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MEDICAL SCIENCES

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MYELODYSPLASTIC SYNDROME ON THE BACKGROUND OF SEVERE HYPOTHYROIDISM: CASE REPORT

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Summary. Myelodysplastic syndrome (MDS) is a heterogeneous group of clonal diseases of the blood system arising from mutation of the hematopoietic stem cell and characterized by cytopenia as a result of ineffective hematopoiesis, signs of dysmyelopoiesis and a high risk of transformation into acute leukemia. More than 80% of patients with MDS are over 60 years old. About 25 thousand new cases are diagnosed annually in Europe. Given the steady aging of the European population, it is believed that the number of patients with MDS will only increase in the coming decades. In addition, signs of myelodysplasia can be detected in the bone marrow or peripheral blood, not only in MDS, but also in other non-clonal diseases. The role of thyroid hormones in hematopoiesis is known, which does not exclude the development of dysmyelopoiesis in hypothyroidism. The article presents the classification, clinical manifestations of primary MDS and a clinical case of the

development of the disease against the background of severe hypothyroidism with features of the course of MDS.

Key words: myelodysplastic syndrome, single-line dysplasia, severe hypothyroidism

Introduction. Myelodysplastic syndrome (MDS) is a heterogeneous group of clonal diseases of the blood system arising from mutation of the hematopoietic stem cell and characterized by cytopenia as a result of ineffective hematopoiesis, signs of dysmyelopoiesis and a high risk of transformation into acute leukemia [1-3]. In Europe and the United States, the incidence in the general population is about 4-5 cases per 100 thousand population per year. More than 80% of MDS patients are older then 60 years old. Overall, approximately 25,000 new cases are diagnosed annually in Europe.

The purpose is representation the clinical case and its differential diagnosis, and the further management of the patient with refractory anemia (single-line MDS) on the background of hypothyroidism.

Material and methods. There was analyzed the long-term results of observation of the clinical course of MDS with refractory anemia in Dniprovskaya clinical hospital of the railway transport (Dnipro) on the example of this clinical case. The patient had been observed prior to MDS reclassification 2017 therefore the terminology of the diagnosis was represented based on the classification of the WHO, 2008.

Case presentation. A 38-year-old Caucasian woman was admitted to the hospital into the internal medicine department in August 2001. She has disability caused by severe hypothyroidism. She complained on the acute progressive chest pain, shortness of breath, headache, dizziness, weakness in the hands, coldness, numbness of fingers and toes, finger convultions, drowsiness, swelling of the face and hands, memory loss, constipation, menstrual disorders, unstable blood pressure, total weakness,low back pain which radiate to the left buttock and the back surface of the left hip, the heaviness in the right upper quadrant and epigastric area, irritability.

This symptoms had appeared a few months ago, and had progressed in 2 weeks before admission.

She has been suffering from hypothyroidism since the age of 11. She received substitution therapy with L-thyroxine for many years with a gradual dose increasing. There has been a deterioration in the patient's condition since 1994. There was worthering of the drowsiness, oedema syndrome, chilliness of hands, menstrual disorders, dizziness. Also at that period of time the anemia was revealed during the examination. The treatment with iron drugs and vitamin B12, which prescribed by a therapist, gave some positive but not persistent effect. The patient was hospitalized 2 or 3 times every year about hypothyroidism and secondary anemia.

Physical examination. Condition on admission is moderate severity. The height is 155 cm. The weight is 86 kg. BMI is 35.8 kg/m2. The face and hands are pasty. The skin is pale and icteric. There is mild lips cyanosis. The thyroid gland has reduced size and the dense consistency during the palpation. Lymphatic nodes are not palpable. There are not found any hemorrhages. The lungs examination finds pulmonary sound during percussion and vesicular breathing. The heart auscultation finds muffled heart sounds and bradycardia. BP 170/100-100/60 Hg mm. HR 60 per min. The tongue is with teeth imprints. The abdomen during the palpation is soft, and sensitive in the epigastric area and right upper quadrant. The liver enlarge in 4 cm, and the spleen is enlarged in 2 cm. There is a tendency to constipancy.

