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КЛИНИЧЕСКИЙ СЛУЧАЙ ДИАГНОСТИКИ АПЛАСТИЧЕСКОЙ АНЕМИИ ПОСЛЕ ПЕРЕНЕСЕННОЙ ИНФЕКЦИИ COVID-19

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A Clinical Case of Aplastic Anemia After COVID-19 Infection

Резюме

Апластическая анемия — редкое заболевание системы крови, для которого характерно угнетение кроветворения во всех линиях гемопоэза, замещение кроветворной ткани на жировую и отсутствие других причин или заболеваний, которые могут подавлять гемопоэз. Частота заболеваемости составляет 2-3 случая на 1 млн населения в год в регионах Европы и Америки, показатели в 2-3 раза выше в Восточной Азии. Чаще заболевание начинается в возрастном промежутке от 10 до 25 лет и старше 60 лет. Этиология в 70-80 % случаях остается неизвестной. Частота приобретенных случаев заболевания преобладает над врожденными. Провоцирующими факторами могут быть химическое, физическое воздействие, лекарственные препараты, вирусные инфекции. В данной работе описан случай развития апластической анемии у пациентки после принесенной коронавирусной инфекции.

Ключевые слова: *апластическая анемия, панцитопения, аутоиммунные заболевания, COVID-19, коронавирусная инфекция*

Конфликт интересов

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Abstract

Aplastic anemia is a rare disease of the blood system characterized by suppression of hematopoiesis in all lines of hematopoiesis, replacement of hematopoietic tissue with fatty tissue and absence of other causes or diseases that can suppress hematopoiesis. The incidence is 2-3 cases per 1 million population per year in the regions of Europe and America, rates are 2-3 times higher in East Asia. The disease most often begins between the ages of 10 and 25 years and over 60 years. The etiology remains unknown in 70-80 % of cases. The frequency of acquired cases predominates over congenital cases. The triggering factors can be chemical, physical exposures, medications, and viral infections. This case report describes a case of a patient developing aplastic anemia, as a result of a coronavirus infection.

Key words: *aplastic anemia, pancytopenia, autoimmune disease, COVID-19, coronavirus infection*

Conflict of interest

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AA — aplastic anemia, MDS — myelodysplastic syndrome, CBC — complete blood count, ESR — erythrocyte sedimentation rate, CD — cluster of differentiation, COVID-19 — infection caused by the novel coronavirus SARS-CoV-2, FISH — fluorescent in situ hybridization, MCH — mean cell hemoglobin, MCHC — mean cell hemoglobin concentration, MCV — mean cell volume, NK — natural killer cells, RDW — red cell distribution width

Introduction

The etiology of aplastic anemia (AA) has not been fully studied yet. However, according to literature data, in most cases, viral infections can be the etiological factors (Epstein-Barr virus, hepatitis B and C, primary immunodeficiency, parvovirus); various drug products (cytostatics, antibacterial drugs, non-steroidal anti-inflammatory drugs, anticonvulsants); chemical compounds (benzene, pesticides, heavy metal salts, aromatic hydrocarbons); and ionizing radiation; the history of autoimmune diseases[1, 6, 7].

The pathogenesis is based on a significant activation of cytotoxic lymphocytes. Viral infection leads to the generation of viral proteins and excessive production of normal cellular proteins. They are captured by antigen-presenting cells; the latter form complexes with histocompatibility complex molecules and are naive T-lymphocytes, which destroy both infected and normal cells [3].

Clinical manifestations are due to bone marrow failure. They depend on the severity of pancytopenia, and are represented by anemic, hemorrhagic syndrome, infectious complications. In at least 80% of cases, the disease develops gradually, and the patients often see the doctor when they notice signs of hemorrhagic syndrome in the form of petechiae, ecchymoses, mucous membrane bleeding and hemorrhages in the conjunctiva, in combination with anemic syndrome and frequent infections. The disease may be asymptomatic and diagnosed by chance during a routine examination or a diagnostic search. In 15% of cases, the onset of aplastic anemia is acute, with severe hemorrhagic syndrome, fever, infectious and inflammatory complications [7].

