

Iron Deficiency Anemia in Children with Chronic Gastroduodenal Pathology

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Abstract: Timely diagnostics and treatment of chronic gastroduodenal pathology in children and adolescents remains one of the urgent directions of modern pediatrics. At the same time the problem of iron deficiency anemia treatment in ChGP in children and adolescents was and still is actual problem in the field of

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Introduction

At the same time, the choice of a more effective method of anemia treatment taking into account the type and severity of the pathology changes from year to year, as the dominant factors themselves in the pathogenesis of the disease change [1,4]. One important cause of IDA in ChGP is impaired iron absorption in the duodenum and proximal jejunum (enteric anemia). At the same time, it should be noted that anemia against CHGP is often accompanied by IDA deficiency not only of iron, but also of vitamin B12, folic acid, and proteins, which gives them a mixed character [1,7]. At the same time, in the practice of a physician, iron-deficient, B12- and folate-deficient anemias are the most common [6].

The pathogenesis of anemia in chronic diseases is complex; it is associated with defective iron reutilization, in which macrophages are unable to release iron into the circulating transferrin transport protein [5].

Iron deficiency states can arise due to insufficient iron intake with food, due to severe restriction of iron-rich food intake while following a diet due to an underlying gastroenterological disease [8]. At the same time, the presence of iron deficiency anemia was a negative premorbid factor for the development of all clinical variants of gastroduodenal pathology (chronic gastroduodenitis without erosions and with erosions) [7].

It should be noted, at IDA of any nature, widespread processes of dystrophy and atrophy develop in all parts of the alimentary canal. These changes are associated with iron deficiency in mucosal cells of the digestive organs, which has a triggering role in the formation of gastritis. Further iron deficiency contributes to deepening of structural changes of glandular elements of the stomach.

The aim of the investigation was to study the effectiveness of correction of iron deficiency anemia in children and adolescents with chronic gastroduodenal pathology.

Materials and methods of research

On the basis of randomized controlled trial including clear criteria of selection of patients, 118 school-aged children with various forms of chronic gastroduodenal pathology in remission period, with clinico-laboratory diagnosed iron deficiency anemia were examined. The mean age of the subjects was $12,4 \pm 1,3$ years. Of them chronic gastroduodenitis was diagnosed in 87 pupils (73,7%), in 31 (26,3%) - chronic gastritis of various forms. Clinic-laboratory examination included detailed blood analysis, determination of serum iron concentration, content of ferritin and transferrin in blood serum.

Results and discussion

Many chronic diseases of the digestive system in children and adolescents are accompanied by development of anemia clinic of various forms and degree of severity. At the same time, anemia can be not only a complication of the underlying disease, but also the first obvious sign of ChGP.

Our studies have shown that in 118 of 251 (47%) children and adolescents with ChGP there was clinical and laboratory evidence of mild to moderate degree iron deficiency anemia. The remaining children were found to have other types of anemia, mainly B12 deficiency anemia (49%).

Analysis of clinical manifestations of LDA in children with ChGP revealed pale skin (48.3%), asthenovegetative disorders as increased fatigability (49.1%), sleep disorders (33%), recurrent headaches (29.7%), presence of specific syderpenic syndrome (35.6%), manifested by dryness and thinning of skin.

After identification of LDA in children with ChGP we started complex therapy of the disease, including nonmedicamental and medicamental treatment of the main disease and anemia.

The course of nonmedicamental treatment consisted of diet and physical therapy. Diet therapy was based on the clinical picture of the underlying disease with a predominance of iron-containing foodstuffs (meat dishes, cereals, some types of vegetables and fruits).

It should be noted that the main principles of drug treatment of LDA are therapy of the underlying disease of the digestive organs, as well as compensation of iron deficiency in the blood and tissues and achievement of complete clinical and hematological remission. As substitution therapy in ALS, iron preparations (ferrotherapy) are used. For this purpose, we chose the anti-anemic drug Ferlatum. The drug contains iron-protein succinylate 800 mg (equivalent to 40 mg Fe³⁺), which is a complex compound where trivalent iron atoms (Fe³⁺) are surrounded by a semi-synthetic protein carrier that prevents damage to the gastric mucosa. Pupils were administered 1 vial (15 ml) per day (40 mg Fe³⁺) in 2 doses, which corresponds to a daily dosage of 1.5 ml/kg/day (in an amount equivalent to 4 mg/kg/day of Fe³⁺).

The choice of this drug was conditioned not only by the presence of a protective protein shell, which eliminates the irritant effect of iron on the gastric mucosa, resulting in a minimal number of side effects and an increased inflammatory process clinic in the gastrointestinal tract, but also by several parameters that affect treatment efficacy:

- high bioavailability (>68%) and rapid absorption, requiring no absorption enhancers:
- The absence of oxidative stress associated with the conversion of Fe²⁺ to Fe³⁺, as well as its damaging effects.

-the absence of concentration peaks and pathological deposition of iron.

Taking into consideration that antacids, calcium preparations and cimetidine decrease iron absorption, we recommended to take FERLATUM 1 hour before or 2 hours after taking these preparations.

Complex treatment of the underlying disease and anemia in schoolchildren with ChGP was carried out for 2 months (9 weeks). Against the background of the complex treatment a number of clinical signs of ChGP and anemia disappeared. After the course of treatment a repeated laboratory-clinical study was carried out. It was found that in children with ChGP hemogram parameters changed depending on the form of gastroduodenal pathology. In particular, more obvious changes in hemogram are characteristic of ChGP. It should be noted that erythrocyte indices are calculated values allowing quantitative characterization of important indicators of erythrocyte condition.