The laboratory and instrumental investigations. Serum iron 3.98 μmol/l, ferritin 10 ng/ml. Erythropoietin 350 mU/ml. Cyanocobalamin – 135 pmol/l. Folic acid – 10 nmol/l. Glucose, renal and hepatic complexes and coagulogram are within normal limits. Cholesterol - 6.12 mmol/L. Blood calcium - 2.05 mmol/L (below normal). TSH is more than 150 μIU/ml (normal range is 0.3-4.0 μIU/ml), T4 - 0.79 nmol/l (normal range is 62-141 nmol/l), antibodies to TPO - 174.0 U/ml. Parathyroid hormone 17,1 pg/ml. Myelogram counting results 13.02.01: blasts 2.4%, neutrophile promyelocytes 0.4%, n. myelocytes 11.0%, n. metamyelocytes 12.0%, n. bandes 17.0%, mature neutrophils 14.6%, eosinophiles 1.6%, basophiles 0.8%, lymphocytes 9.6%, monocytes 3.0%, plasma cells 1.4%, erythroblastes 1.8%, pronormocytes

1.2%, basophillic normocytes 1.2%, polychromatic normocytes 5.4%, orthochromatic normocytes 15.6%. Megakaryocytes 2 / 500 cells, L/Er 3:1 (N), neutrophil maturation index 0.74 (N), erythroblast maturation index 0.83 (N), signs of dysplasia of cellular elements red row. There were signs of nuclear rejuvenation in normocytes without their pronounced megaloblastoidity in 27% of cells; large cells were rare, more often they were slightly enlarged. Dysplasia of granulocytes was expressed as a decrease in granularity, signs of its immaturity, and a decrease in the number of segments in the cell nucleus. Dysplasia of the megakaryocytic lineage was not observed. The reaction to peroxidase in neutrophils is reduced, there are peroxidase-negative cells larger than lymphocytes, which is typical for MDS with dysplasia [1,2]. Tables 1 and 2 show the indicators of peripheral blood in dynamics over the years.

Table 1

Peripheral blood in dynamics

Date	Red blood cells 10^{12}/l	Hemoglobin g/l	color indicator	WBC 10^9/l	Eosinophiles %	Basophiles %	neutrophile bandes %	neutrophile natures %	Lymphocytes %	Monocytes %	Reticulocytes %	Plateletes 10^9/l	ESR mm/hour
08-Aug-2006	2.2	60	0.82	3.2									29
22-Aug-2006	3	87	0.87	4.9			4	46	34	10			34
12-Feb-2008	3.2	95	0.9	5	2		8	50	28	11	0.8	288	10
26-Feb-2008	3.2	98	0.92	6.8	1		2	60	33	4	0.3	224	26
29-Apr-2008	3.2	85	0.8	4.8									10
13-May-2008	4.1	87	0.64	4.6			8	54	30	8	0.2	340	14
08-Aug-2008	3	78	0.9	3.6	8		3	54	31	10	11	270	10
20-Aug-2008	3.8	118	0.92	7.8	2		16	61	14	3	1.1	193.8	12
26-Sep-2008	2.5	75	0.9	4.8									10
10-Oct-2008	2.7	78	0.87	4.6			8	54	30	8	0.2	340	14
05-Mar-2009	2.4	68	0.86	8.6		1	12	57	23	7	0.5	225	7
12-Mar-2009	2.6	75	0.88	6.2	3	1	11	43	33	9	0.8	156.8	6
05-May-2009	2.6	74	0.9	4.8	6		9	45	24	16	1.3	208	17
13-May-2009	3	92	0.9	6.2	3	1	11	43	33	9	0.8	158.6	6
27-Aug-2009	2.4	84	0.92	4.1	1		11	48	33	7			12
04-Sep-2009	3.4	100	0.88	4.2									10
14-Apr-2010	3.4	97	0.86	6.6	4	4	7	43	33	9		316.2	22
20-Apr-2010	3.1	93	0.91	5.4	5	1	10	37	42	5			30
13-Aug-2010	3.2	85	0.8	5.2	1		12	47	31	9		272	11
21-Aug-2019	4.7	100	0.7	6.7								229	16
21-May-2020	3.9	102	0.8	3.7									6
01-Sep-2020	2.5	89	1.1	4.6								198	4

