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ANEMIC SYNDROME IN PATIENTS WITH ACUTE LEUKEMIA

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Abstract: This article describes the opinions of our country and foreign scientists about the treatment of anemia syndrome in patients with acute leukemia.
Keywords: Anemic syndrome, erythropoiesis-stimulating drugs, frequent blood loss, indirect effects of the tumor, bone marrow damage.

INTRODUCTION.

Anemic syndrome is a common complication in patients with acute leukemia. This condition is characterized by a decrease in the level of red blood cells (erythropenia) and/or hemoglobin in the blood (hemoglobinopenia), which leads to a decrease in the blood's ability to carry oxygen throughout the body. Anemia can worsen fatigue, weakness, shortness of breath, and reduce the quality of life of patients.

With acute leukemia, anemia can develop due to several factors, such as:

1. Indirect effects of the tumor: Leukemia leads to a significant increase in the number of immature white blood cells in the blood, which leads to competition for nutrients and space in the bone marrow, which can suppress the normal erythroid lineage.
2. Bone marrow damage: Tumor cells can infiltrate and replace normal bone marrow cells, resulting in disruption of the formation of hematopoietic cells.
3. Frequent blood loss: Leukemia can lead to bleeding due to platelet dysfunction or increased bleeding, which can worsen anemia.

The following approaches may be used to treat anemia in patients with acute leukemia:

- Blood transfusions: Transfusion of red blood cells or platelets to improve hemoglobin and/or platelet levels in the blood.
- Erythropoiesis-stimulating drugs: These can stimulate the production of red blood cells in the bone marrow.
- Treatment of the underlying disease: Effective treatment of leukemia can help restore normal bone marrow function and improve anemia.

It is important to note that the treatment of anemic syndrome in patients with acute leukemia requires an individual approach and supervision by qualified specialists.

The treatment of anemia syndrome in patients with acute leukemia involves a comprehensive approach that addresses the underlying causes of anemia and aims to improve the patient's overall well-being. Anemia in acute leukemia can occur due to decreased production of red blood cells, increased destruction of red blood cells, or blood loss, and its management is essential for the overall care of the patient.

Blood Transfusions

In the acute setting, blood transfusions are commonly used to manage anemia. Red blood cell transfusions help to raise the hemoglobin levels, improve oxygen delivery to tissues, and alleviate symptoms associated with anemia such as fatigue and shortness of breath.

Erythropoiesis-Stimulating Agents

Erythropoiesis-stimulating agents (ESAs) such as erythropoietin-stimulating agents may be considered to stimulate the production of red blood cells in the bone marrow. These agents can be

used to reduce the need for blood transfusions and may be beneficial in certain cases of anemia associated with acute leukemia.

Addressing Underlying Leukemia

Effective treatment of the underlying leukemia is crucial for improving anemia. Chemotherapy, targeted therapy, and in some cases, stem cell transplantation are used to address the leukemia itself. By reducing the leukemia burden and promoting bone marrow recovery, these treatments can indirectly improve anemia.

Supportive Care

Patients with acute leukemia require comprehensive supportive care, including nutritional support, management of infections, and close monitoring for potential side effects of intensive therapy. Nutritional support plays an important role in managing anemia and providing essential nutrients for the body's recovery.

Bone Marrow Stimulants

In some cases, medications that stimulate the production of blood cells in the bone marrow, such as granulocyte colony-stimulating factors (G-CSF), may be used to support the recovery of blood cell counts, including red blood cells.

Personalized Approach

It's important to highlight that the management of anemia in patients with acute leukemia should be individualized and tailored to each patient's specific condition, age, overall health, and response to treatment. It requires close collaboration among hematologists, oncologists, and other healthcare professionals to ensure the most effective and tailored care.

Ongoing Monitoring and Support

Regular monitoring of blood counts, symptom management, and treatment adjustment based on the patient's response are essential components of the care of patients with acute leukemia and anemia. This requires a multidisciplinary approach that considers the patient's overall well-being and quality of life.

In summary, the treatment of anemia in patients with acute leukemia is a multifaceted process that involves addressing the underlying leukemia, managing symptoms, and providing supportive care to improve the patient's overall health and well-being.

Anemia syndrome in patients with acute leukemia can have a profound impact on vital organs, influencing their function and overall well-being. Anemia, characterized by a decrease in the number of red blood cells and hemoglobin levels, leads to reduced oxygen delivery to tissues, resulting in a wide range of physiological effects on vital organs.

Cardiovascular System

In patients with acute leukemia and anemia, the cardiovascular system faces significant challenges. Decreased oxygen-carrying capacity in the blood can lead to reduced oxygen delivery to the heart, resulting in increased cardiac workload. This could potentially exacerbate existing cardiac conditions and lead to symptoms such as palpitations, chest pain, and fatigue.

