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Histopathological Changes in Placenta during Fetal Asphyxia

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ABSTRACT

Background: *Neonatal asphyxia is state of respiratory insufficiency in the newborn immediately after birth, clinically characterized by prolonged fetal asphyxia, with prolonged fetal asphyxia, with prolonged respiratory failure or prolonged respiratory arrhythmia, and is classified as cerebral asphyxia, pulmonary asphyxia, and cardiac asphyxia. Neonatal asphyxia is disruption of metabolic processes caused by hypoxic conditions of the fetal-placental system.* ***The Aim:*** *We aimed to investigate the histopathological changes of the placenta during fetal asphyxia.* ***Methods:*** *The study population included 237 placentas from single pregnant women who had been delivered for the past five years. Among them, 110 gravidas who underwent emergency caesarean section due to fetal asphyxia were included as the study group and 127 gravidas who delivered normal without fetal asphyxia were included as the control group. After tissue sections from the placenta were obtained, H-E stained specimens were prepared according to the conventional tissue sampling technique, and the appearance was calculated by observing inflammatory changes as indicated by congestion, edema, hemorrhage, and infiltration of inflammatory cells and observing degenerative changes with the index of degeneration, atrophy, necrosis, and defect under ×400 light microscope view.* ***Results and Conclusions:*** *The histopathological changes of the placenta during fetal asphyxia are as follows. The percentage of regressive changes in the placenta during fetal asphyxia was degeneration 89.1%, atrophy 70.0%, necrosis 53.6% and defect 37.3%, and the percentage of inflammatory changes was congestion 53.6%, edema 30.0%, hemorrhage 59.1% and inflammatory cell infiltration 47.3%. The more severe the severity of fetal asphyxia, the more severe the histopathological changes of the placenta.*

Keywords: Fetal asphyxia, Neonatal asphyxia, Histopathological change.

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INTRODUCTION

Neonatal asphyxia is state of respiratory insufficiency in the newborn immediately after birth, clinically characterized by prolonged fetal asphyxia, with prolonged fetal asphyxia, with prolonged respiratory failure or prolonged respiratory arrhythmia and is classified as cerebral asphyxia, pulmonary asphyxia, and cardiac asphyxia[4,8]. Neonatal asphyxia leads to acidosis and changes in circulatory dynamics, initially with decreased blood partial pressure, elevated partial pressure of carbon dioxide and decreased pH and respiratory acidosis. The prolonged asphyxia does not stop at the level of respiratory asphyxia, but causes pathological changes in the brain, kidney, lung and other solid organs due to hypoxia, which results in neurological symptoms including spasm[3,5]. Neonatal asphyxia is state of hypoxia, hypercapnia, and metabolic acidosis in the blood and tissue which leads to organ dysfunction which, if not resuscitated rapidly, leads to irreversible organ damage or necrosis.

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Neonatal asphyxia is divided into grade 1 (mild) and grade 2 (severe) asphyxia according to symptoms and is defined as Apgar score, which is measure of neonatal life expectancy, neurological prognosis and survival benefit[1,6,7]. If fetal asphyxia is inadequate adaptation to fetal hypoxia, neonatal asphyxia is also syndrome of various pathologies due to hypoxia at birth. The fetus and newborn are the only differences in the inner and outer parts of the uterus and as such, it is possible to treat fetal asphyxia and neonatal asphyxia as continuous pathology when examining its pathology or impact. It is better to use the term perinatal asphyxia because asphyxia can occur in utero at birth or after birth.

WHO defined perinatal asphyxia as 'not starting or maintaining breathing at birth'. Perinatal asphyxia is caused by impaired gas exchange and this condition continues to gradually develop hypoxia and hypercapnia resulting in metabolic acidosis. Neonatal asphyxia is disruption of metabolic processes caused by hypoxic conditions of the fetal-placental system. Hypoxic ischemic encephalopathy triggered by neonatal asphyxia is metabolic disruption caused by hypoxic conditions of the fetal-placental system and multiple organs are damaged[2]. We aimed to investigate the histopathological changes of the placenta during fetal asphyxia.