Table 2

Laboratory tests results in dynamics

Indicator	Date	Range
T4	13.02.08	5.05 mmol/l
	10.09.08	0.5 mmol/l
TPO antibodies	10.09.08	226 mmol/l
TSH	13.02.08	13.51 mmol/l
	10.09.08	6.25 mmol/l
Serum iron	26.02.08	4.3 mmol/l
	13.05.08	5,84 mmol/l
	04.09.09	15 mmol/l
	13.08.10	5,4 mmol/l
	07.02.11	5,4 mmol/l
	08.05.12	9,7 mmol/l
	14.04.13	4,93 mmol/l
	11.08.14	5,85 mmol/l
Total iron binding capacity	11.08.14	62,93 mmol/l
Transferin saturation	11.08.14	9,3 mmol/l
Total bilirubin	26.02.08	12.3 mmol/l
	20.08.08	10.5 mmol/l
	10.09.08	12,5 mmol/l
	13.05.09	22,5 mmol/l
	04.09.09	11 mmol/l
	20.04.10	12,5 mmol/l
	13.08.10	12,5 mmol/l
	07.02.11	12,5 mmol/l
	01.11.11	10,5 mmol/l
	08.05.12	17,5 mmol/l
	14.04.13	13 mmol/l
	11.08.14	12,5 mmol/l
	29.03.16	12,5 mmol/l
ALT	10.09.08	0,5 μmol/l
	13.05.09	1 μmol/l
	04.09.09	0,24 μmol/l
	20.04.10	0,25 μmol/l
	13.08.10	0,3 μmol/l
	07.02.11	0,2 μmol/l
	01.11.11	0,8 μmol/l
	14.04.13	0,4 μmol/l
	11.08.14	0,2 μmol/l
	29.03.16	0,2 μmol/l
Thymol test	10.09.08	5 U
	13.05.09	1,2 U
	04.09.09	8,6 U
	13.08.10	3 U
	20.04.10	11 U
	07.02.11	3 U
	01.11.11	4,5 U
	08.05.12	2 U
	11.08.14	6 U
	14.04.13	5 U
	29.03.16	3,5 U
ALP	14.04.13	1200 U/l
	29.03.16	985 U/l
Creatinine	10.09.08	94 μmol/l
	13.05.09	90 μmol/l
Creatinine Urea	04.09.09	84 μmol/l
	20.04.10	93 μmol/l
	13.08.10	89 μmol/l
	07.02.11	83 μmol/l
	01.11.11	92 μmol/l
	08.05.12	75 μmol/l
	11.08.14	97 μmol/l
	14.04.13	85 μmol/l
	29.03.16	101 μmol/l
	26.02.08	5.3 mmol/l
	20.08.08	4,8 mmol/l
Urea	10.09.08	4,6 mmol/l
	13.05.09	4,7 mmol/l
	04.09.09	4,8 mmol/l
	20.04.10	6,3 mmol/l
	13.08.10	4,7 mmol/l
	07.02.11	3,9 mmol/l
	01.11.11	5,3 mmol/l
	08.05.12	2,6 mmol/l
	14.04.13	4,3 mmol/l
	11.08.14	4,6 mmol/l
	29.03.16	6 mmol/l
	26.02.08	3.9 mmol/l
	04.09.09	4,1 mmol/l
Blood urea nitrogen	20.04.10	9,9 mmol/l
	14.04.13	18,6 mmol/l
	20.08.08	2,27 mmol/l
	10.09.08	2,18 mmol/l
Blood urea nitrogen Cholesterol	13.05.09	2,22 mmol/l
	04.09.09	2,23 mmol/l
	20.04.10	2,96 mmol/l
	13.08.10	2,22 mmol/l
	07.02.11	1,85 mmol/l
	01.11.11	2,49 mmol/l
	08.05.12	1,2 mmol/l
	14.04.13	2 mmol/l
	11.08.14	2 mmol/l
	29.03.16	2,8 mmol/l
	10.09.08	4.49 mmol/l
	13.05.09	3,25 mmol/l
Cholesterol	04.09.09	3,72 mmol/l
	20.04.10	7,51 mmol/l
	13.08.10	5,84 mmol/l
	07.02.11	4,12 mmol/l
	01.11.11	10,2 mmol/l
	08.05.12	5,8 mmol/l
	14.04.13	4,96 mmol/l
	11.08.14	4,49 mmol/l
	29.03.16	4,57 mmol/l
	10.09.08	1,3 mmol/l
	04.09.09	0,85 mmol/l
Triglycerides	20.04.10	2,2 mmol/l
	13.08.10	1,8 mmol/l
	07.02.11	1,72 mmol/l
	01.11.11	0,8 mmol/l
	08.05.12	1,18 mmol/l
	14.04.13	0,5 mmol/l
	11.08.14	1 mmol/l