Mean corpuscular hemoglobin (MCH, Mean Corpuscular Hemoglobin) is an index that characterizes the absolute weight content of hemoglobin in one erythrocyte in picograms. In children with ChGD and ChG this index was almost similar, although after a course of anti-anemic treatment in children with CG it exceeded the index of ChGD by 4.2%.

Table 1
Changes in the hemogram of schoolchildren with CHGP before and after a single course of ferrotherapy (n=118)

Bloodcounts	ChG		ChGD	
	Before treatment	After the first course of treatment	Before treatment	After the first course of treatment
Hemoglobin (g/l)	112,7± 3,87*	118,63±4,91*	111,7±3,61* *	115,3±3,87**
Erythrocytes (10 x12/l)	4,5 ± 0,13*	4,6 ± 0,21*	4,4 ± 0,25*	4,5 ± 0,19**
ЦПСР	0,75 ± 0,04*	0,93±0,04*	0,76 ± 0,03*	0,92±0,02**
Hematocrit	33,3 ± 1,16*	39,4±1,08*	31,4±1,51*	38,6 ± 1,58*
MCH, pg	25,1 ± 0,71*	29,8±1,03*	25,4 ± 0,97**	28,6 ± 0,69**
MCHC, %	29,8 ± 1,16*	35,5±1,50*	29,1±1,52**	33,8± 2,04**
Ferritin, µg/L	12,2±0,86*	22,8±0,77*	10,9±0,47**	21,9±0,69**
Transferrin, g/l	3,7±0,82**	3,1±0,91**	3,9±0,07**	3,3±0,75**
TDF, µmol/l	77,3±1,12*	67,3±2,01*	81,6±0,19**	69,7±0,89**

* $p < 0,05$, ** $p < 0,01$

Mean erythrocyte hemoglobin concentration (MCSH, Mean Corpuscular Hemoglobin Concentration) reflects the percentage saturation of erythrocytes with hemoglobin. Decrease of MSNS below 30% is characteristic of erythrocyte absolute hypochromia, which we observed in our study. At the same time, in children with ChG, the MSNS index was slightly higher than in CGD, but after a course of anti-anemic treatment, this index increased by 5.5%, indicating a marked increase in the concentration of hemoglobin in erythrocytes. However, it should be kept in mind that a decrease in MSNS can also occur in macrocytic and especially megalocytic forms of anemia. In these cases, there is a disproportionately large increase in erythrocyte volume compared to an increase in its hemoglobin saturation.

An integral part of the study of iron metabolism in blood is the analysis of ferritin and transferrin content. In the children with ChGP observed, a decrease in ferritin levels was determined. Ferritin itself is the main indicator of intracellular iron depot in the body, playing an important role in maintaining iron in a biologically useful form. Children with ChG decreased ferritin levels to 10 $\mu\text{g/L}$, whereas those with ChGD decreased to 9 $\mu\text{g/L}$. After a course of anti-anemic treatment, the ferritin level increased 1.8-fold on average, reaching normal values, indicating the effectiveness of the comprehensive therapy.

Studies carried out in the 1970s proved that there is a relationship between the total iron content in the body, both in children and adults, and the concentration of ferritin; namely, the total iron content is directly proportional to the concentration of ferritin in the serum. Consequently, the determination of serum ferritin is one of the leading laboratory indicators in the evaluation of iron stores. But ferritin concentration does not always reflect the true state of iron stores, and therefore we carried out a transferrin assay. The main function of transferrin is the transport of iron absorbed in the intestine to its depot (liver, spleen), to reticulocytes and their precursors in the bone marrow. Children with CGD had increased transferrin from 3.6 to 3.9 g/L, an average of 11% more than the maximum reference value (2-3.5 g/L).

Transferrin synthesis is carried out in the liver and depends on iron reserves in the body. In particular, iron from food is accumulated in the epithelial cells of the mucosa of the small intestine, after which transferrin transports iron absorbed in the intestine to its depot (liver, spleen), to reticulocytes and their precursors in the bone marrow

All the above-mentioned differences between ChGP and ChGD are directly related to the physiology of the small intestine and its role in the process of iron metabolism. Consequently, in ChGP children due to chronic inflammatory processes occurring in the small intestine, not only iron absorption, but also its deposition is disturbed, as we noted during the interpretation of the obtained results of the study. In particular, in children with ChGD the transferrin content was 5.4% higher than in ChG children.

One molecule of transferrin binds two iron atoms, the Fe^{3+} ion, and 1g of transferrin, respectively, is about 1.25 mg of iron. Knowing this ratio, one can calculate the amount of iron that serum transferrin can bind; it approximates the value of the total iron-binding capacity of serum. Thus, in children with CHGP, the total serum iron-binding capacity increased to 84 $\mu\text{mol/L}$, reflecting the degree of serum iron starvation and transferrin iron saturation.

After complex therapy and therapeutic and prophylactic measures with simultaneous administration of Ferlatum (Ferlatum) in an age-related dosage, a decrease in the level of total serum iron-binding capacity together with transferrin to a reference value (44.7 - 71.6 $\mu\text{mol/l}$) was observed.

Consequently, the rational prescription of iron preparations, in particular Ferlatum, to children with LDA against CHGP is an integral part of a comprehensive anti-inflammatory therapy. But at the same time, it is necessary to exclude children with gastric and duodenal ulcer disease, as well as other types of anemia, by laboratory and instrumental means.

Conclusions

The data obtained show that in children with chronic gastroduodenal pathology depending on the type of the disease the clinical and laboratory picture of LDA changes, so in children with ChGP it is more pronounced in relation to ChG. All children with laboratory-detected LDA on the background of CHGP should be prescribed ferrotherapy taking into account the drug therapy of the underlying disease.

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