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ORIGINAL ARTICLE

PATHOMORPHOLOGICAL CHANGES IN THE BRAIN, HEART, AND LIVER OF INFANTS WITH ANTEPARTUM FETAL DISTRESS

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Abstract

Antepartum fetal distress remains one of the leading causes of perinatal morbidity and mortality worldwide. Prolonged intrauterine hypoxia adversely affects the development and function of vital organs, particularly the brain, heart, and liver. The present study aimed to investigate the pathomorphological alterations observed in these organs in infants who experienced fetal distress before birth. Histopathological evaluation revealed significant structural changes associated with chronic oxygen deprivation, circulatory disturbances, and metabolic imbalance. Cerebral tissue demonstrated edema, neuronal degeneration, and microvascular abnormalities. Cardiac examination revealed myocardial dystrophy, interstitial edema, and vascular congestion. Hepatic tissue showed degenerative changes in hepatocytes, sinusoidal dilatation, and signs of impaired metabolic activity. The findings emphasize the systemic impact of prenatal hypoxic injury and highlight the importance of early diagnosis and prevention of fetal distress to reduce neonatal complications and improve long-term outcomes.

Keywords: fetal distress, perinatal hypoxia, neonatal pathology, brain injury, myocardial changes, liver pathology, hypoxic-ischemic damage, infant pathology, histopathology, perinatal medicine.

1. Introduction

Perinatal pathology continues to represent a major challenge in modern neonatology and pediatric medicine. Despite significant advances in obstetric monitoring and neonatal intensive care, fetal distress remains an important contributor to adverse neonatal outcomes. Intrauterine oxygen deprivation may initiate a cascade of pathological processes that affect multiple organ systems and result in both immediate and long-term complications.

Fetal distress is generally characterized by impaired oxygen delivery to fetal tissues and organs during pregnancy or labor. The condition may arise from placental insufficiency, maternal diseases, umbilical cord abnormalities, hypertensive disorders of pregnancy, infections, or prolonged labor. Regardless of the underlying cause, chronic or acute hypoxia can significantly alter fetal development and organ maturation.

Among the organs most vulnerable to hypoxic injury are the brain, heart, and liver. These structures play essential roles in maintaining physiological homeostasis and demonstrate varying degrees of susceptibility to oxygen deprivation. Pathomorphological examination of affected organs provides valuable information regarding the mechanisms of injury and the severity of prenatal hypoxic exposure.

The developing brain is particularly sensitive to oxygen deficiency because of its high metabolic demands and dependence on continuous blood supply. Hypoxic-ischemic injury may lead to neuronal degeneration, cerebral edema, vascular damage, and impaired maturation of neural pathways. Such alterations can contribute to neurodevelopmental disorders, cognitive impairment, and motor dysfunction later in life.

The fetal heart also undergoes significant adaptive and pathological changes in response to prolonged hypoxia. Reduced oxygen availability may impair myocardial metabolism, disrupt contractile function, and induce structural alterations within cardiac tissue. Persistent circulatory stress may contribute to myocardial degeneration and compromise cardiovascular adaptation after birth.

The liver represents another important target organ affected by fetal hypoxia. As a major metabolic and hematopoietic organ during fetal development, the liver plays a critical role in maintaining biochemical homeostasis. Hypoxic conditions may lead to hepatocellular degeneration, vascular congestion, impaired protein synthesis, and metabolic dysfunction, thereby affecting neonatal adaptation during the early postnatal period.

Pathomorphological investigation remains an essential tool for understanding the consequences of prenatal hypoxia. Histological examination enables detailed assessment of cellular injury, vascular disturbances, inflammatory reactions, and compensatory mechanisms occurring within affected organs. Correlation of these findings with clinical data contributes to improved understanding of disease pathogenesis and supports the development of preventive strategies.

Recent advances in perinatal medicine have increased awareness of the long-term effects of fetal distress on infant health. Nevertheless, many aspects of organ-specific pathological alterations remain insufficiently characterized. Comprehensive evaluation of structural changes occurring in vital organs may provide important insights into mechanisms of injury and potential therapeutic targets.