ECG shows sinus bradycardia and diffuse myocardial changes. Echocardiography shows the dilatation of the left atrium cavities, left ventricular hypertrophy, mild mitral valve regurgitation.

Thyroid ultrasound shows signs of hypoplastic thyroid follicular cysts at the left lobe, signs of the thyroiditis.

Ultrasound of the abdominal cavity shows hepatomegaly and splenomegaly.

Clinical diagnosis: severe autoimmune hypothyroidism stage with chronic autoimmune thyroiditis. Methabolic cardiomyopathy. Heart failure I NYHA. Methabolic hepatosis. Raynaud's syndrome. Mild secondary hypoparathyroidism. Myelodysplastic syndrome with severe progressive refractory anemia. Iron deficiency. Chronic gastritis with secretory insufficiency. Chronic cholecystitis remission stage. Widespread osteochondrosis with pain syndrome. Obesity II stage.

The prescribed treatment are L-thyroxine 350 mcg, Inosinum, Piracetamum, Folic acid, Ferrous sulfate, Iron polymaltose, Cyanocobalamin, Pancreatin, Neurovitan, Midocalm, Diclofenac, Simvastatinum, Celecoxib.

The patient received the permanent outpatient treatment for hypothyroidism and anemia in next years. Also she received inpatient treatment several times each year. Red blood counts and thyroid hormone levels were unstable, but always outside the reference values. The tab.1 shows its dynamic.

In next years the usual serum iron level was in ranges 5-6 μmol/L. Its maximum value was 15.0 μmol/l in 2009; the ferritine was less then 5 ng/ml.

There were episodes of a slight increase in the total bilirubin level up to 22.5 μmol/l with a predominance of indirect bilirubin (13.5 μmol/l).

The cholesterol level decreased from 6.12 mmol/L in 2001 up to 4,5 mmol/l. And triglycerides became less than 1.3 mmol/l.

In 2010 cholesterol increased up to 7.51 mmol/l, triglycerides increased up to 2.2 mmol/l. Glucose level was 9.9 mmol/l. The metabolic syndrome was diagnosed. The diagnosis diabetes mellitus type 2 was diagnosed in next year. Antidiabetic medication was prescribed. The insulin therapy has been started since 2012.

Signs of hypothyroidism could not be stopped a long period of time. The dose

of L-thyroxine was increased up to 450 μg/day. However, it was not possible to achieve the compensation (table 2).

Secondary hypoparathyroidism was persist (2019): parathyroid hormone 82.2 pg/ml (normal range is 15-65 pg/ml), vitamin D content was low (11.8 ng/ml; a normal range more than 30 ng/ml).

Cardiac pathology was progressing. The cardiac diagnosis was made in 2019 is CAD: diffuse cardiosclerosis. Mitral valve insufficiency II stage. Tricuspidal valve insufficiency II stage. Arterial hypertension II grade. LVH. Heart failure NYHA II.

According to the ultrasound scan, moderate splenomegaly was revealed in 2019.

Discussion. According to the literature, idiopathic MDS with dysplasia is more common in adults over 60 years of age [2-6]. The patient was younger than 30 y.o when MDS with hypothyroid background with a complex genesis anemia developed. The leading syndrome in anemic syndrome was MDS (single-line). The single-line MDS prior to the updated of the classification, was interpreted as a refractory anemia (without the presence of blast cells in the blood and their increase in medulla, annular sideroblasts). When choosing a therapeutic regimen, for patients with a karyotype that is not evaluated for technical reasons, it is recommended to take into account the morphological variant of MDS according to the WHO classification, the number of blast cells in bone marrow and the dynamics of their changes, bone marrow cellularity and severity of cytopenias [2]. It corresponds to a low risk of transformation into acute leukemia in our clinical case and determines the survival rate of more than 9 years according to the evaluation criteria [2,7]. The disease's duration was more long-term.