Brain and Central Nervous System

Reduced oxygen supply to the brain due to anemia can result in symptoms such as dizziness, cognitive impairment, and decreased concentration. Severe anemia may lead to hypoxia, potentially increasing the risk of neurologic complications such as confusion, disorientation, and in severe cases, seizures.

Kidneys

Anemia can impact renal function, particularly in individuals with compromised kidney function. Reduced oxygen delivery to the kidneys may lead to impaired renal perfusion and function. Chronic anemia can contribute to the progression of existing renal disease and may exacerbate complications associated with kidney dysfunction.

Muscles and Exercise Tolerance

Decreased oxygen delivery to skeletal muscles can lead to reduced exercise tolerance, fatigue, and overall decreased physical endurance. Patients with acute leukemia and anemia may experience increased weakness, reduced muscle strength, and decreased capacity for physical activity.

Immune System

Anemia can potentially compromise the immune system, making patients more susceptible to

infections and impairing their ability to combat pathogens. This is particularly significant in the context of acute leukemia, where immune function may already be compromised due to the underlying disease and its treatment.

Overall Quality of Life

Beyond its impact on vital organs, anemia can significantly affect the overall quality of life of patients with acute leukemia. Patients may experience reduced energy levels, increased fatigue, and diminished ability to engage in daily activities, impacting their emotional well-being and social interactions.

In patients with acute leukemia, the impact of anemia on vital organs underscores the importance of prompt and effective management. Addressing anemia not only involves improving hemoglobin levels but also requires a multidisciplinary approach aimed at optimizing the patient's overall health, restoring organ function, and enhancing their quality of life.

Given the complex interplay of anemia and acute leukemia, individualized treatment plans tailored to each patient's specific medical condition, overall health, and unique needs are crucial in mitigating the impact of anemia syndrome on vital organs and enhancing patient outcomes.

Acute leukemia is a clonal (oncological) disease that primarily arises in the bone marrow as a result of a mutation in a blood stem cell. The consequence of the mutation is the loss of the descendants of the mutated cell's ability to differentiate into mature blood cells. The morphological substrate of acute leukemia is blast cells.

As with most other tumor diseases, it is impossible to identify a specific etiological factor for acute leukemia.

Acute leukemias are divided into a number of types, among which the most important are acute lymphoblastic and acute myeloblastic leukemias.

Acute leukemia never becomes chronic, and chronic leukemia never worsens. Thus, the terms "acute" and "chronic" are used only for convenience; the meaning of these terms in hematology differs from the meaning in other medical disciplines. The only exception is chronic myeloblastic leukemia, the acute or terminal phase of which is characterized by the development of blast crisis - the appearance of 30-90% of blast cells in the blood and bone marrow, that is, the development of acute myeloblastic (or lymphoblastic) leukemia.

Kinds

Acute myeloblastic leukemia (AML)

Acute poorly differentiated leukemia

AML without maturation

AML with maturation

Acute promyeloblastic leukemia

Acute myelomonoblastic leukemia

Acute monoblastic leukemia

Acute erythromyelosis

Acute megakaryoblastic leukemia

Acute lymphoblastic leukemia (ALL)

Pre-pre-B-ALL

Pre-B-ALL

B-ALL

T-ALL

Epidemiology

Every year, 35 new cases of acute leukemia are registered per 1 million population. The structure of morbidity largely depends on age. ALL often develops in childhood and after 40 years. The incidence of AML is the same in all age groups. Men and women get sick with the same frequency.

Pathogenesis

The pathogenesis of acute leukemia is based on a mutation of a blood stem cell, which entails an almost complete loss of the ability of the descendants of the mutated cell to mature. The mutant clone is autonomous from any regulatory influences of the body and quite quickly displaces normal hematopoietic cells, replacing all hematopoiesis.

The degree of malignancy of tumor cells in acute leukemia increases over time (as for other groups

of tumors, the law of tumor progression is valid for acute leukemia). Since tumor cells in acute leukemia in most cases initially have a pronounced maturation defect, greater malignancy is often manifested by the appearance of extramedullary foci of hematopoiesis, an increase in proliferative activity, and the development of resistance to therapy.

The consequence of a stem cell mutation is the development in the bone marrow of a clone of cells that have lost the ability to mature. The neoplastic clone displaces normal hematopoietic cells, which leads to the development of a deficiency of mature cells in the peripheral blood. A decrease in the number or complete absence of mature peripheral blood cells causes a loss of the corresponding functions of the peripheral blood, which entails the development of clinical symptoms of the disease.

