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Магнитно-резонансная томография в диагностике редкого генетического заболевания — недержания пигмента (синдром Блоха–Сульцбергера) — на примере клинического случая

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АННОТАЦИЯ

Недержание пигмента (синдром Блоха–Сульцбергера) — редкое наследственное заболевание, проявляющееся характерными кожными высыпаниями и поражением других органов и систем. Магнитно-резонансная томография является приоритетным методом для визуализации структурной патологии головного мозга и прогноза неврологической манифестации у ребёнка.

Ключевая роль диагностики заболевания недержания пигмента принадлежит дерматологу; подтверждающая диагностика проводится путём молекулярно-генетического анализа гена *IKBKG*.

В представленном клиническом наблюдении у новорождённой девочки с высыпаниями на кожных покровах, типичными для синдрома Блоха–Сульцбергера, и выявленной делецией в гене *IKBKG* проводилась магнитно-резонансная томография головного мозга, где были обнаружены многочисленные очаги ишемии, кровоизлияния и поражение проводящих путей.

Магнитно-резонансная томография головного мозга у пациентов с синдромом Блоха–Сульцбергера используется для оценки тяжести поражения вещества мозга, что позволяет объяснить причину неврологических симптомов, скорректировать реабилитационные мероприятия, а также прогнозировать развитие ребёнка.

Ключевые слова: недержание пигмента; синдром Блоха–Сульцбергера; магнитно-резонансная томография; дегенерация проводящих путей; ген *IKBKG*.

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Magnetic resonance imaging for diagnosing a rare disease: incontinentia pigmenti (Bloch–Sulzberger syndrome) on the example of a clinical case

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ABSTRACT

Incontinentia pigmenti, also known as Bloch–Sulzberger syndrome, is a rare hereditary disease characterized by typical skin rashes and involvement of other organs and systems. Magnetic resonance imaging stands as the primary method for visualizing the structural pathology of the brain and predicting neurological manifestations in an affected child.

Diagnosing incontinentia pigmenti predominantly falls within the domain of dermatologists; verification is performed by molecular genetic analysis of the *IKBKG* gene. This study involved magnetic resonance imaging of the brain in a patient with skin rashes, characteristic of Bloch–Sulzberger syndrome, and deletion in the *IKBKG* gene, where numerous foci of ischemia, hemorrhages, and lesions of the tracts were detected.

Magnetic resonance imaging of the brain in patients with Bloch–Sulzberger syndrome is used to evaluate the severity of damage to the brain substance, which makes it possible to explain the cause of neurological symptoms and correct habilitation, as well as predict the development of the child.

Keywords: incontinentia pigmenti; Bloch–Sulzberger syndrome; magnetic resonance imaging; white matted tracts degeneration; *IKBKG* gene.

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磁共振成像在罕见遗传性疾病（即色素失禁症，也称布洛赫-苏兹伯格综合征）诊断中的应用：临床病例

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简评

色素失禁症（布洛克-苏兹伯格综合征，Bloch-Sulzberger Syndrome）是一种罕见的遗传性疾病，表现为特征性皮炎以及其他器官和系统的损坏。磁共振成像是显示出大脑结构病变和预测儿童神经系统表现的优先方法。

皮肤科医生在色素失禁症的诊断中起着关键作用；需要通过对IKBKG基因进行分子遗传分析，以确诊。

一名新生女婴患有典型的布洛赫-苏兹伯格综合征皮炎和IKBKG基因缺失，在进行脑磁共振成像检查后，医生发现了多处缺血、出血和传导通路病变。

布洛赫-苏兹伯格综合征患者的脑磁共振成像可用于评估脑物质损坏的严重程度，这有助于解释神经症状的原因、调整康复措施和预测患儿的发展。

关键词：色素失禁症；布洛克-苏兹伯格综合征；磁共振成像；传导通路变性；IKBKG基因。

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BACKGROUND

Skin, hair, teeth, nails, eyes, and the central nervous system (CNS) are all damaged by incontinentia pigmenti (OMIM 308300: Familial male-lethal type Bloch–Sulzberger syndrome; incontinentia pigmenti type II). The condition typically appears in the first few days or weeks of life.

