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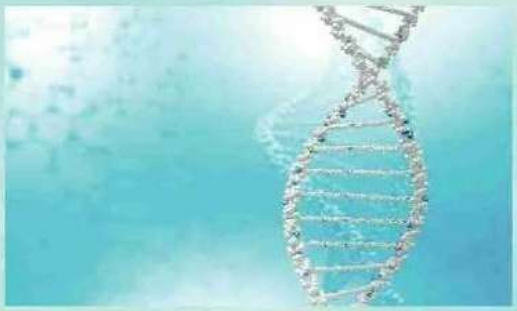

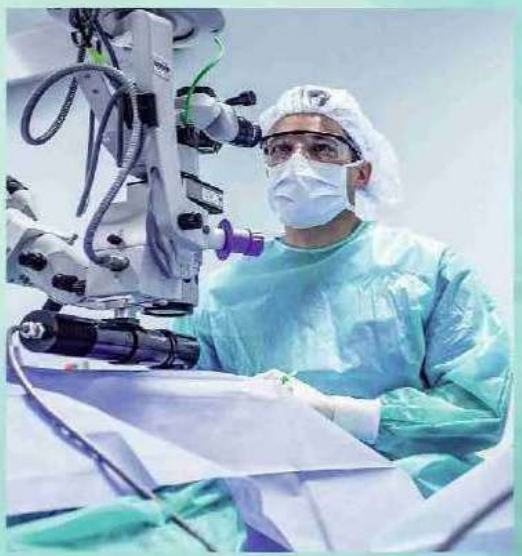

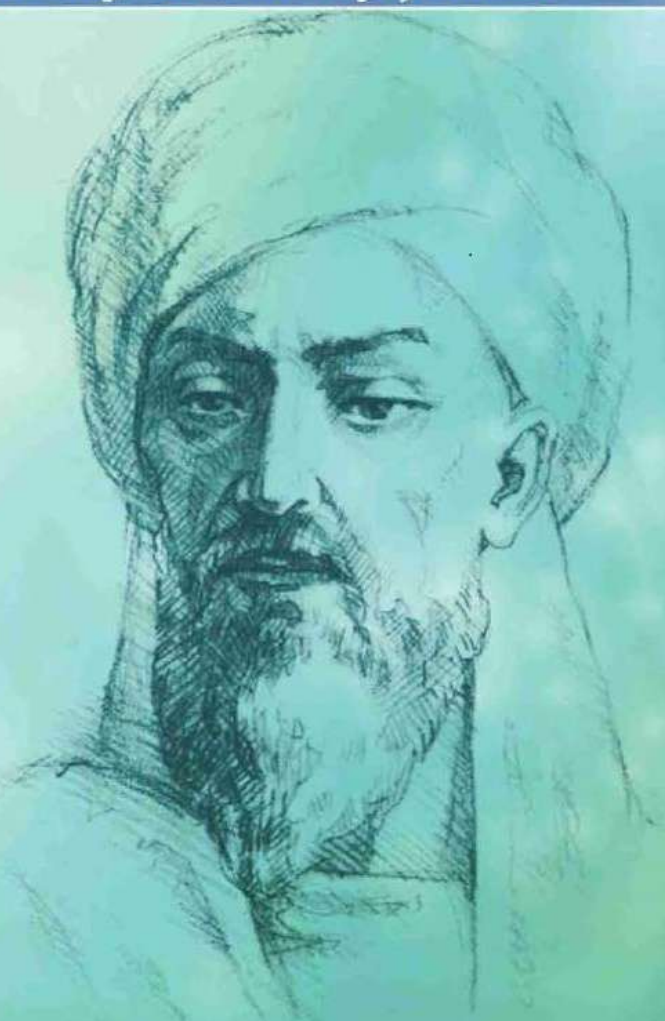

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APLASTIC ANEMIA ASSOCIATED WITH HERPES SIMPLEX VIRUS-1

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✓ ***Resume***

Aplastic anemia is a rare disease characterized by peripheral pantsitopenia against the background of hypocellular bone marrow damage. In most cases, it has an idiopathic origin. However, some drugs and toxins are associated with exposure, autoimmune processes and viral infections. This study shows that in anemia caused by herpes virus-1 infection, we can observe the elimination of anemia from the use of anti-viral therapy. But the above situation is considered not yet fully justified.

Key words: bone marrow, herpesviruses, anemia.

ОДДИЙ ГЕРПЕС ВИРУСИ-1 БИЛАН БОҒЛИҚ АПЛАСТИК АНЕМИЯ

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✓ ***Резюме***

Апластик анемия - бу гипоцеллюлар суяк илиги шикастланиши фонида периферик панцитопения билан тавсифланган кам учрайдиган касаллик. Кўпгина ҳолларда, у идиопатик келиб чиқишга эга. Бироқ, баъзи дорилар ва токсинлар таъсир қилиш, аутоиммун жараёнлар ва вирусли инфекциялар билан боғлиқ. Ушбу тадқиқот шуни кўрсатадики оддий герпес вируси-1 инфекциясида вирусга қарши терапия қўлланилгандан кейин касалликни клиник кўринишларнинг тез яхшиланишини ҳисобга олган ҳолда, муҳим, аммо ҳали тан олинмаган ҳолат бўлган, апластик анемия оддий герпес вируси ривожланиши ўртасидаги боғлиқликни кўрсатди.