Morphology

Various forms of acute leukemia have stereotypical morphological manifestations: leukemic infiltration of the bone marrow in the form of focal and diffuse infiltrates of cells with large light nuclei containing several nucleoli. The size and outline of the nuclei, as well as the width of the plasma rim, can vary. Blasts make up 10-20% of brain cells. The cytogenetic affiliation of blasts, as a rule, can only be revealed using special research methods - cytochemical and immunohistochemical. Reactions to peroxidase, staining for lipids with Sudan black, PIC reaction, histoenzyme-chemical actions to detect nonspecific esterase, chloroacetate esterase, and acid phosphatase are used. Immunohistochemically it is possible to determine markers of B-, T-lymphocytes, cells of the myeloid and monocyte series.

In the peripheral blood and bone marrow, the phenomenon of “leukemic failure” (“hiatus leucemicus”) is described, developing due to the presence of only blast and differentiated cells and the absence of intermediate forms.

In the bone marrow tissue, normal hematopoietic cells are displaced by tumor cells, reticular fibers are thinned and reabsorbed, and myelofibrosis often develops. With cytostatic therapy, the bone marrow is devastated with the death of blast forms, the number of fat cells increases and connective tissue grows.

Leukemic infiltrates in the form of diffuse or focal accumulations are found in the lymph nodes, spleen and liver. This leads to an increase in the size of these organs. The liver is characterized by the development of fatty degeneration. Leukemic infiltration of the mucous membranes of the oral cavity and tonsil tissue is possible.

Clinical manifestations

Clinical manifestations are the same for all variants of acute leukemia and can be quite polymorphic. The onset of the disease can be sudden or gradual. There is no characteristic onset or any specific clinical signs for them. Only a thorough analysis of the clinical picture makes it possible to recognize a more serious disease hidden under the guise of a “banal” disease.

A combination of bone marrow failure syndromes and signs of a specific lesion is characteristic.

Due to leukemic infiltration of the mucous membranes of the oral cavity and tonsil tissue, necrotizing gingivitis and tonsillitis (necrotizing tonsillitis) appear. Sometimes a secondary infection occurs and sepsis develops, leading to death.

The severity of the patient's condition may be due to severe intoxication, hemorrhagic syndrome, respiratory failure (due to compression of the respiratory tract by enlarged intrathoracic lymph nodes).

The use of active cytostatic therapy influenced the course of acute leukemia, that is, it led to drug-induced pathomorphosis. In this regard, the following clinical stages of the disease are currently distinguished:

first attack

remission (complete or incomplete),

relapse (first, repeated).

Bone marrow failure

It manifests itself in the form of infectious complications, disseminated intravascular coagulation syndrome, hemorrhagic and anemic syndromes.

The development of infectious complications occurs due to immunodeficiency caused by dysfunction of leukocytes. Most often, infectious complications are of bacterial origin; fungal and

viral infections are less common. A sore throat, gingivitis, stomatitis, osteomyelitis of the maxillofacial area, pneumonia, bronchitis, abscesses, phlegmon, and sepsis may develop.
Hemorrhagic syndrome in acute leukemia is caused by thrombocytopenia, damage to the liver and vascular walls. It manifests itself as hemorrhagic diathesis of the petechial-spot type. “Bruises” and small petechiae appear on the skin and mucous membranes. The appearance of hemorrhages is easily provoked by the most insignificant influences - friction of clothing, slight bruises. Nosebleeds, bleeding from the gums, metrorrhagia, and bleeding from the urinary tract may occur. Hemorrhagic syndrome can lead to very dangerous complications - brain hemorrhages and gastrointestinal bleeding.
Anemic syndrome manifests itself in the form of pallor, shortness of breath, rapid heartbeat, and drowsiness.
DIC syndrome most often occurs in promyelocytic leukemia.
Specific lesion
Signs of intoxication are noted: weight loss, fever, weakness, sweating, loss of appetite.
Infiltration of the gums by leukemic cells may be observed, while the gums are hyperplastic, hang over the teeth, and are hyperemic.
Proliferative syndrome can manifest itself as an increase in the size of the lymph nodes (lymphadenopathy), spleen, and liver. In some cases, leukemids appear on the skin - formations of soft or dense consistency that rise above the surface of the skin. Their color can match the color of the skin or be light brown, yellow, or pink.
Damage to the central nervous system (neuroleukemia) occurs especially often in ALL and significantly worsens the prognosis. Metastasis of leukemia cells occurs into the membranes of the brain and spinal cord or into the substance of the brain. Clinically, manifestations of varying severity are possible - from headaches to severe focal lesions.
The manifestation of acute leukemia can be sudden or subtle.

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