In the global literature, 2,000 cases of incontinentia pigmenti have been described, and the number is increasing. The global prevalence of incontinentia pigmenti is estimated to be 0.7 cases per million people. The incidence in Europe is 1.2 cases per 100,000 neonates [1]. Mutations in the inhibitor of kappa B kinase gamma (*IKBKG*) gene at position Xq28 on the X chromosome's long arm cause the disorder. The gene regulates apoptosis, cell cycle, inflammation, immunology, and other processes [2–4].

The condition has a wide spectrum of clinical manifestations, ranging from a mildly reduced quality of life to deadly results. The changes in the nucleotide sequence in the gene result in structural changes in the brain [5]. Magnetic resonance imaging (MRI) can detect anomalies, which is useful in identifying various monogenic [6] and multifactorial inherited disorders [7].

Incontinentia pigmenti is inherited in an X-linked dominant trait that kills most male embryos during development. The affected girl-to-boy ratio is 20:1.

Multiple organs and systems are involved in incontinentia pigmenti, most of which emerge from the ectoderm. In 100% of cases, several skin eruptions are found. The disease is distinguished by the linear alignment of the vesicles and pustules along the lines of Blaschko. Patients with incontinentia pigmenti have been documented to have alopecia, oral development anomalies, and fingernail dystrophy. Furthermore, affected patients are more likely to develop ocular pathologies, such as retinal hypervascularization, which can lead to retinal detachment if left untreated (typically occurs during the first 6 yr of life) [8]. Strabismus, cataracts, optic atrophy, retinal pigmentation, and microphthalmia have also been reported. According to several authors, the CNS is involved in 10% to 30% of cases [9], resulting in seizures of varying severity (cases ranging from a single seizure episode to chronic epilepsy), cognitive impairment, mental retardation, and spastic paresis. Less common disorders include breast aplasia, supernumerary nipples, primary pulmonary hypertension, and leukocytosis.

Molecular genetic testing identifies *IKBKG* mutations, and histological examination of the skin tissue samples is used to diagnose the condition. There is a clinical indication [10] to suspect pigmentary incontinence.

The treatment is symptomatic to prevent skin infections, retinal detachment, and epileptic seizures. Dental implants or orthodontic correction are recommended for dental problems, whereas therapy is recommended for spastic activity or paresis.

CASE REPORT

A 14-day-old girl was taken to the Neonatal and Early Age Pathology Department of the National Medical Research Center for the Children's Health of the Ministry of Health of the Russian Federation.

Prenatal history

In the first and second trimesters of pregnancy, there was a threat of miscarriage due to cytomegalovirus infection (confirmed by polymerase chain reaction) in the first trimester; the mother had inpatient therapy. The mother experienced three episodes of acute respiratory virus infections in the third trimester and herpetic eruptions twice.

The infant was delivered at 38 weeks gestation as the second full-term spontaneous childbirth: body weight at birth, 3,470 g; body length, 53 cm; and Apgar score: 9/9.

The post-natal condition was satisfactory. Extensive exanthema covered the entire body, including the trunk, face, and limbs (Figure 1). The eruption had been removed from the scalp. The laboratory results were age-appropriate; the inflammatory indicators were not present.

The general condition began to deteriorate on Day 4: cyanotic spots appeared on the limbs; the skin was icteric, with a grayish hue; the breathing became weakened, with a tendency to tachypnea; low blood oxygen saturation (SpO₂: 81%–95%) was determined; hyperexcitability at examinations and depressed consciousness at rest were observed; upper and lower limb muscles were hypertonic; and head lag as well as seizures were demonstrated.



Fig.1. Vesicles aligned with the lines of Blaschko.

Laboratory data showed the following changes: blood pH reduced to 7.242, blood lactate increased to 6.4 mmol/L, and HCO_3^- decreased to 16.7 mmol/L (metabolic acidosis).

Dermatological examination revealed that the skin lesions had receded and that linear brown-to-light pink pigmentation had appeared, which had turned to hypopigmentation during the follow-up. Bloch–Sulzberger syndrome was the preliminary diagnosis.

