

PEDIATRICS

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IRON DEFICIENCY ANEMIA IN CHILD WITH LEFT-SIDED DIAPHRAGMATIC HERNIA (case report)

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Abstract: Diaphragmatic hernia is a common pathology of the digestive system in children. The most dangerous complications in surgical practice include inflammation of hernia, strangulation of hernia, bleeding and intestinal obstruction. In pediatric practice complications of diaphragmatic hernia are as follows: gastroesophageal reflux disease, peptic ulcer, pyothorax, pneumonia, cardiac arrhythmias, dysuric disorders, and deficiency anemia. Since diaphragmatic hernias in children are characterized by poor clinical symptoms, they cause difficulties in diagnosis. In examination of patients with refractory forms of anemia, it is necessary to consult a gastroenterologist and a hematologist to exclude digestive tract abnormalities, perform therapeutic correction and provide adequate therapy for this pathology. Clinical observation of a 7-year-old child who underwent inpatient treatment in the Regional Children's Clinical Hospital and was diagnosed with iron deficiency anemia of moderate severity and left-sided diaphragmatic hernia.

KeyWords: diaphragmatic hernia, anemia, clinical case, children.

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This problem is relevant not only in connection with the increased incidence, but also the high probability of complications. According to the statistics, more than 700 000 children have been born with diaphragmatic hernia since 1 January 2000. Diaphragmatic hernia occurs with a frequency of 1 per 1700. Mortality in this disease is 1-3% of the total infant mortality rate, and during the first year of life in 10% of deaths due to malformations.

Illustrative is the data that 147 children are born with this disease every day, that is, a child with diaphragmatic hernia is born every 10 minutes [1].

Hiatal hernia is more common at older age. Hiatal hernia affects about 0.5% of the total adult population [2].

A 7-year-old boy was admitted to hospital presenting with pallor, weakness, decrease in appetite, dysgeusia. He developed the abovementioned symptoms a year ago. According to his medical history he was registered for pediatric consultation this year, and was administered bivalent iron preparations, but without effect. He did not seek hematology consultation.

On examination: state of moderate severity, pale skin, epithelial changes (trophic disorders of the skin, nails, hair, mucous membranes).

On auscultation: vesicular breathing, murmur in the left side of the chest. Loud and rhythmic heart sounds. Soft and painless abdomen, parenchymal organs are not enlarged.

Due to atypical auscultation findings above the left lung the patient was referred to chest X-ray. Chest X-ray findings: lung fields without focal changes. Heart, mediastinum shifted to the right. Left diaphragmatic cupula and gas-filled gastric fundus are at the level of the 4-th rib. Right, left sinuses are clear. Relaxation of the left diaphragmatic cupula. Conclusion: left-sided diaphragmatic hernia.

Laboratory findings. Complete blood count: RBC-3.4 * 10¹²/L; HGB - 85g/L; MCH - 0,7; PLT - 180 * 10⁹/L; Retic - 0,2%; WBC - 4.0 * 10⁹/L; BSR-15.

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Biochemical blood assay: serum iron - 8mcm /l; total iron binding capacity - 50mcm/L; latent iron binding capacity - 45mcm/L; serum ferrites - 10 mcm/L.

Diagnosis: Iron deficiency anemia of moderate severity. Left-sided diaphragmatic hernia.

Therapy. Operative correction: laparotomy, surgical repair of the diaphragmatic cupula, with local tissues.

After surgery he was prescribed bivalent iron supplements at a dose of 5 mg per kg per day for 6 months. The child's condition improved significantly, no complaints, laboratory indices (RBC-4.0 10¹²/L; HGB - 110 g/L; MCH - 0.98). He received hematological consultations for a year, after which he was deregistered.

There are various complications of diaphragmatic hernias which include inflammation of hernia, strangulation of hernia, bleeding and intestinal obstruction. The most common complications in pediatrics are gastroesophageal reflux disease, peptic ulcer, pyothorax, pneumonia, cardiac arrhythmias, dysuric disorders, and deficiency anemia.

Major pathogenic mechanisms of anemia development in diaphragmatic hernias include gastrointestinal bleeding in strangulation, disruption of iron absorption [3].

In physiological conditions absorption occurs mainly in the duodenum and first part of the jejunum. In iron deficiency the absorption zone extends distally. Iron is absorbed both as heme (10% of the absorbed iron) and non-heme (9%). The degree of its assimilation is determined by a number of factors, which may both interfere and promote iron absorption. Most of ferric iron (III) forms insoluble salts, for example, with phytin, tannin and phosphate present in food, and is excreted in the feces. Bioavailability of ferric iron in food and synthetic hydroxide iron complexes (III) is determined by iron release rate and the concentration of iron-binding proteins such as transferrin, ferritin, mucins, integrins and mobilferrin.