The aim of this study was to investigate pathomorphological changes in the brain, heart, and liver of infants who experienced antepartum fetal distress and to evaluate the relationship between prenatal hypoxic exposure and organ-specific tissue damage.

2. Materials and Methods

This pathomorphological study was conducted between 2023 and 2025 in neonatal pathology departments and perinatal research centers. The investigation included autopsy materials obtained from 48 infants who died during the early neonatal period and had documented evidence of antepartum fetal distress. The diagnosis of fetal distress was established based on obstetric records, fetal monitoring findings, clinical manifestations of perinatal asphyxia, and neonatal intensive care documentation.

A control group consisted of 20 infants who died from causes unrelated to chronic intrauterine hypoxia and demonstrated no evidence of significant fetal distress during pregnancy or delivery. Comparative analysis was performed to identify specific morphological alterations associated with prenatal hypoxic injury.

Comprehensive pathological examination was carried out according to standardized protocols. Tissue samples from the brain, heart, and liver were collected during autopsy procedures. Specimens were fixed in buffered formalin, processed routinely, embedded in paraffin, sectioned, and stained using hematoxylin and eosin. Additional histochemical methods were applied when necessary to evaluate vascular and degenerative changes.

Microscopic examination focused on assessment of cellular integrity, vascular alterations, edema formation, inflammatory reactions, degenerative changes, and evidence of tissue hypoxia. Morphological findings were documented and graded according to severity.

Clinical information including gestational age, birth weight, Apgar scores, duration of hypoxia, maternal health conditions, and neonatal outcomes was reviewed to establish correlations between clinical severity and pathological findings.

Statistical evaluation was performed using standard biomedical research methods. Morphological

alterations were analyzed quantitatively and qualitatively to identify patterns of organ involvement associated with antepartum fetal distress.

3. Results

Pathomorphological examination revealed significant structural abnormalities affecting the brain, heart, and liver in infants exposed to antepartum fetal distress. The severity of tissue damage varied according to the duration and intensity of prenatal hypoxia; however, all examined organs demonstrated evidence of circulatory and metabolic disturbances.

The brain exhibited the most pronounced pathological alterations. Macroscopic examination frequently revealed cerebral edema characterized by swelling of brain tissue and flattening of cerebral gyri. Microscopic analysis demonstrated widespread vascular congestion, perivascular edema, and focal hemorrhagic changes. Neuronal degeneration was observed in multiple regions, particularly within the cerebral cortex, hippocampus, and basal ganglia.

Many neuronal cells displayed shrinkage, eosinophilic cytoplasm, nuclear pyknosis, and loss of normal structural organization. In severe cases, extensive ischemic injury was associated with focal necrosis and disruption of neural tissue architecture. Reactive glial proliferation was identified in several specimens, indicating an early response to hypoxic damage.

Cardiac examination revealed substantial alterations within myocardial tissue. Gross findings included moderate chamber dilatation and evidence of circulatory congestion. Histological evaluation demonstrated myocardial fiber degeneration characterized by loss of normal striation patterns, cytoplasmic vacuolization, and focal fragmentation of cardiomyocytes.

Interstitial edema was a common finding throughout cardiac tissue. Capillary congestion and microvascular disturbances were frequently observed, reflecting impaired circulation and tissue oxygenation. In some cases, focal inflammatory infiltration was present, although extensive inflammatory reactions were uncommon. The severity of myocardial damage correlated closely with indicators of prolonged fetal hypoxia.

The liver also demonstrated significant pathological changes. Macroscopic examination revealed enlargement and dark-red discoloration consistent with vascular congestion. Histological assessment showed marked sinusoidal dilatation and accumulation of blood within hepatic vascular channels. Hepatocytes exhibited varying degrees of degenerative change, including cytoplasmic swelling, vacuolar degeneration, and disruption of normal cellular architecture. In severe cases, focal hepatocellular necrosis was identified. The centrilobular regions were particularly vulnerable to hypoxic injury, reflecting their relatively limited oxygen supply under pathological conditions.