To diagnose the disease, it is important to provide a thorough history-taking, identification of factors that could contribute have contributed to the disease and affected the patient for one to six months before the onset of the first symptoms: previous viral infections, taking medications, contact with chemical compounds, harmful industrial factors. The complete blood count (CBC) identifies pancytopenia with relatively normal lymphocyte counts. Normochromic anemia, low reticulocyte count. The myelogram shows the reduction of bone marrow cellularity and the absence of megakaryocytes according to bone marrow puncture. Low bone marrow cellularity in the trephine biopsy specimen. Hypocellular areas sometimes alternate with high cellularity areas [6, 7]. Lymphoid aggregation is sometimes detected in AA associated with an autoimmune process. In some cases, dyserythropoiesis, without dysplasia of other hematopoietic lineage [1].

Severe AA corresponds to:

1) bone marrow cellularity < 25%, or 25–50% with < 30% of residual hematopoietic cells.
2) the presence of two of three indicators: 1) neutrophils< 0.5×10^9/l; 2) platelets < 20×10^9/l;
3) reticulocytes < 20×10^9/l.

Super-severe AA meets the criteria for severe, with neutrophil count < 0.2×10^9/l. Non-severe AA is defined if criteria for severe and super-severe AA are not diagnosed [7].

Clinical case

Patient A.A., female 54 years old, nurse. Since 2005, the patient underwent regular endocrinologist's check-up with the diagnosis of "Autoimmune thyroiditis" and took levothyroxine sodium 75 mcg/day. The hormonal status was monitored, there were no complaints, the patient felt well. In June 2020, she complained of body temperature rise to 37.5°C, cough, headache for two days. The patient was treated on an outpatient basis: nasopharyngeal irrigation with furacilin solution for 5 days, ascorbic acid injections 100 mg per day IM 3 times daily, every other day. Considering the COVID-19 pandemic, the patient had a sputum PCR test, the result was positive. Chest computed tomography showed no pneumonia. CBC showed lymphocytosis — 48.35%, low white blood cell count of 2.0×10^9, neutrophils — 0.6×10^9, platelets — 138×10^9, which was regarded as a manifestation of a viral infection. There were no CBC changes since 2019 (Table 1). Further, the patient's condition deteriorated, weakness worsened, and hemorrhagic syndrome, manifested by bleeding gums and "bruising", developed. Progressive platelet count decrease from 138×10^9 to 50×10^9 was recorded in real-time for three months. In early October, the patient visited a hematologist and had a sternal puncture. Myelogram results: hypocellular bone marrow, megakaryocytes — 3%; myeloblasts — 1.0%; myelocytes — 10%; metamyelocytes — 5.5%; stab neutrophils — 4.0%; segmented neutrophils -18.5%; total neutrophils — 39%; eosinophils — 1%, basophils — 0.5%; lymphocytes — 28%; plasma cells — 4.5%; monocytes — 3.0%; basophilic normoblasts — 6.5%; polychromatophilic normoblasts — 11.5%; oxyphilic normoblasts — 6.0%; leuko-erythroblastic ratio — 3.2:1, low cellularity polymorphic punctate; blasts — 1.0%, monocytes — 3%. The myelogram results were regarded as secondary cytopenia associated with a viral infection.

The analysis of CBC changes from June to December 2020 showed a decrease in hemoglobin level from 132 to 68 g/l, red blood cell count — from 4.83×10^{12} to 1.83×10^{12}, platelets — from 243×10^9 to 13×10^9, white blood cell count — from 3.84×10^9 to 1.5×10^9, neutrophils — from 1.53×10^9 to 0.3×10^9; increased mean cell volume — from 79.1 to 103.2 fL, lymphocytosis — from 44.0% to 72.3%, ESR increased from 6 to 27 mm/h.