SUBJECTS AND METHODS

The study population included 237 placentas from single pregnant women who had been delivered at the Pyongyang Maternity Hospital for the past five years. Among them, 110 gravidas who underwent emergency caesarean section due to fetal asphyxia were included as the study group and 127 gravidas who delivered normal without fetal asphyxia were included as the control group. Subjects with complications such as gestational hypertension syndrome, placental abruption and prophase rupture were excluded. After tissue sections from the placenta were obtained, H-E stained specimens were prepared according to the conventional tissue sampling technique and the appearance was calculated by observing inflammatory changes as indicated by congestion, edema, hemorrhage and infiltration of inflammatory cells and observing regressive changes with the index of degeneration, atrophy, necrosis and defect under ×400 light microscope view. Significance tests were performed by Student's t-test for the mean of the study results and by Chi-square test for the percentage of the study results. Histopathological evaluation of the placenta was performed according to clinicopathological diagnosis and treatment guidelines published by the Ministry of Public Health, DPRK.

RESULTS

Table 1. Percentage of regressive changes in placental tissue

Regressive change parameter	Group	Cases	-	+
Degeneration	Study	110	12(10.9)	98(89.1)*
	Control	127	95(74.8)	32(25.2)
Atrophy	Study	110	33(30.0)	77(70.0)*
	Control	127	114(89.8)	13(10.2)
Necrosis	Study	110	51(46.4)	59(53.6)*
	Control	127	117(92.1)	10(7.9)
defect	Study	110	69(62.7)	41(37.3)*
	control	127	120(94.5)	7(5.5)

*; $p<0.05$ (compared with control group), ():%

As shown in Table 1, the percentage of regressive changes was significantly higher in the group with degenerative, atrophic, necrotic and defect than 25.2%, 10.2%, 7.9% and 5.5% in the control group, 89.1%, 70.0%, 53.6% and 37.3%, significantly.

Table 2. Percentage of inflammatory changes in placental tissue

Inflammatory change parameter	Group	Cases	-	+
Congestion	Study	110	51(46.4)	59(53.6)*
	Control	127	107(84.3)	20(15.7)
Edema	Study	110	77(70.0)	33(30.0)*
	Control	127	121(95.3)	6(4.7)
Hemorrhage	Study	110	45(40.9)	65(59.1)*
	Control	127	108(85.0)	19(15.0)
Infiltration of inflammatory cell	Study	110	58(52.7)	52(47.3)*
	Control	127	115(90.6)	12(9.4)

*; p<0.05 (compared with control group), ():%

As shown in Table 2, the percentage of inflammatory changes was significantly higher in the study group with congestion, edema, hemorrhage, and infiltration of inflammatory cells than in the control group of 53.6%, 30.0%, 59.1% and 47.3%, significantly, than 15.7%, 4.7%, 15.0% and 9.4% of the control group.

Table 3. Percentage of regressive changes in placental tissue according to the severity of asphyxia

Group	Cases	Regressive change parameter			
		Degeneration	Atrophy	Necrosis	Defect
Mild birth asphyxia	77	65 (84.4)	48 (62.3)	35 (45.5)	20 (26.0)
Severe birth asphyxia	33	33* (100.0)	29* (87.9)	24* (72.7)	21* (63.6)

*: p<0.05 (compared to the mild birth asphyxia group), ():%

As shown in Table 3, the percentage of regressive changes was significantly higher than 84.4%, 62.3%, 45.5% and 26.0% in mild asphyxia, with degeneration, atrophy, necrosis, and defect of 100.0%, 87.9%, 72.7% and 63.6%, significantly, in severe asphyxia.

Table 4. Percentage of inflammatory changes in placental tissue according to the severity of asphyxia

Group	Cases	Inflammatory change parameter			
		Congestion	Edema	Hemorrhage	Infiltration of inflammatory cell
Mild birth asphyxia	77	35 (45.5)	18 (23.4)	37 (48.1)	31 (40.3)
Severe birth asphyxia	33	24* (72.7)	15* (45.5)	28* (84.8)	21* (63.6)

*: p<0.05 (compared with mild birth asphyxia group), ():%

As shown in Table 4, the percentage of inflammatory changes was significantly higher than 45.5%, 23.4%, 48.1% and 40.3% of mild asphyxia, with congestion, edema, hemorrhage, and infiltration of inflammatory cells in severe asphyxia being 72.7%, 45.5%, 84.8%, and 63.6%, significantly.

