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Hemoglobinopathies in neonates with intrauterine growth restriction: frequency and clinical implications

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Hemoglobinopathies are among the most common inherited disorders worldwide and represent a significant cause of pediatric morbidity. However, their role in neonatal populations, particularly in infants with intrauterine growth restriction (IUGR), remains insufficiently studied.
Aim – to assess the frequency and clinical characteristics of hemoglobinopathies in neonates with IUGR.
Materials and methods. A total of 103 term neonates with IUGR (Main group) and 50 healthy term neonates (Control group) were examined. Hemoglobin fractions (HbF, HbA_1, HbA_2) were analyzed in umbilical cord blood. Screening for hereditary hemoglobinopathies, including alpha- and beta-thalassemia and structural hemoglobin variants (HbS, HbD, HbC, HbE), was performed using isoelectric focusing, capillary electrophoresis, and high-performance liquid chromatography. Follow-up assessments were conducted at 6 months of age. Statistical analysis was performed using standard methods, with $p<0.05$ considered significant.
Results. Neonates with IUGR demonstrated significantly lower levels of fetal hemoglobin (HbF) and higher levels of HbA_1 and HbA_2 compared with controls. Hemoglobinopathies were identified in 16 (15.5%) infants with IUGR, including alpha-thalassemia (2.91%), beta-thalassemia (7.77%), sickle cell trait (1.94%), homozygous beta-thalassemia (1.94%), and compound heterozygous forms. In contrast, only 2 (4.0%) cases of heterozygous hemoglobinopathies were detected in the Control group. The frequency of hemoglobinopathies was higher in symmetrical IUGR compared with asymmetrical forms.
Conclusion. Children with IUGR exhibit a significantly higher frequency of hemoglobinopathies and distinct alterations in hemoglobin fractions, suggesting a potential role of abnormal hemoglobin genes in the pathogenesis of fetal growth restriction. These findings highlight the importance of early screening and genetic evaluation in neonates with IUGR to improve diagnosis and long-term outcomes.
The study was conducted in accordance with the principles of the Declaration of Helsinki. The informed consent was obtained from patients.
The authors declare no conflict of interest.
Keywords: intrauterine growth restriction, hemoglobinopathy, newborn, thalassemia, sickle cell disease, fetal hemoglobin.

Гемоглобінопатії у новонароджених із затримкою внутрішньоутробного розвитку: частота та клінічні наслідки

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Гемоглобінопатії є одними з найпоширеніших спадкових захворювань у світі та є значною причиною дитячої захворюваності. Однак їхня роль у неонатальній популяції, особливо в немовлят із затримкою внутрішньоутробного розвитку (ЗВУР), залишається недостатньо вивченою.
Мета – оцінити частоту та клінічні характеристики гемоглобінопатій у новонароджених зі ЗВУР.
Матеріали та методи. Обстежено 103 доношених новонароджених зі ЗВУР (основна група) та 50 здорових доношених новонароджених (контрольна група). Фракції гемоглобіну (HbF, HbA_1, HbA_2) проаналізовано в пуповинній крові. Скринінг на спадкові гемоглобінопатії, зокрема альфа- та бета-таласемію та структурні варіанти гемоглобіну (HbS, HbD, HbC, HbE), проведено за допомогою ізоелектричного фокусування, капілярного електрофорезу та високоефективної рідинної хроматографії. Подальші оцінки здійснено у віці 6 місяців. Статистичний аналіз проведено стандартними методами, при цьому значення $p<0,05$ вважалося значущим.
Результати. У новонароджених зі ЗВУР спостерігалися значно нижчі рівні фетального гемоглобіну (HbF) та вищі рівні HbA_1 та HbA_2 порівняно з контрольною групою. Гемоглобінопатії виявлено у 16 (15,5%) немовлят зі ЗВУР, зокрема альфа-таласемію (2,91%), бета-таласемію (7,77%), серповидноклітинну анемію (1,94%), гомозиготну бета-таласемію (1,94%) та складні гетерозиготні форми. Натомість у контрольній групі виявлено лише 2 (4,0%) випадки гетерозиготних гемоглобінопатій. Частота гемоглобінопатій була вищою при симетричному ЗВУР порівняно з асиметричними формами.
Висновок. Діти зі ЗВУР демонструють значно вищу частоту гемоглобінопатій та чіткі зміни у фракціях гемоглобіну, що свідчить про потенційну роль аномальних генів гемоглобіну в патогенезі затримки росту плода. Ці результати акцентують увагу на важливості раннього скринінгу та генетичної оцінки в новонароджених зі ЗВР для покращення діагностики та довгострокових результатів.
Дослідження проведено відповідно до принципів Гельсінської декларації. Від пацієнтів було отримано інформовану згоду.
Автори заявляють про відсутність конфлікту інтересів.
Ключові слова: затримка внутрішньоутробного росту (ЗВР), гемоглобінопатія, новонароджений, таласемія, серповидноклітинна анемія, фетальний гемоглобін.