The amount of iron absorbed by the body is strictly controlled by the mechanism, the details of which have not yet been fully studied. Absorption of iron from ion compounds depends on iron ions valence. Iron is absorbed by mature enterocytes mainly in the form of ferro-ions (Fe²⁺). Ferric iron (ferric ions) is partially imported into enter-

ocytes, but the majority is reduced to ferro-ions. Ferric ions (Fe³⁺) interact with mucin and β 3-integrin and are imported into intracellular space of enterocyte villi of duodenal mucosa membrane by extracellular chaperone, namely calreticulin-like mobilferrin. A certain part of Fe³⁺ ions on apical surface of the villous epithelium under the impact of membrane-bound ferrireductase enterocyte brush border (duodenal cytochrome b - Dcytb) is reduced to the ferrous state (ferro-ions Fe²⁺). Full iron absorption in the duodenum occurs only if mucosa of the intestine functions normally. If a person has gastrointestinal diseases, it will provoke a damage of the intestinal mucosa, thus reducing the inflow rate of iron in the body.

Malabsorption of iron can be triggered by inflammation, cicatrical or atrophic processes in the small intestine, resection of the small intestine and the presence of gastrointestinal hernias. Clinical manifestations include pallor, lethargy, loss of appetite and taste perversion [4].

The development of iron deficiency has a clear staging. Sequentially developing stages of iron deficiency are as follows:

- Latent iron deficiency is characterized by a decrease in iron accumulation and the beginning of iron deficiency erythropoiesis;
- Iron deficiency anemia is characterized by a combination of sideropenic and anemic syndromes (Table 1) [5].

The main criteria for diagnosis in children: peristaltic noises in the lungs on auscultation, increasing fatigue, lethargy, lack of presentation typical for diaphragmatic hernia.

According to statistics deficiency anemia is observed in 15% of children, they vary widely - some are related to the lack of B vitamins, while others - with folic acid deficiency, but the overwhelming majority of anemia cases develop because of iron deficiency, these types are called iron-deficiency anemia (IDA), and they are the most common (about 80% of anemia cases) [6].

Table 1
Stages of iron deficiency states and their main characteristics

The stage of iron deficiency	Main characteristics
The first stage Latent iron deficiency Decreased accumulation of iron.	<ul style="list-style-type: none"> • Decrease in tissue stores of iron. • Parameters of the iron transport fund (serum iron, total iron binding capacity, latent iron binding capacity, ferric iron saturation ratio) within the limits of age norms. • Hb concentration is normal. • In adults, there is a compensatory increase in iron absorption in the intestine. • In children, the absorption of iron from food decreases due to reduced activity of enzymes involved in the absorption of iron in the intestine. • There are no clinical manifestations. • Laboratory criteria: a decrease in serum ferritin concentration, an increase in the concentration of soluble transferrin receptors.
The second stage Latent iron deficiency Iron deficiency erythropoiesis.	<ul style="list-style-type: none"> • Decrease in tissue stores of iron. • Reduced content of deposited iron and iron transport fund. • Gradual decrease in the activity of enzymes containing iron. • Hb synthesis rate, its concentration, total number and saturation of Hb red blood cells are not changed, so there is no anemia. • There are clinical manifestations, caused by a decrease in the activity of enzymes containing iron (sideropenic syndrome). • Laboratory criteria: decreased serum ferritin concentration, increased concentration of soluble transferrin receptor, increased total iron binding capacity; serum iron concentration may be normal.
The final iron deficiency stage. Iron-deficiency anemia Clinically manifested state.	<ul style="list-style-type: none"> • Iron stores are depleted. • Hb synthesis and its concentration decrease. • Zinc protoporphyrin increases. • There are morphological changes in erythrocytes: microcytosis, anisocytosis, and poikilocytosis. • Reduced saturation of Hb erythrocytes and as a result, increased hypochromia. • Development of anemic hypoxia. • Dystrophic changes in tissues and organs.

According to the World Health Organization (WHO), more than 500 thousand people in the world suffer from IDA.

The prevalence of IDA in children in Ukraine and developed European countries is approximately 50% in preschool children and 20% in teenagers [7].

Iron deficiency leads to various pathological states, infectious diseases of the gastrointestinal tract and respiratory system; brain structures cannot function normally without iron and psychological development is disturbed. Children diagnosed with iron-deficiency anemia in infancy, at the age of 3-4 years are found to have disruption of the transmission of nerve impulses from the brain centers to the organs of hearing and visual impairment due to violations of myelination and, as a consequence, violation of nerve conduction [8].

Establishment and elimination of its causes are crucial in the treatment of iron deficiency anemia. Therapy only with iron preparations in the presence of underlying disease that led to the IDA does not bring good results. Complete absence of any complaints typical for gastrointestinal disorders was a clinical feature of the abovementioned clinical case where the only symptom of diaphragmatic hernia was anemia with leading sideropenic syndrome [9, 10].

4. CONCLUSIONS

Gastrointestinal abnormalities may be one of the factors triggering iron deficiency anemia and thus, patients with prolonged iron deficiency require comprehensive examination of the gastrointestinal tract.

CONFLICT OF INTERESTS

There is no conflict of interests.

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