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STATE OF HEMOPOIESIS IN CHILDREN WITH APLASTIC ANEMIA

Introduction

Aplastic anemia (AA) is a rare but serious blood disorder characterised by the failure of the bone marrow to produce sufficient blood cells. This condition leads to pancytopenia, a reduction in red blood cells (RBCs), white blood cells (WBCs), and platelets and is associated with increase in the risk of haemorrhage, infection, organ dysfunction and death [1,2]. It is estimated that the incidence of Aplastic Anemia (AA) is 0.7–4.1 cases per million people worldwide, with the prevalence between men and women being approximately equal (1:1). The incidence rate of aplastic anemia in Asia is 2–3 times higher than it is in the West; the incidence in the United States is 300–900 cases per year. The disease most commonly affects adults aged 15–25 and over the age of 60, but it can be observed in all age groups [3,4]. The development of AA is associated with multifactorial pathophysiological mechanisms. The primary disorder is an intrinsic defect in multi-potent hematopoietic stem cells, which is either manifested or exacerbated by various etiological factors: physical (ionising radiation, high-frequency currents, vibrations), chemical (medications, benzene derivatives, heavy metal salts, pesticides), and infectious (cytomegalovirus, herpes, hepatitis, Epstein-Barr virus, parvovirus B19, measles, mumps; bacteria; fungi) [1,2,5].

AA can be either acquired or congenital. In 80% of cases, AA is an acquired disease, which includes idiopathic and secondary forms, while in 20% of cases, AA develops due to inherited genetic disorders. Inherited forms of the disease include Diamond-Blackfan anemia, Fanconi anemia, Estren-Dameshek anemia, reticular dysgenesis, and congenital dyskeratosis. The clinical picture of AA is characterised by hemorrhagic and anemic syndromes, as well as infectious complications. The definitive diagnosis of AA is based on the histological picture of the bone marrow. Treatment includes the following approaches: allogeneic hematopoietic stem cell transplantation (HSCT), immunosuppressive therapy, and supportive care. Understanding the initial clinical and laboratory manifestations is crucial for the primary diagnosis of AA [1,2,3].

Goal

To assess the state of hemopoiesis in children with aplastic anemia.

Materials and methods of research

A retrospective and prospective analysis of the incidence of aplastic anemia (AA) in children of the Gomel region from 2014 to 2024 was conducted. A total of 18 patients aged 3 to 24 years were examined, with a median age of 11 years. The study was conducted at the “Republican Scientific and Practical Center” in Gomel. The material included outpatient records and medical histories, where clinical data, peripheral blood parameters (including erythrocyte indices: MCV – mean corpuscular volume, MCH – mean corpuscular haemoglobin, MCHC – mean corpuscular haemoglobin concentration), and bone marrow data were analysed. The diagnosis of AA was confirmed based on pancytopenia, bone marrow aspirate, and trephine biopsy. Bone marrow aspirate analysis included cellularity, neutrophil content (%), lymphocyte content (%), and megakaryocyte count (per 1 μl). The level of CD34+ cells (a stem cell marker) in the bone marrow was also determined. The severity of AA was classified according to Camitta

B. criteria: non-severe-granulocytopenia >0.5×10^9/L; severe-granulocytopenia <0.5×10^9/L, thrombocytopenia <20×10^9/L; very severe-granulocyte level <0.2×10^9/L [4]. The severity of AA was determined based on at least three peripheral blood tests at the time of diagnosis before treatment initiation. Given the frequent association of AA with paroxysmal nocturnal hemoglobinuria (PNH), all patients underwent immunophenotyping to detect PNH clones based on the expression of CD55 and CD59 on blood cell surfaces. The study was conducted with informed consent from medical staff, adhering to ethical standards and confidentiality. Statistical analysis was performed using frequency characteristics and non-parametric methods, with the calculation of the median (Me), upper and lower quartiles, using the Statistika 6 program.