Hemoglobinopathies in neonates constitute a complex group of inherited disorders influenced by genetic mutations in the human globin genes. These disorders are broadly categorized into quantitative defects (thalassemias), characterized by insufficient production of normal globin chains, and qualitative defects resulting in structural alterations of hemoglobin, such as sickle cell disease (SCD). The perinatal transition from fetal to adult globin chain synthesis critically dictates the timing of clinical presentation, making early detection vital for effective management [4]. Recent therapeutic advances further emphasize the role of neonatal screening in achieving timely diagnosis and improving long-term survival outcomes [11].

Globally, the implementation of neonatal screening exhibits profound disparity. While well-established in the USA and parts of Europe, screening remains heavily restricted in regions with the highest disease burden, such as sub-Saharan Africa, the Caribbean, and India, highlighting a critical global health inequity [12]. In India, where 7,500 to 12,000 babies with beta-thalassemia (ß-thalassemia) are born annually alongside widespread HbS and HbE variants, national management strategies face severe economic and geographical constraints [5]. Similar mutational heterogeneity and challenges in preventive healthcare are observed across the Middle East, including Iraq, where localized program outcomes demonstrate a pressing need for comprehensive screening expansion [1,7].

In the Republic of Azerbaijan, hereditary hemoglobinopathies, particularly ß-thalassemia, represent a paramount public health priority due to a high carrier frequency of 4.0-8.6% [2,9]. Population-based genotyping has revealed 32 distinct *HBB* gene mutations, dominated by codon 8 [-AA] (34.96%), IVS-II-1 [G>A] (16.35%), and IVS-I-110 [G>A] (10.12%), with strong regional clustering, such as the association of codon 8 [-AA] with the Shaki-Zaqatala Region. Additionally, the local population displays a wide spectrum of alpha-thalasssemia (α-thalassemia) deletions (primarily 20.5 kb and 3.7 kb) and structural variants like HbS, HbD-Punjab, and HbE [2]. To mitigate this burden, Azerbaijan launched a national mandatory premarital screening and prenatal diagnosis program in 2015. Over its first five years, the program evaluated 271 high-risk fetal samples, identifying 63 affected cases, which successfully led to medical terminations and a subsequent decline in affected births [9].

Despite these preventive achievements, managing affected infants from birth remains a critical task for clinical neonatology. A particularly vulnerable and high-risk cohort is newborns with intrauterine growth restriction (IUGR). Modern epidemiological data indicate that maternal anemia is significantly associated with an increased risk of fetal growth restriction, with severe forms substantially compounding fetal hypoxia [8]. Furthermore, placental abnormalities, including placenta previa and placenta accreta spectrum (PAS), significantly disrupt placental function and are strongly linked to IUGR and small-for-gestational-age (SGA) outcomes [3,6].

Crucially, the combination of IUGR and placental insufficiency induces severe hematological disturbances in the neonate. Growth-restricted newborns frequently demonstrate significantly elevated nucleated red blood cell (nRBC) counts alongside lower neutrophil and platelet levels at birth, requiring an increased volume of erythrocyte and platelet transfusions during the first week of life (N.J. Reibel et al., 2021). Moreover, IUGR neonates face significantly higher rates of early-onset infections, severe bronchopulmonary dysplasia, and neonatal mortality [10].

