high-risk placentas (52.7%) versus 8 controls (8.0%). Stromal fibrosis and remodeling were identified in 83 cases (55.3%) in the high-risk group and in 5 cases (5.0%) among controls. Signs of vascular insufficiency, including reduced villous vascularization, were present in 68 high-risk cases (45.3%) compared with 6 control cases (6.0%).

The frequency of placental structural damage increased proportionally with the number of maternal comorbidities. Women with a single comorbid condition demonstrated placental abnormalities in 49.2% of cases, whereas those with two or more comorbidities showed structural damage in 82.1% of cases. This association was statistically significant ($\chi^2 = 18.6$, $p < 0.001$). Notably, markers of acute inflammatory injury were minimal and did not differ significantly between groups ($p > 0.05$), indicating a predominantly chronic pathological process.

Discussion

The results demonstrate that placental pathology in high-risk pregnancies is strongly associated with maternal gynecological and endocrine comorbidities. Rather than acute inflammatory damage, the observed changes reflect a chronic process characterized by progressive structural remodeling of placental tissue. Villous degeneration, stromal fibrosis, and vascular insufficiency appear to represent interconnected mechanisms that compromise placental function over time. These findings support the concept that chronic placental insufficiency serves as a final common pathway linking maternal health disturbances to adverse pregnancy outcomes.

Conclusion

Maternal gynecological and endocrine comorbidities play a decisive role in the development of chronic placental structural damage. The cumulative effect of multiple risk factors significantly increases the likelihood of placental insufficiency. Incorporating placental morphological assessment into prenatal risk evaluation may improve early identification of vulnerable pregnancies and contribute to the prevention of adverse perinatal outcomes.

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