Molecular genetic testing was run to identify exons 4–10 deletion in *IKBK*: multiplex allele-specific polymerase chain reaction with primers as described by Haque et al. [11] was used. The findings revealed heterozygous deletion of *IKBK* exons 4–10 in patients with the Bloch–Sulzberger syndrome, as published in the Human Gene Mutation Database Professional. This frequent mutation in incontinentia pigmenti occurs in 65% of patients [12].

On Day 4, the girl's condition was assessed as moderately severe due to the syndrome of CNS depression. Neurosonography revealed extensive ischemia abnormalities in the brain parenchyma and numerous focal changes in the parasagittal and periventricular areas.

On Day 7, an MRI indicated large areas of tiny focal lesions in the cerebral hemispheres, with secondary involvement of the corpus callosum and the corticospinal tract. The findings were interpreted as the result of repeated ischemic strokes induced by incontinentia pigmenti (Bloch–Sulzberger syndrome).

DISCUSSION

Bloch–Sulzberger syndrome is caused by a genetic abnormality that increases the sensitivity of cells developing from ectoderm to cytokine effects, resulting in apoptosis [13]. The skin and its derivatives (nails, hair, and teeth) and the nervous system are formed from the ectoderm. Disease

symptoms commonly arise within the first 10 days of life. Because the disease stage at birth varies, the previous stages are assumed to occur *in utero*.

The disease manifested in the described patient in a typical manner during the first days of life, beginning with linear skin eruptions, which later experienced incremental modifications in accordance with the disease pathomorphosis and were accompanied by neurological symptoms.

Multiple small (min. 2 mm) restricted diffusion lesions were found in the deep white matter, in and under the cortex, in the corpus callosum, posterior limb of the internal capsule, cerebral peduncles, along the corticospinal tract, and other tracts (Figure 2). Changes in the spinal pathways can be interpreted as either direct injury from incontinentia pigmenti or an early indicator of Wallerian degeneration (pre-Wallerian condition). The latter is characterized by spinal tract injury caused by neuron death and the degradation of the myelin sheath [14]. Small patches of restricted diffusion were regarded as tissue necrosis (infarctions).

There are reports in the literature of minor focal brain infarctions caused by injury to the walls of small and medium-sized arteries, resulting in microhemorrhages and thrombosis. There have also been reports of extensive bilateral hemorrhagic necrosis cases with broad brain tissue damage [15].

Among the numerous lesions in the presented instance, the brain MRI revealed single regions with hemorrhagic features compatible with ischemia (Figure 3a). This suggests that not all ischemia foci were accompanied by bleeding. Although necrotic areas eventually turn into encephalomalacia areas, some areas with minimal damage may recover entirely and even be consistent with the normal structure of brain matter in MRI [16]. The hyperintense signal was seen in the cortical area and at the gray/white matter interface in the frontal and parietal lobes in T1-weighted images (T1-WI)

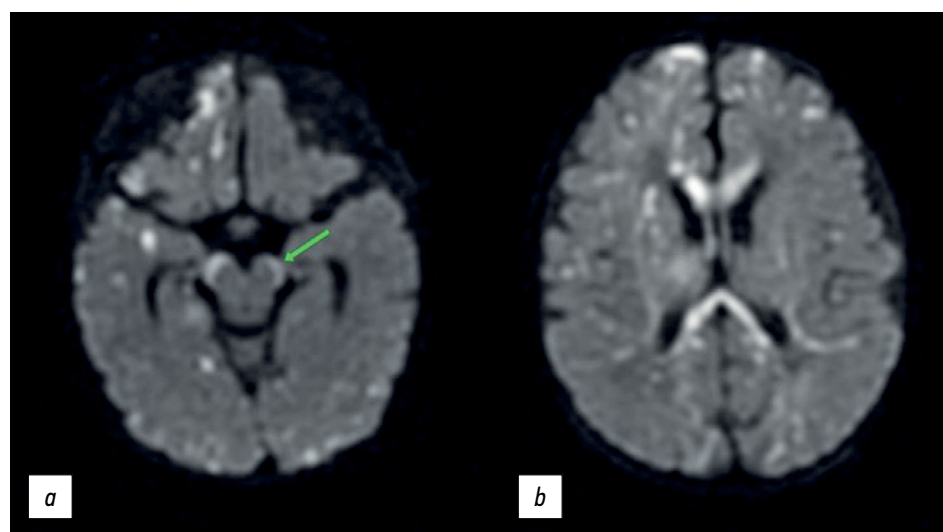


Fig. 2. Diffuse-weighted brain images in the axial plane: (a) the arrow shows the hyperintense signal from the spinal tracts in the brain peduncles and (b) multiple lesions and involvement of the corpus callosum.