When an underlying hemoglobinopathy co-exists with IUGR, these baseline hematological abnormalities and tissue hypoxia may be profoundly exacerbated. However, data regarding the precise frequency and specific clinical implications of hemoglobin variants in neonates born with growth restriction remain scarce, particularly in regions endemic for thalassemia. Therefore, this study aims to evaluate the prevalence of hemoglobin disorders within a cohort of growth-restricted neonates in Azerbaijan and to elucidate how these genetic traits influence their early clinical course, hematological status, and neonatal adaptation.

Materials and methods of the study

A total of 103 term newborns with IUGR were examined (Main group), including 50 infants with the symmetrical type and 53 with the asymmetrical type. The Control group consisted of 50 term newborns without signs of IUGR.

The study material included blood samples obtained from newborns and their mothers. The levels of fetal hemoglobin (HbF) and hemoglobin fractions (HbA, HbA_2) were analyzed in neonatal blood. Umbilical venous blood was collected into tubes containing anticoagulants (ethylenediaminetetraacetic acid or heparin). All newborns with IUGR and their mothers were screened for hereditary hemoglobinopathies, including α-thalassemia, ß-thalassemia, and structural hemoglobin variants (HbS, HbD, HbC, HbE). In cases of compound heterozygosity or homozygosity, additional blood analyses were performed. Hereditary hemoglobinopathies were diagnosed using analytical isoelectric focusing (IEF), followed by quantitative assessment of hemoglobin fractions using capillary electrophoresis (CE) or high-performance liquid chromatography (HPLC). Quantitative measurements were performed using automated HPLC systems. Abnormal hemoglobin S was identified using erythrocyte-based tests. Phenotypic and genotypic frequencies were calculated.

Statistical analysis was performed using Microsoft Excel 2010. Differences were considered statistically significant at $p<0.05$.

Table 1

Analysis of umbilical cord blood in newborns

Biochemical Parameters	IUGR (n=103)	Asymmetrical (n=53)	Symmetrical (n=50)	Control Group (n=50)
HbF	40.7±0.4 (32.8–48)*	42.2±0.6 (32.8–48)*	39.0±0.6 (32.8–48)*,#	64.8±0.8 (53.6–77)
HbA_1	59.3±0.4 (51.8–67.2)*	57.7±0.6 (51.8–67.2)*	61.0±0.6 (52–67.2)*,#	35.3±0.8 (23–47.8)
HbA_2	0.107±0.010 (0.02–0.4)*	0.102±0.013 (0.02–0.4)*	0.111±.015 (0.02–0.4)*	0.038±0.001 (0.02–0.06)

Notes: statistically significant difference compared with the indicators: * – of the Control group (p<0.001); # – of the asymmetrical IUGR group (p<0.001).

Results of the study and discussion

To evaluate the association between IUGR and hemoglobinopathies, umbilical cord blood parameters were analyzed in both the Main and Control groups (Table 1).

In the Control group, the levels of hemoglobin fractions were as follows: HbA_1 – 35.3%, HbA_2 – 0.8%, minor fractions – 0.038% and 0.001%, and HbF – 64.8%.

The level of HbF decreases and accounts for up to 2.5% of total hemoglobin. A characteristic feature of HbF is its higher affinity for oxygen, which ensures effective oxygen exchange between mother and fetus. A reduced level of HbF leads to oxygen deprivation in the fetus and contributes to the development of chronic intrauterine hypoxia.

Apparently, a low level of HbF is one of the main factors influencing IUGR. However, the possibility of the influence of pathological hemoglobin genes on the development of IUGR cannot be excluded. Each pathological hemoglobin gene, depending on its expression, results in specific clinical conditions in newborns. Rare cases of combinations of hemoglobin variants have also been identified in children with IUGR. These include combinations of α- and ß-thalassemia, as well as combinations of each separately with one of four stable abnormal hemoglobin types: HbS, HbD, HbE, and HbC.

HbA_1 levels in the Control group (35.3±0.8%) significantly differed from those in newborns with IUGR (59.3±0.4%, asymmetrical type – 57.7±0.6%, symmetrical type – 61.0±0.6%).