A feature of the morphology of cells in the bone marrow in our case was, in contrast to idiopathic MDS, the absence of giant erythrokaryocytes, immature nuclei in the form of budding, trefoils, remnants of nuclei in erythrocytes, and hypersegmentation of neutrophils. Dysplasia of granulocytes was expressed, on the contrary, in a decrease in the number of segments in the cell nucleus. Anemia was predominantly hypochromic in blood; there were no nucleated precursors of

erythrocytes, pronounced signs of megaloblastoid cells.

From the literature data, hematological changes in subcompensated hypothyroidism are not only persistent, but are aggravated in the case of prolonged persistence of thyroid insufficiency; the negative dynamics of hemogram parameters and iron metabolism is expressed in a progressive decrease in iron stores in the body and an increase in the proportion of persons with micro- and normocytic anemia [8].

In general, iron deficiency and the presence of autoimmune thyroid pathology take a significant place in the structure of etiopathogenetic factors of anemia in hypothyroidism [9,10].

The hemoglobin level below 90 g/L for men and 80 g/L for women is an independent prognosis factor associated with a high risk of fatal cardiovascular complications. This rule works with any variant of MDS. Any hemoglobin level can be recommended as a threshold and definitive indication for transfusion support. The decision should be made based on the patient's symptoms and comorbidities [11]. Shortness of breath and chest pain due to an expressing of the hypoxia into the respiratory organs and heart in combination with hemoglobin level of 60 g/l required donor erythrocytes transfusion. The prescribing of corticosteroids in consequence to episodes of hemolysis with increased indirect bilirubin, slight paroxysmal splenomegaly, reticulocytosis, which may occur with MDS [2-5]. However, the patient categorically refused blood transfusions and hormone therapy by religious reasons.

In hypothyroidism, anemia most often occurs due to a decrease in the production of erythropoietin by the kidneys, as well as due to a deficiency of iron, folic acid, vitamin B12, which is caused by damage to the organs of the gastrointestinal tract. [2,12].

She had been receiving iron drugs in tablet and vitamin B12 several years before. In this case, on the contrary, ferritin and saturation of transferrin (ratio of serum iron and TIBC values) was low in contrast to typical increasing of serum ferritin increasing of serum ferritin due to MDS which needs therapy by Chelation deferasirox amid ineffective hematopoiesis and blood transfusion [13]. The patient

has background heart failure so there was grounds for parenteral iron drugs, which was performed in case of increasing hypoxia and decreasing hemoglobin below 80 g/l. This procedures were performed 2-3 times per year and gave a positive effect in combination with the correction of hypothyroidism, the appointment of diuretics.

The mechanism of the effect of iron supply on the thyroid gland is still unclear [14]. Iron deficiency anemia can disrupt thyroid metabolism due to decreased oxygen transport [15]; the control of the central nervous system over thyroid metabolism may change [16]; possible violation of binding of nuclear triiodothyronine. As a result, the serum concentration of thyroxine and triiodothyronine decreases, the peripheral transformation of one into another decreases, the metabolism of the latter decreases, and the amount of circulating thyroid-stimulating hormone of the pituitary gland may increase [17].

The administration of iron alone in FH provides a small therapeutic effect, which, however, can be potentiated by the addition of thyroid hormones [18].

Patients with low-risk MDS with endogenous erythropoietin less then 500 IU/ml and hemoglobin <100 g/l are recommended to treatment with erythropoiesis with stimulating erythropoietin drugs in order to increase hemoglobin parameters, reduce dependence from donor erythrocytes transfusions [19].

In our case, a decrease in the level of erythropoietin in the patient's blood was also due to a decrease in thyroid function.Thyroid hormones and especially hormone T4 activate the synthesis of erythropoietin to stimulate the proliferation of cells which sensitive to eritropoetin [20]. Therefore, erythropoietin (EPO) preparations were included into the therapy (Recormon, Hemax, Epobiocrin, etc.) in doses of 2-4 million UI subcutaneous 2-3 times a week, what is significantly less recommended due to social reasons. According to the literature the frequency of the response to erythropoietin monotherapy in case of MDS with attention to an endogenous EPO efficiency composed close to 60% with 2 doses of donor red blood cells and red germ Myelogram in more than 10% [21].