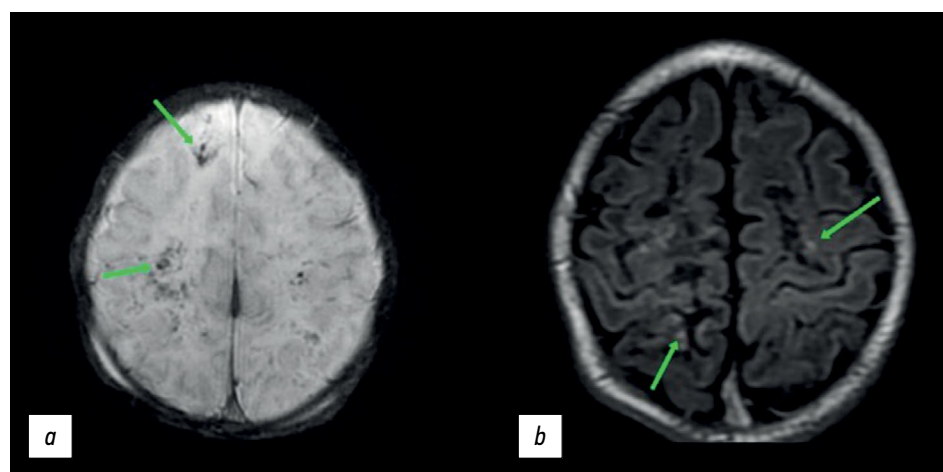


Fig. 3. Magnetic resonance imaging (MRI) of the brain: (a) susceptibility-weighted images (the arrows show microhemorrhages) and (b) T1-weighted images (the arrows indicate hyperintense areas of the cortical necrosis).

(Figure 3b). These areas resemble cortical necrosis, which occurs after ischemia damage to the cortex, resulting in monocyte infiltration and phagocytosis of cell fragments in the damaged structures. T1 images are hyperintense due to deposits of denatured protein of the dead cells and/or lipid-loaded macrophages [17].

Furthermore, histologically validated cases of inflammatory changes of the arachnoid mater and the brain with eosinophilic infiltration in patients with incontinentia pigmenti have been documented, mimicking infection-related damage without evident vascular disorders [18].

First, an MRI-based differential diagnosis is made for encephalitis and neonatal hypoxic-ischemic damage. Encephalitis is distinguished by alternating short and long areas of hyperintense signal in T2-WI with possible restricted or hyperintense diffusion, whereas incontinentia pigmenti is distinguished by chaotic small-lesion damage primarily localized in the white matter. The perinatal ischemia image corresponds to brain structural development patterns; full-term neonates generally have periventricular leukomalacia, basal ganglia damage, or violations that follow the artery patterns. Differential diagnosis of embolic stroke is complex and requires a comprehensive approach, with medical history collection and patient examination.

Vesicular rash and neurological signs may be mistaken for manifestations of herpes infection. In this case, MRI results, typical skin eruptions with evident stages of pathomorphosis, and a negative test for herpes infection should prompt clinicians to consider incontinentia pigmenti.

CONCLUSION

Because the skin eruptions are specific and follow clear stages, a dermatologist is essential in identifying incontinentia

pigmenti. The molecular genetic test for *IKBKG* mutations is also important in diagnosis.

Brain MRI is the first imaging tool to assess brain matter injury when patients with Bloch–Sulzberger syndrome develop neurological symptoms. This method is safe for dynamic follow-up and enables objectively assessing rehabilitation potential, correcting the rehabilitation plan, and predicting the child's development.

ADDITIONAL INFORMATION

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