The concentration of the minor hemoglobin fraction HbA_2 in the Control group was 0.038±0.001%, which was significantly lower compared with children with IUGR (0.107±0.010%, asymmetrical type – 0.102±0.013%, symmetrical type – 0.111±0.015%).

Statistically significant decreases in HbF and increases in hemoglobin fractions HbA_1 and HbA_2 in newborns with IUGR suggest the presence of pathological hemoglobin genes. To verify the obtained data, longitudinal follow-up of children was conducted over a period of 6 months.

In the first case, repeated examination at 6 months in children with IUGR revealed heterozygous carrier states in 13 (12.62%) of 103 cases: 3 (2.91%) cases of α-thalassemia, 8 (7.77%) cases of ß-thalassemia, and 2 (1.94%) cases of sickle cell trait. In 2 (1.94%) cases, homozygous states of ß-thalassemia were identified, and in 1 (0.97%) case, a double heterozygous condition for ß-thalassemia with a gene of SCD was detected. In total, 16 (15.5%) newborns were diagnosed with various forms of hemoglobinopathies.

In cases of α-thalassemia, isoelectric focusing revealed minor fractions of abnormal hemoglobin Bart's and HbH, characteristic of this pathology. All children were heterozygous carriers of α-thalassemia.

In ß-thalassemia, isoelectric focusing demonstrated an increase in the minor hemoglobin fraction HbA_2 by more than 3.5%, ranging from 4.06% to 6.23% (mean 5.18%). HbF levels exceeded the normal range only in 8 (7.77%) children and varied between 2.71% and 4.22% (normal up to 2.5%).

In 2 (1.9%) cases, children with IUGR at 6 months showed high levels of HbF (44.83% and 86.38%), corresponding to homozygous ß-thalassemia. In one (0.97%) case (HbF 44.83%), all hemoglobin fractions (HbF, HbA_1, HbA_2, HbA_3) were identified, whereas in the second case, only HbF was detected, indicating a complete absence of beta-globin chain synthesis. These findings reflect defects in beta-polypeptide chain biosynthesis and correspond to the ß-thalassemia phenotype. In contrast to the first child, in the second case (HbF 86.38%), IEF did not detect the HbA_1 fraction or its derivatives. Isoelectric focusing revealed three hemoglobin fractions: HbA_2, metHbF, and HbF, indicating a complete impairment of beta-polypeptide chain biosynthesis. Such a defect in beta-polypeptide chain biosynthesis corresponds to the $ß^0$-thalassemia phenotype.

In 2 (1.9%) children, abnormal hemoglobin was identified by IEF (6.48%), corresponding to HbS

Table 2

Frequency of hemoglobinopathies in children with IUGR

Type of Hemoglobinopathy	Phenotypic frequency (%)	Gene frequency (proportion)
α-thalassemia	2.91	0.0073
ß-thalassemia	7.77	0.0388
SCD	1.94	0.0097
Homozygous ß-thalassemia	1.94	0.0197

Table 3

Distribution of hemoglobinopathy cases by clinical variants of IUGR

Hemoglobinopathy	Asymmetric IUGR		Symmetric IUGR	
	Phenotypic Frequency, %	Gene Frequency	Phenotypic Frequency, %	Gene Frequency
α-T/N	3.77 (n=2)	0.0094	2.0 (n=1)	0.0047
β-T/N	7.55 (n=4)	0.0377	8.0 (n=4)	0.0400
HbS/N	1.89 (n=1)	0.0094	2.0 (n=1)	0.0094
β-T/β-T	3.77 (n=2)	—	—	—
β-T/HbS	1.89 (n=1)	—	—	—
α-thalassemia gene	—	0.0094	—	0.0047
β-thalassemia gene	—	0.0849	—	0.0400
HbS gene	—	0.0189	—	0.0094

(SCD). Identification was confirmed by identical electrophoretic mobility on IEF and hemoglobin electrophoresis with cellulose acetate, as well as positive sickling tests. All blood samples with abnormal hemoglobin HbS showed heterozygous carrier states.