The results of the research and their discussion

Among the examined patients with AA (n=18), the acquired form predominated – 82.4% (n=15), while congenital AA accounted for 17.6% (n=3). Males predominated among children with AA – 76.5% (n=13), with a ratio of 3.3:1. Girls accounted for 23.5% (n=5). The hemorrhagic syndrome was predominant in the clinic – 58.8% (n=10). Anemic syndrome was observed in 52.9% (n=10) of patients. In 4 children (23.5%), the disease debuted with various infectious complications. Analysis of peripheral blood laboratory parameters showed that haemoglobin (HGB) levels ranged from 52 g/L to 118 g/L, with a median of 88.35 [79.1;103] g/L, corresponding to moderate anemia. Red blood cells (RBC) ranged from 1.94–3.98×10^12/L, with a median of 2.75 [2.5;3.6]×10^12/L. Erythrocyte indices were characterised as follows: MCH – 21–35 pg, median 33 [31.6;34] pg; MCV – 66.5–104 fl, median 92.5 [87.7;95.1] fl. Thus, the anemia was normochromic and normocytic. Reticulocyte levels in AA typically correspond to an aregenerative type of anemia. However, in our study, reticulocyte levels ranged from 0.7–4.7‰, with a median of 1.57 [1.4;3]‰, indicating a regenerative nature of anemia. Platelet (PLT) levels ranged from 1–130×10^9/L, with a median of 48.1 [17;69.8]×10^9/L. Leukocyte levels ranged from 1.5–12×10^9/L, with a median of 3.5 [3.3;4.7]×10^9/L. Immunophenotyping results showed the presence of PNH clones in 2 patients.

The main diagnostic criterion for AA is fatty degeneration of the bone marrow based on trep-hine biopsy. Myelogram examination is mandatory for primary diagnosis of AA. However, it should be noted that bone marrow aspirate parameters may be within normal limits, which can be explained by the presence of areas with normal hemopoiesis in the bone marrow. Therefore, bone marrow aspiration from at least two sites is recommended. Stem cell level determination is also recommended. Myelogram alone cannot definitively diagnose AA, but it can reveal signs of aplasia, such as reduced neutrophil content (<50%), lymphocytosis, and complete absence of megakaryocytes. In our study, all patients exhibited hypocellular bone marrow. Neutropenia ranged from 12–40% (median NEU – 24.7 [10;44]%), lymphocytosis – 45–78% (median LYM – 61.75 [28;72.2]%). Megakaryocyte counts ranged from complete absence to 10 per µl. CD34+ cell levels in the bone marrow ranged from 0.1–2.85%, with a median of 0.5 [0.11;0.9]%. All patients underwent histological examination of the bone marrow, revealing fatty degeneration with predominant adipose tissue (>95%).

Conclusion

The results of the analysis of AA incidence in children led to the following conclusions.

Acquired forms of AA predominated (82.4%). The median age of children with acquired AA was 12 years, while congenital AA was 10 years. Boys were more frequently affected, with a ratio of 3.3:1. Signs of aplasia based on myelogram included neutropenia, lymphocytosis, and reduced megakaryocyte counts. Stem cell levels in the bone marrow were an important marker of aplasia. The main diagnostic criterion was extensive replacement of bone marrow by adipose tissue. Thus, myelogram examination during primary diagnosis of AA helps exclude infiltrative bone marrow lesions (e.g., by blast cells) and suggests AA, prompting the need for trephine biopsy.

LITERATURE

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PITTSBURGH SLEEP QUALITY INDEX (PSQI-P) SCALE IN HOSPITALIZED PATIENTS

Introduction

Sleep-related problems and poor sleep quality are significant public health issues with high sentence searching prevalence worldwide [1, 2]. Poor sleep quality may be due to physical illness, side effects of medications, psychiatric disorders (such as depression, anxiety disorder, insomnia, schizophrenia), or use of psychoactive substances. Poor sleep quality and sleep disturbances can adversely impact quality of life by reducing productivity at the workplace and by making social activities less enjoyable [3, 4]. Thus, assessment of sleep quality is important for a wide range of clinical and behavioural research and for practitioners of medicine and psychology. Standardized questionnaires provide comprehensive assessments of sleep quality, but few such questionnaires are available. The Pittsburgh Sleep Quality Index (PSQI) [2], a standardized self-administered questionnaire, was introduced in 1989 and has gained widespread acceptances a useful instrument for the assessment of sleep problems that may be associated with anxiety, stress, depression, and schizophrenia. Its reliability and validity have been demonstrated for patients with psychiatric and sleep disorders and for patients with other somatic diseases. ThePSQI consists of seven clinically derived components that assess sleep difficulty, and the sum of these component scores yields a global score of subjective sleep quality.

Goal

This study aimed to evaluate the Pittsburgh Sleep Quality Index (PSQI-P) among the hospitalized patients in therapeutic departments.

Material and methods of research

This is cross sectional study of patients about sleep quality, conducted in the different therapeutic departments in Gomel city clinical hospital N3, Belarus. This study was conducted in a month January 2025.The mean onset of age starts from 25 years to 80 years. A total number of patients included in the study were 30 members with sleep cycle. The ratio of male to female was 1.34;1 of all Psqi scale showing female predominance. PSQI is a self-rating questionnaire with 19 questions and seven component scores: sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction. It was originally designed as a simple and valid instrument for use in diverse