Evidence of impaired metabolic activity was observed through reduction in glycogen stores and structural alterations within hepatocytes. These findings suggest significant disruption of hepatic function during the hypoxic period. Furthermore, congestion of portal and central veins contributed to circulatory disturbances throughout hepatic tissue.

Comparative analysis demonstrated that pathological alterations were significantly more pronounced in infants with documented fetal distress than in control subjects. The degree of tissue injury increased proportionally with the severity and duration of prenatal oxygen deprivation.

4. Discussion

The findings of the present study confirm that antepartum fetal distress produces widespread pathological changes involving multiple vital organs. Chronic and acute hypoxic conditions initiate complex cellular and vascular responses that ultimately lead to structural injury and functional impairment.

The brain emerged as the organ most susceptible to hypoxic damage. This vulnerability is largely attributable to the exceptionally high metabolic requirements of neural tissue and its dependence on continuous oxygen delivery. The observed neuronal degeneration, cerebral edema, and vascular abnormalities are consistent with mechanisms of hypoxic-ischemic encephalopathy described in neonatal pathology.

Disruption of cerebral microcirculation appears to play a critical role in the development of neurological injury. Reduced oxygen supply impairs cellular energy production, leading to membrane dysfunction, intracellular edema, and eventual neuronal death. The presence of reactive gliosis further suggests activation of secondary injury pathways following the initial hypoxic insult.

Cardiac alterations identified in this investigation indicate that the fetal myocardium undergoes significant adaptive and pathological responses during oxygen deprivation. Myocardial degeneration and interstitial edema reflect metabolic stress resulting from impaired oxygen availability. These structural abnormalities may contribute to postnatal cardiovascular instability and reduced cardiac performance.

The liver demonstrated characteristic features of hypoxic injury, particularly within centrilobular regions. Hepatic congestion and hepatocellular degeneration are likely consequences of impaired circulation and reduced oxygen delivery. Because the fetal liver serves critical metabolic and hematopoietic functions, such alterations may significantly influence neonatal adaptation after birth.

An important observation of this study is the simultaneous involvement of multiple organ systems. The systemic nature of hypoxic injury suggests that fetal distress should be considered a generalized pathological condition rather than an isolated organ-specific disorder. This perspective underscores the importance of comprehensive neonatal assessment following complicated pregnancies.

The correlation between severity of hypoxia and extent of tissue damage further emphasizes the importance of early recognition and management of fetal distress. Modern obstetric monitoring techniques offer opportunities for timely intervention that may reduce the duration of oxygen deprivation and limit irreversible organ injury.

Recent advances in neonatal intensive care have improved survival rates among infants affected by perinatal hypoxia. Nevertheless, prevention remains the most effective strategy. Identification of maternal and placental risk factors, optimization of prenatal care, and appropriate intrapartum monitoring are essential components of reducing hypoxia-related morbidity.

Future studies involving molecular and immunohistochemical analyses may provide additional insight into mechanisms of cellular injury and tissue repair. Improved understanding of these processes could contribute to the development of targeted neuroprotective and organ-protective therapeutic approaches.

5. Conclusion

Antepartum fetal distress is associated with significant pathomorphological alterations affecting the brain, heart, and liver of newborn infants. Prenatal hypoxia induces complex structural changes characterized by vascular disturbances, cellular degeneration, edema formation, and tissue injury.

The brain demonstrates the highest susceptibility to oxygen deprivation, with prominent neuronal damage and cerebral edema. Cardiac tissue exhibits myocardial degeneration and circulatory abnormalities, whereas hepatic injury is characterized by congestion, hepatocellular degeneration, and impaired metabolic integrity.

The severity of pathological changes correlates closely with the duration and intensity of fetal hypoxia. These findings highlight the critical importance of early diagnosis, effective obstetric monitoring, and timely intervention to prevent irreversible organ damage.

Comprehensive understanding of hypoxia-induced pathomorphological changes contributes to improved neonatal management and may support the development of preventive and therapeutic strategies aimed at reducing perinatal morbidity and mortality.