In one (0.97%) case, a newborn with IUGR at 6 months demonstrated elevated HbF levels (44.9% of total hemoglobin). Under conditions of high HbF levels, differentiation of hemoglobin fractions was not possible. The absence of HbA_1 and the high HbF level (44.9%), in combination with decreased HbA_2 levels, indicate a compound heterozygous state involving ß-thalassemia and SCD.

In the Control group, only 2 (1.9%) children exhibited heterozygous states: in one case α-thalassemia, and in another case ß-thalassemia, each with a frequency of 2%.

Table 2 presents the frequencies of hemoglobinopathies in children with IUGR. The highest phenotypic and corresponding gene frequencies were observed for heterozygous beta-thalassemia (7.77% and 0.076, respectively). Alpha-thalassemia ranked second (2.91% and 0.029). Sickle cell trait and homozygous beta-thalassemia were each detected in 2 (1.94%) children, with identical frequencies and gene frequencies of 0.0194. Only 1 (0.97%) newborn with IUGR had a double heterozygous state for 2 alleles of the beta-globin gene.

Among children with IUGR, 16 (15.5%) cases of hemoglobinopathies were identified, with equal distribution between symmetrical and asymmetrical variants (Table 3).

In the asymmetrical IUGR group, 10 (18.9%) children were identified: 4 heterozygous beta-thalassemia (7.55%), 1 HbS carrier (1.89%), 2 heterozygous alpha-thalassemia (3.77%), 2 compound beta-thalassemia (β-T/β-T) – 3.77%, and 1 combined genotype β-T/HbS (1.89%).

In the symmetrical IUGR group, the phenotypic frequency and gene frequency were 12.0% 6 children) and 0.0541, respectively. In this group, 1 (2.0%) children had heterozygous alpha-thalassemia, 4 (8.0%) had heterozygous beta-thalassemia, and 1 (2.0%) had HbS/N.

The results of the present study suggest a possible association between IUGR and hereditary disorders of hemoglobin synthesis. Term newborns with IUGR demonstrated significantly lower HbF levels and higher HbA_1 and HbA_2 concentrations compared with the Control group. Since HbF plays a key role in fetal oxygen transport due to its high affinity for oxygen, its reduction may contribute to chronic intrauterine hypoxia and impaired fetal growth.

Follow-up examination at 6 months confirmed the presence of various hemoglobinopathies in 16 children with IUGR, accounting for 15.5% of cases. The most frequent abnormality was heterozygous ß-thalassemia, followed by α-thalassemia, sickle cell trait, homozygous ß-thalassemia, and compound ß-tha-

lassemia/HbS genotype. In contrast, only two heterozygous hemoglobinopathy cases were identified in the Control group.

The predominance of ß-thalassemia is clinically significant, as increased HbA_2 and, in some cases, elevated HbF are typical laboratory markers of impaired beta-globin chain synthesis. The detection of Hb Bart's and HbH in children with α-thalassemia also confirms the diagnostic value of isoelectric focusing for identifying abnormal hemoglobin fractions in early childhood.

Hemoglobinopathies were detected in both symmetrical and asymmetrical variants of IUGR. However, gene frequency was higher in the symmetrical IUGR group, which may indicate a closer relationship between hereditary hemoglobin disorders and early, prolonged impairment of fetal growth. At the same time, their presence in both groups suggests that hemoglobinopathies should be considered not as the only cause of IUGR, but as an additional factor contributing to fetal growth restriction.

Conclusions

The study demonstrated a relatively high frequency of hereditary hemoglobinopathies among children with IUGR. Altered hemoglobin fraction composition, characterized by reduced HbF and increased HbA_1 and HbA_2 levels, may indicate the presence of pathological hemoglobin genes and reflect intrauterine compensatory responses to chronic hypoxia. Pathological hemoglobin genes may contribute to IUGR through impaired oxygen transport, chronic fetal hypoxia, and abnormal globin chain synthesis. These findings support the need for early postnatal screening for hemoglobinopathies and medical genetic counseling, particularly in regions with a high prevalence of thalassemia and abnormal hemoglobin variants. The conducted studies confirm a high prevalence of hereditary hemoglobinopathies in children with IUGR and underscore the importance of developing preventive strategies for hereditary diseases, including early postnatal diagnosis and medical genetic counseling.

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