Table 1. *Dynamics of Patient A.A.'s general blood count from 2019 to 2021*

Complete blood count indicators	June 2019	June 2020	July 2020	September 2020	October 2020	November 2020	December 2020
Hematocrit	38,2 %	38,0 %	35,6 %	30,6 %	34,4 %	25,6 %	18,9 %
Hemoglobin	132 г/л	133 г/л	124 г/л	112 г/л	123 г/л	93 г/л	68 г/л
Erythrocytes	$4,83\times10^{12}$	$4,48\times10^{12}$	$4,22\times10^{12}$	$3,47\times10^{12}$	$3,74\times10^{12}$	$2,68\times10^{12}$	$1,83\times10^{12}$
MCV	79,1 фл	84,8 фл	84,4 фл	88,2 фл	92 фл	25,6 фл	103,2 фл
RDW	12,6 %	13,6 %	13,4 %	14,0 %	15,5 %	17,2 %	19,1 %
MCH	27,3 пг	29,7 пг	29,4 пг	32,3 пг	32,9 пг	34,6 пг	37,3 пг
MCHC	346 г/л	350 г/л	348 г/л	366 г/л	358 г/л	362 г/л	361 г/л
Platelets	234×10^9	138×10^9	148×10^9	57×10^9	50×109	19×10^9	13×10^9
Leukocytes	$3,84\times10^9$	$2,0\times10^9$	$2,56\times10^9$	$2,36\times10^9$	$2,2\times10^9$	$1,9\times10^9$	$1,5\times10^9$
Neutrophils	39,8 %	28,8 %	33 %	26 %	23,5 %	21,6 %	19,8 %
Lymphocytes	44,0 %	48,35 %	54 %	63 %	66,7 %	70,4 %	72,3 %
Monocytes	11,5 %	24 %	8 %	8 %	8.1 %	7,2 %	7,5 %
Eosinophils	4,4 %	0,8 %	1 %	2 %	1,2 %	0,5 %	0,3 %
Basophils	0,3 %	0,7 %	1 %	1 %	0,5 %	0,3 %	0,1 %
Neutrophils	$1,53\times10^9$	$0,6\times10^9$	$0,84\times10^9$	$0,61\times10^9$	$0,5\times10^9$	$0,4\times10^9$	$0,3\times10^9$
Lymphocytes	$1,69\times10^9$	1×10^9	$1,38\times10^9$	$0,49\times10^9$	$1,5\times10^9$	$1,3\times10^9$	$1,1\times10^9$
Monocytes	$0,44\times10^9$	$0,4\times10^9$	$0,20\times10^9$	$0,19\times10^9$	0.2×10^9	$0,1\times10^9$	$0,1\times10^9$
Eosinophils	$0,17\times10^9$	$0,01\times10^9$	$0,03\times10^9$	$0,05\times10^9$	$0,02\times10^9$	$0,01\times10^9$	$0,01\times10^9$
Basophils	$0,01\times10^9$	$0,01\times10^9$	$0,03\times10^9$	$0,02\times10^9$	$0,01\times10^9$	$0,01\times10^9$	$0,01\times10^9$
ESR	6 мм/ч	9 мм/ч	5 мм/ч	18 мм/ч	21 мм/ч	24 мм/ч	27 мм/ч

Note: MCV — mean corpuscular volume, RDW — red cell distribution width, MCH — mean corpuscular hemoglobin, MCHC — mean corpuscular hemoglobin concentration, ESR — erythrocyte sedimentation rate

Iron metabolism, tested in November 2020, demonstrated no major abnormalities of indicators: ferritin — 194.1μg/l; serum iron — 22.4 μmol/l.

Due to the deterioration of clinical and laboratory data in the beginning of December, the patient had a bone marrow trephine biopsy with the results showing a cellularity decrease and erythroid and myeloid lineage involvement. Medical report: bone marrow cellularity 30%, represented mainly by granulocytic lineage cells — promyelocytes, myelocytes, mature cells, single plasma cells, erythroblasts, adipose tissue 70%. Megakaryocytes — 2-0-1 in sight. CD20 — 10%, CD45 — 10%, myeloperoxidase — 35%; glycophorin A — 50%.