Patient’s erythroid germ was more than 10%. Although EPO was slightly lower than the recommended values for therapy, there was observed a positive and more

persistent effect compared to therapy with iron and vitamine B12 without EPO. As result the hemoglobin increased up to 100 g/l. Obviously, the development of MDS the refractive anemia with the background of hypothyroidism already presupposes the prescription of EPO, given its dependence on synthesis on thyroid hormones. EPO therapy was more effective. Against the background of substitution therapy, stimulation of erythropoiesis takes place due to an increase in the production of erythropoietin, but the actual hematological changes after reaching euthyroidism are not eliminated. To a certain extent, carbohydrate metabolism also depends on the level of thyroid hormones. The poor thyroid gland function can be a prerequisite for diabetes mellitus [22]. This case was observed at our patient. The diabetes mellitus type 2 was joined to clinic of hypothyroidism and anemic syndrome, which with some time transformed into insulin-dependent diabetes.

However, Raynaud's syndrome developed before the manifestation of diabetes mellitus. This indirectly indicates the connection between tissue involved in the pathological process and the development of the phenomena of fibrosis of the vascular wall, which does not exclude its foci in medulla. The patient refused trepanobiopsy. The literature contains data on the possible presence of fibrosis in MDS and refracted anemia [9]. The last one may be one of the reasons for the inhibition of erythropoiesis in a patient with hypothyroidism.

The anemia of chronic diseases (ACD) develops with diabetes mellitus without a state of iron deficiency against the background of nephropathy, heart failure. In our case, against the background of hematopoietic dysplasia with the presence of ineffective erythropoiesis, as well as frequent inflammatory processes, such an anemia pathogenesis is more likely. The literature data shows, there are data on an increase or normal level of transport serum iron in idiopathic MDS RA. The data of the study of the test with 59Fe indicate a reduced utilization and its excessive accumulation in organs and tissues [23]. However, this patient had iron deficiency, and needed the prescription of the iron supplements therapy. The pathogenetic features of the hypothyroidism state are more significant in changing the known pathogenesis of ACD and essential MDS.

According to the literature, the duration of the hemodepression phase ranges from several weeks to 20 years or more with periods of a favorable course (3-5 years). The final phase is a blast transformation by morphological and cytochemical characteristics, which occurs mainly as an acute non-lymphoblastic leukemia (myeloblastic, myelomonoblastic, erythroleukemia) [23].

In our case, in a patient with MDS on the background of severe hypothyroidism with a low risk of transformation into acute leukemia, the hemodepression phase lasts more than 20 years. However, there were complications from the cardiovascular, digestive, osteoarticular systems associated with a decompensated state, concomitant diabetes mellitus, which increased the risk of death. The literature has drawn attention to comorbid conditions in low-risk patients with MDS, which affect the prognosis, directly increasing the risk of death not associated with transformation into acute leukemia. And it is necessary to take into account the factors associated with both the disease and the patient himself, which significantly improves the predictive power of individual models, especially in low-risk groups [24].

Conclusion.

1. The pathogenesis of the MDS is multifactorial.
2. It is necessary to make a differential diagnosis of the MDS with dysmyelopoiesis on the background of somatic pathology including thyroid diseases.
3. The single-line MDS of the erythroid gerns is needed to make a differential diagnosis with megaloblastic anemia.

REFERENCE

1. Clinical Guidelines "Myelodysplastic Syndrome" Year of approval (frequency of revision) [Internet]; 2020 [updated 2020; cited 2020]. Available from: https://diseases.medelement.com › disease › миелодисп...

2. Kokhno A.V., et al. Myelodysplastic syndromes and aplastic anemia. In: Savchenko V.G. editor. Programmed treatment of blood diseases. 2012. p. 83–150.

3. Garcia-Manero G. Myelodysplastic syndromes: 2015 Update on diagnosis,

riskstratification and management. Am J Hematol. 2015; 90 (9): 831–41.

4. Malcovati L., et al. Diagnosis and treatment of primary myelodysplastic syndromes in adults: recommendations from the European LeukemiaNet. Blood. 2013; 122 (17): 2943–64.