DISCUSSION

Neonatal asphyxia is syndrome that is the main symptom of respiratory circulatory failure at birth with occult fetal asphyxia occurring during pregnancy and fetal asphyxia occurring during labor with a frequency of approximately 10% of the total birth and 0.5%. In obstetric practice, asphyxia in neonates accounting for 10% of the birth rate causes irreversible organ damage and necrosis due to hypoxia of blood and tissue.

These functional changes in fetal asphyxia result in ischemic changes in the placenta which pathologically manifest as regressive changes such as degeneration, atrophy, necrosis and defect of the placental villi and series of histopathological changes including inflammatory changes such as congestion, edema, hemorrhage, and infiltration of inflammatory cells in the placental tissue.

We investigated histopathological changes in the placenta in 110 patients with emergency caesarean section diagnosed with fetal asphyxia to determine the histopathological changes in the placenta, with regressive changes and inflammatory changes as the main parameters. The percentage of regressive changes was significantly higher in the group with degeneration, atrophy, necrosis and deletion than 25.2%, 10.2%, 7.9% and 5.5% in the control group, significantly (Table 1).

The authors attributed the development and maturation of the placenta to various regressive changes (infarction, calcification, fibrinoid deposition, stenosis or closure of the intervillous space) that are the so-called senescence of the placenta which leads to extensive fibrosis and calcification of the placental villi leading to marked placental hypofunction with rapid progression of placental senescence compared to early pregnancy. In our study, the percentage of regressive changes in the placenta of asphyxia neonates was significantly higher compared with that of non-asphyxia neonates which is consistent with the literature. The regressive changes in the placental villi such as degeneration, atrophy, necrosis and defect are thought to interfere with the exchange of material in the villi leading to fetal asphyxia.

The percentage of inflammatory changes was significantly higher in the groups with congestion, edema, hemorrhage and infiltration of inflammatory cells than in the control group of 53.6%, 30.0%, 59.1% and 47.3%, significantly, than in the control group of 15.7%, 4.7%, 15.0%, and 9.4% (Table 2). Ischemia and ischemia-reperfusion have been shown to promote the migration of leukocytes to endothelial cells by increased blood vessel permeability and to induce their activation and to increase adhesion to endothelial cells through adhesion molecules.

Histologically, infarction is typical of necrosis with collapse and disappearance of nuclei which become eosinophilic over time, hyperemia and hemorrhage in perinecrotic tissues and macrophage or leukocyte infiltration. As red infarction is also known as hemorrhagic infarction and as well as the evidence that the organ with congestion, the vasculature and the vasculature developed, our results also suggest that the presence of profound inflammatory changes in the placental tissue during fetal asphyxia is likely to be an inflammatory event in organs with abundant blood vessels and high oxygen demand. Next, we examined histopathological changes in the placenta according to the severity of asphyxia in 77 patients with mild asphyxia and 33 patients with severe asphyxia with regressive changes and inflammatory changes as the main parameters.

The percentage of regressive changes was significantly higher than 84.4%, 62.3%, 45.5% and 26.0% in mild asphyxia with 100.0%, 87.9%, 72.7% and 63.6% degenerative,

atrophic, necrotic and defect in severe asphyxia, significantly (Table 3). The percentage of inflammatory changes in placental tissue was significantly higher than 45.5%, 23.4%, 48.1% and 40.3% of mild asphyxia, with congestion, edema, hemorrhage and infiltration of inflammatory cells in severe asphyxia being 72.7%, 45.5%, 84.8% and 63.6%, significantly (Table 4).

According to the authors, there was significant correlation between low Apgar score and meconium amniotic fluid contamination and statistically significant correlation between histopathological findings with greater severity of asphyxia with significant change in histopathological findings. Our results are consistent with the literature that the more severe the severity of asphyxia, the more severe the percentage of placental regressive changes and inflammatory changes. Thus, we confirmed that fetal asphyxia was associated with marked regressive and inflammatory changes in the placenta and that the more severe the asphyxia was the more marked the histopathological findings in the placenta were.

CONCLUSION

The histopathological changes of the placenta during fetal asphyxia are as follows.

1) The percentage of regressive changes in the placenta during fetal asphyxia was degeneration 89.1%, atrophy 70.0%, necrosis 53.6% and defect 37.3% and the percentage of inflammatory changes was congestion 53.6%, edema 30.0%, hemorrhage 59.1% and inflammatory cell infiltration 47.3%.

2) The more severe the severity of fetal asphyxia, the more severe the histopathological changes of the placenta.

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