At the end of December, the patient was admitted to the hematology unit of the G.G. Kuvatov Republic Clinical Hospital. On admission, she complained of general weakness, rapid fatigability, dizziness, dyspnea on exertion, bruising, gum bleeding. The repeated sternal puncture demonstrated low bone marrow cellularity, the narrowing of the megakaryocytic lineage, an increased number of myelocytes, lymphocytes, and polychromatophilic normoblasts. Report: myelokaryocytes — 9.0 × 10^9/l; megakaryocytes -6 × 10^6 /l; undifferentiated blasts — 1.8%; neutrophilic myelocytes — 15.8%; neutrophilic metamyelocytes — 3.6%; stab neutrophils — 14.2% segmented neutrophils — 6.8%; eosinophils segmented — 1.0%; lymphocytes — 25.6%; monocytes — 3.6%; erythroblasts — 0.2%; basophilic normoblasts — 1.6%; polychromatophilic normoblasts — 20.2%; oxyphilic normoblasts — 4.2%; plasma cells — 1.4%; neutrophil maturation index — 0.92; leukoerythroblastic ratio — 2.7; erythrokaryocyte maturation index — 0.92 (Fig. 1)

Bone marrow immunophenotyping demonstrated no blast cells and immunophenotypic features of granulocytes. The predominance of cytotoxic cells (CD3+CD8+, NK) was noted in the lymphocytic link. T- and B- lymphocytes — without immunophenotypic features.

Immunophenotyping result: lymphocytes — 36.6%, CD3+ — 73.1%; CD4+ — 31%; CD8+ — 41%; CD57+ — 3%; CD77+ — 3%; CD56+ — 8%; CD19+ — 12.6%; CD11c — not found; CD103 — not found; CD10 — not found; CD34 — not found; CD117 — not found; CD1a — not found.

Abdominal ultrasound examination on 28.12.2020 demonstrated no abnormality.

Taking into account the previous coronavirus infection in June 2020, the three-lineage cytopenia present in the peripheral blood, and involvement of the erythroid and myeloid lineage based on the bone marrow biopsy, the following diagnosis was made: Myelodysplastic syndrome (MDS) with hypocellular bone marrow, unclassified, severe course. Complications: hemorrhagic syndrome (skin damage, bleeding gums). Aplastic anemia?