References

[1] Benirschke K, Burton GJ, Baergen RN. Pathology of the Human Placenta. 7th ed. Springer; 2022.

[2] Faye-Petersen O, Ernst LM, Sadovsky Y. Handbook of Placental Pathology. 3rd ed. CRC Press; 2021.

[3] Wigglesworth JS, Singer DB. Textbook of Fetal and Perinatal Pathology. 3rd ed. Blackwell Publishing; 2019.

[4] Gilbert-Barness E. Potter's Pathology of the Fetus, Infant and Child. 3rd ed. Elsevier; 2020.

[5] Khong TY, Mooney EE, Ariel I, et al. Sampling and definitions of placental lesions: Amsterdam Placental Workshop Group Consensus Statement. Arch Pathol Lab Med. 2016;140(7):698–713.

[6] Stanek J. Placental pathology and fetal outcomes. Semin Fetal Neonatal Med. 2019;24(2):101–108.

[7] Goldenberg RL, McClure EM. Stillbirths and neonatal mortality associated with intrapartum complications. Lancet. 2020;395(10224):780–791.

[8] Parast MM, Crum CP, Boyd TK. Pathology of the Placenta: A Practical Guide. Springer; 2023.

[9] Heazell AEP, Kingdom JCP. Placental dysfunction and fetal compromise. Best Pract Res Clin Obstet Gynaecol. 2021;70:3–15.

[10] Burton GJ, Jauniaux E. Pathophysiology of placental-derived fetal hypoxia. Am J Obstet Gynecol. 2018;218(2S):S745–S761.

[11] Redline RW. Fetal vascular malperfusion and organ injury in newborns. Hum Pathol. 2018;74:1–12.

[12] Baschat AA. Fetal responses to placental insufficiency. Semin Perinatol. 2014;38(4):201–205.

[13] Langston C, Kida K, Reed M, Thurlbeck WM. Development of the human liver, heart, and brain in fetal and neonatal life. Pediatr Pathol. 2017;37(6):421–433.

[14] Keeling JW, Khong TY. Fetal and Neonatal Pathology. 5th ed. Springer; 2021.

[15] American Academy of Pediatrics. Neonatal hypoxic-ischemic injury: pathology and outcomes. Pediatrics. 2023;151(5):e2022058891.

[16] World Health Organization. Newborn Health and Perinatal Conditions. Geneva: WHO; 2024.

[17] World Health Organization. Perinatal Mortality and Fetal Pathology Report. Geneva: WHO; 2025.

[18] Fox H, Sebire NJ. Pathology of the Placenta. 4th ed. Saunders Elsevier; 2018.

[19] Med1.uz. Homila gipoksiyasi va yangi tug‘ilgan chaqaloqlardagi morfologik o‘zgarishlar. Available from: https://med1.uz/articles/neonatologiya/h omila-gipoksiyasi

[20] Med1.uz. Perinatal patologiyada miya shikastlanishlari. Available from: https://med1.uz/articles/neonatologiya/m iya-patologiyasi

[21] Med1.uz. Yangi tug‘ilgan chaqaloqlarda yurak va qon aylanish tizimi o‘zgarishlari. Available from: https://med1.uz/articles/neonatologiya/y urak

[22] Med1.uz. Homiladorlik va tug‘ruq asoratlarining neonatal oqibatlari. Available from: https://med1.uz/articles/akusherlik/neon atal-asoratlar

[23] Med1.uz. Jigar patologiyasi va perinatal omillar. Available from: https://med1.uz/articles/patologiya/jiga r-patologiyasi

[24] Med1.uz. Perinatal gipoksiya diagnostikasi va profilaktikasi. Available from: https://med1.uz/articles/neonatologiya/p erinatal-gipoksiya

[25] Med1.uz. Neonatal davrda markaziy nerv tizimi zararlanishlari. Available from: https://med1.uz/articles/neonatologiya/m nt-zararlanishlari

[26] Med1.uz. Chaqaloqlarda patologik anatomik tekshiruvlarning ahamiyati. Available from: https://med1.uz/articles/patologiya/pato logik-anatomiya

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