5. Greenberg P.L., et al. NCCN Clinical Practice Guidelines in Oncology. Myelodysplastic syndromes. J Natl Compr Canc Netw. 2011 Jan; 9 (1): 30–56. doi: 10.6004/jnccn.2011.0005.

6. Goldberg S.L., et al. Incidence and clinical complications of myelodysplastic syndromes among United States Medicare beneficiaries. J Clin Oncol. 2010; 28 (17): 2847–52.

7. Malcovati L., et al. Impact of the degree of anemia on the outcome of patients with myelodysplastic syndrome and its integration into the WHO classification-based Prognostic Scoring System (WPSS). Haematologica. 2011; 96 (10): 1433–40.

8. Nekrasova T.A., Strongin L.G., Ledentsova O.V.. Hematological disorders in subclinical hypothyroidism and their dynamics during replacement therapy. Clinical medicine. 2013; (9): 29–33.

9. Goncharova O.A.. Associative relations between hypofunction of the thyroid gland of various origins and iron deficiency anemia. Reproductive Endocrinology. 2020; (51): 60–3. DOI: https://doi.org/10.18370/2309-4117.2020.51.60-62.

10. Gromova O.A., Rakhteenko A.V., Gromova M.A.. Iron-deficiency anemia. Therapeutic Reference. Guidelines for medical appointments: a modern guide for practitioners: Vol. 3, ed. by Martynov A.I. Moscow: Bionics Media; 2016.

11. Hoeks M.P.A., et al. Impact of red blood cell transfusion strategies in haemato-oncological patients: a systematic review and meta-analysis. Br J Haematol. 2017; 178 (1): 137–51.

12. Dorgalaleh A., et al. Effect of thyroid dysfunctions on blood cell count and red blood cell indice. Iran J Pediatr Hematol Oncol. 2013; 3 (2): 73–7.

13. Zeidan A.M., et al. Deferasirox therapy is associated with reduced mortality risk in a medicare population with myelodysplastic syndromes. J Comp Eff

Res Future Medicine Ltd. 2015; 4 (4): 327–40.

14. Ghiya R., Ahmad S. Severe Iron-Deficiency Anemia Leading to Hypothyroidism. J Endocr Soc. 2019; 3 (Suppl. 1): SUN-591. DOI: 10.1210/js.2019-SUN-591.

15. Ashart T.S., De Sanctis V., Yassin M., et al. Chronic anemia and thyroid function. Acta Biomed. 2017; (88.1): 119–27. DOI: 10.23750/abm.v88i1.6048.

16. Moreno-Fernandez J., Diaz-Castro J., Alferez M.J.M., Lopez-Aliaga I.. Iron Deficiency and Neuroendocrine Regulators of Basal Metabolism, Body Composition and Energy Expenditure in Rats. Nutrients. 2019; (11.3): E 631. DOI: 10.3390/nu11030631.

17. Shuxiang Li, Xin Gao, Yancai Wei, et al. The relationsheep between iron deficiency and thyroid function in Chinese women during early pregnancy. J Nutr Sci Vitaminiol. 2016; (62): 397–401. DOI: 10.3177/jnsv.62.397.

18. Omar S., Hadj Taeib S., Kanoun F., et al. Erythrocyte abnormalities in thyroid dysfunction. Tunis Med. 2010; 88 (11): 783–8.

19. Park S., et al. Clinical effectiveness and safety of erythropoietin-stimulating agents for the treatment of low- and intermediate-1-risk myelodysplastic syndrome: a systematic literature review. Br J Haematol. 2019; 184 (2): 134–60.

20. Эритропоэз | EUROLAB | Гематология

21. Park S., et al. Efficacy and safety of darbepoetin alpha in patients with myelodysplastic syndromes: a systematic review and metaanalysis. Br J Haematol Blackwell Publishing Ltd. 2016; 174 (5): 730–47.

22. Thyreo Support – Argo thyroid, thyroid hormones [Internet]; 2020 [updated 2020; cited 2020]. Available from: https://www.argo-shop.com.ua/article-7110.html.

23. LekMed.ru › bolezni/opyholi…sindromy.html

24. Porta M. Della, et al. Risk stratification based on both disease status and extra-hematologic comorbidities in patients with myelodysplastic syndrome. Haematologica. 2011; 96 (3): 441–9.