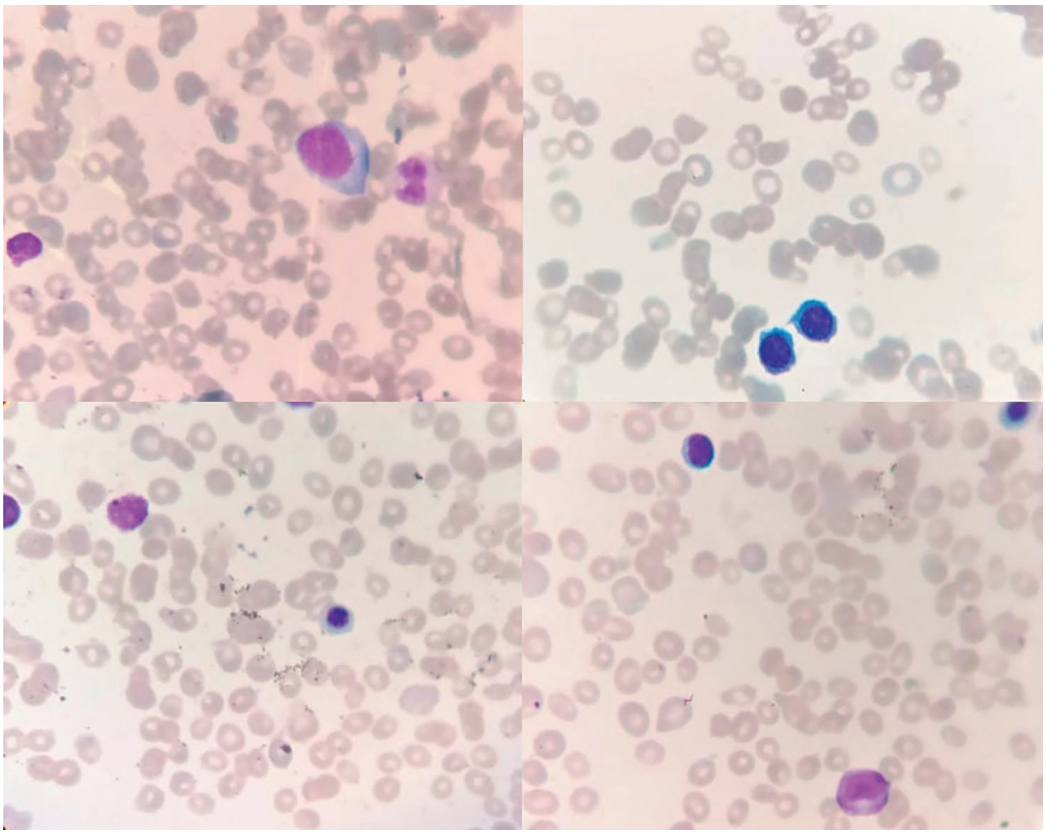

Figure 1. *Hypocellular bone marrow. Romanowsky-Giemsa staining*

To clarify the diagnosis, the bone marrow trephine biopsy block was sent to the National Medical Research Center for Hematology (Moscow) for review. The review enabled to exclude MDS and verify the diagnosis of aplastic anemia. Bone marrow biopsy specimen report: bone trabeculae with signs of resorption. Bone marrow cavities are wide, they contain hypocellular bone marrow (relative to the age norm). The granulocytic lineage is narrowed, rejuvenated. Erythroid lineage is moderately represented by clusters of normoblastic erythrokaryocytes. Megakaryocytes are single, small. Small lymphoid cells, mature plasma cells are interstitially scattered. Stroma with hemorrhages. A well-defined lymphoid accumulation of small cells, rather of reactive nature, is visualized intertrabecularly. To exclude the minimum signs of MDS, an immunohistochemical test was performed on paraffin block slices using antibodies to CD34 and CD42b. Upon reacting with antibodies to CD34, vessels and single positive cells are visualized. CD42b+ megakaryocytes are scarce, small and visually normal. Report: taking into account the immunohistochemical test, there is no convincing evidence of MDS in the examined material. The morphological pattern in the bone marrow characterizes the hypoplasia of hematopoietic tissue. Also, the material was sent for bone marrow cytogenetic FISH-test to the Republic Medical Genetic Center, which excluded the mutations characteristic of MDS**:** no translocation involving the MECOM/3q26 gene locus was identified; no deletion of 5p15.3 and 5q31.2 regions was identified; no deletion of regions 7q22.1–q22.2 and 7q31.2 was identified; no deletion of 20q12 and 20q13.12 was identified.

As a result, the final diagnosis was: Acquired severe aplastic anemia. Complications: hemorrhagic syndrome (skin damage, bleeding gums and nasal mucosa). Convalescent COVID-19 patient.

Erythrocyte mass and thrombocyte concentrate blood transfusion therapy was performed. The patient also received erythropoietin drugs subcutaneously every other day, etamsylate and folic acid. The patient was discharged with improvement at the end of December 2020 with the following blood counts: hemoglobin — 122 g/l, red blood cell count — 3.77 x 10^{12}, platelets — 18.0 × 10^9, leukocytes — 2.4 × 10^9, neutrophils — 0.6 × 10^9; mean cell volume — 92.3 fL, lymphocytes — 1.3 × 10^9, ESR — 22 mm/h

During the planned hospitalization to the hematology unit of the G.G. Kuvatov Republic Clinical Hospital at the end of January 2021, the patient complained of weakness and general malaise. The following decrease in blood counts was observed: hemoglobin — 76 g/l, red blood cell — 2.33 × 10^{12}, mean cell volume (MCV) — 94 fL, platelets — 12 × 10^9, white blood cell — 1.6 × 10^9, neutrophils — 0.4 × 10^9; lymphocytes — 1.1 × 10^9. The patient was administered erythropoietin subcutaneously every other day, received erythrocyte mass and thrombocyte concentrate transfusion therapy and ethamsylate and folic acid. She had a telemedicine consultation with the National Medical Research Center for Hematology in order to clarify the diagnosis and determine the treatment approach. The **report** was as follows: based on the examination performed, the patient is diagnosed with acquired aplastic anemia; recommendation: combined immunosuppressive therapy.

At the beginning of March 2021, the patient was admitted to the hematology unit of the G.G. Kuvatov Republic Clinical Hospital for combined immunosuppressive therapy. During admission, the blood counts were as follows: hemoglobin — 88 g/l, red blood cell count — 2.69 × 10^{12}/l, mean cell volume (MCV) — 93.8 fL, platelets — 16 × 10^9/l, white blood cell count — 2.3 × 10^9/l, neutrophils — 1.1 × 10^9/ l, lymphocytes — 1.2 × 10^9/l.

After a course of anti-thymocyte globulin and blood component transfusion, the patient was discharged with improvement. In CBC, the level of hemoglobin increased to 121 g/l, red blood cell count — to 3.77 × 10^{12}/l, white blood cell count — to 3.8 × 10^9/l, neutrophils — 1.4 × 10^9/l, platelets — 24 × 10^9/l, mean cell volume — 92.7 fL, lymphocytes — 1.0 × 10^9/l. Upon discharge, the patient was recommended to start taking Cyclosporin A, in accordance with National Guidelines. [9].

In mid-July 2021, a follow-up examination by a hematologist at the G.G. Kuvatov Republic Clinical Hospital showed an improvement of the general condition, and the absence of hemorrhagic syndrome. The changes included a slight decrease in hemoglobin to 115 g/l, leukocytes — to 2.5 × 10^9/l, neutrophils — to 0.6 × 10^9/l. Red blood cell count was 3.13 × 10^{12}/l, platelets — 46 × 10^9/l, lymphocytes — 1.6 × 10^9/l. According to the test results, the absence of hemorrhagic syndrome and blood transfusion dependence, the patient had partial clinical and hematological remission.

Discussion

AA is an orphan disease that occurs in most regions of Europe and America, with an incidence of 2–3 cases per 1 million population a year [7]. Because AA is a rare disease, it was not immediately suspected in this patient.

The hematological changes associated with COVID-19 were regarded as secondary cytopenia associated with a viral infection. In view of the pandemic, bone marrow trephine biopsy was not performed at that time, which made the timely diagnosis of AA difficult.

According to the literature data, some patients with COVID-19 may develop leukopenia and thrombocyto-

penia [8]. However, according to CBC in the presented clinical case, pancytopenia gradually progressed, lymphopenia was not observed, moderate manifestations of hemorrhagic syndrome appeared, which indicated the development of a hematological disease. Therefore, the diagnosis of AA was made after six months of observation, after a sternal puncture and trephine biopsy.

Taking into account the satisfactory condition and the absence of changes in the patient's blood tests before coronavirus disease, it can be assumed that COVID-19 was a trigger to the onset of AA in this case.

Conclusion

Therefore, AA must be differentiated from other conditions that lead to pancytopenia. The clinical case is given as an example that confirms the literature data on the impact of coronavirus on the development and course of autoimmune diseases. [2, 3, 4, 5]. We believe that in this case, the development of the autoimmune disease AA could have been triggered by the SARS-CoV-2 virus. The follow-up observation of patients in the post-COVID period will yield new information on the impact of COVID-19 on the hematopoietic system and the development and course of hematological diseases.

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