Калит сўзлар: суяк илиги, герпес вируслари, анемия.

АПЛАСТИЧЕСКАЯ АНЕМИЯ, СВЯЗАННАЯ С ВИРУСОМ ПРОСТОГО ГЕРПЕСА-1

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✓ ***Резюме***

Апластическая анемия - редкое заболевание, характеризующееся периферической панцитопенией на фоне гипоцеллюлярного поражения костного мозга. В большинстве случаев она имеет идиопатическое происхождение. Однако некоторые лекарственные препараты и токсины связаны с воздействием, аутоиммунными процессами и вирусными инфекциями. Это исследование показывает, что при анемии, вызванной инфекцией, вызванной вирусом герпеса-1, мы можем наблюдать устранение анемии при применении противовирусной терапии. Но вышеуказанная ситуация считается еще не полностью оправданной.

Ключевые слова: костный мозг, вирусы герпеса, анемия.

Relevance

Aplastic anemia is a condition characterized by pantsitopenia against a background of hypocellular bone marrow. Based on data from the population of Europe and Israel, in the international assessment of aplastic anemia and agranulocytosis, the disease corresponds to 2,2 cases per 1000,000 people per year [1]. A number of conditions have been associated with aplastic anemia, including drug exposure, autoimmune diseases, and viral infections. Of the viruses associated with aplastic anemia, the most reliable are hepatitis viruses, Parvovirus and Human Immunodeficiency Virus (HIV) [3-5]. However, no published cases of herpes simplex virus 1 (HSV-1) have been found in modern medical literature.Having been in the last phase of kidney failure in hemodialysis for several years, the 56-year-old African American woman had hypertension, diabetes, odinophagia for three days, dysphagia, fever (38,5) and a bloody sputum complaint. During the medical examination, mucositis, swelling and bloody wounds were detected in the patient's oral cavity. On initial examination, pantocytopenia: hemoglobin (Hg) 8.9 g/dl; leukocytes (WBC), 1.1 k/mkl, absolute neutrophil count (ANC) 0-53; and platelets 8-4 k/ml. The absolute number of reticulocytes was 4-6 thousand/MCL with a reticulocyte index (RTSI) of 0.04, indicating a hypoproliferative state. Within 72 hours after the initial appeal, the number of blood cells.

The patient was denied a fungal infection. The patient was prescribed broad-spectrum antibiotics based on the criteria for fibrill neutropenia, including piperasillin/tazobactam and vancomycin. Amphotericin was included in the treatment for suspected fungal infection. Despite these measures, pantsitopenia and stomatitis intensified and transfusions were performed many times. Aerobic and anaerobic infections in the blood were negative. A viral panel was found to contain herpes virus Type 1 (HSV-1) in a sample from the oral mucosa. An additional investigation was conducted due to the migration of pantsitopenia.Severe anemia, neutropenia, and thrombocytopenia in the peripheral blood were found without obvious morphological changes. In connection with the prediction of hypoproliferasia, a bone marrow biopsy was obtained based on peripheral blood results, which did not detect a hypocellular bone marrow (<5% cellular) without dysplastic or lymphoproliferative signs, as well as a normal XX karyotype and chromosomal or immunophenotypic anomaly. Fluorescent hybridization (FISH) assay mutations in Retinic acid alpha receptors (RARA) and promielocytic leukemia protein (PML) gave negative results.

HSV-1 was negative when the bone marrow was immunogystochemically stained. Based on the detection of HSV-1 in the oral mucosal floor, an Acyclovir treatment regimen (dose of 5 mg/kg) was used for seven days, which led to a rapid recovery of all cell lines and symptomatic improvement. A second bone marrow biopsy confirmed rapid cell (20-30%) recovery with unchanged immunophenotypic, chromosome and genetic profiles. From Anamnesis, the patient has not been identified for the effect of radiation or related anti-inflammatory, antimicrobial, anticonvulsant or chemotherapeutic agents. The levels of heavy metals have not been measured.The patient did not have autoimmune diseases/connective tissue diseases.The autoimmune Panel 1:160 норма (Titr <1:80) titre has been found to contain an antinuclear antitanacha (ana).

Based on the rapid recovery of the number of cells in the peripheral blood, as well as bone marrow biopsies with normal cytogenetics, the patient was diagnosed with aplastic anemia after the start of anti-viral treatment, possibly triggered by an HSV-1 infection. Although the patient had elevated ana titers, the autoimmune condition was considered less likely based on the absence of other signs of an autoimmune condition, and the improvement of stomatitis and pantsitopenia without the introduction of corticosteroids or immunosuppressants.

Discussion

Aplastic anemia is a rare condition [1], defined by peripheral cytopenia and hypocellularity of the bone marrow (<30%), which cannot be associated with dysplasia, lymphoproliferative diseases or bone marrow fibrosis [2]. Finally, bone marrow hypocellularity can also be associated with an autoimmune process. In response to drug exposure, viral infection, or other conditions, T-cell mediated hematopoiesis cell destruction occurs, leading to subsequent peripheral pantsitopenia [2,3]. This last mechanism may explain why the patient does not have a viral agent in the bone marrow biopsy. This mechanism is associated with immunological diseases (rheumatoid arthritis and lupus) and pregnancy [2], as well as viral infections [3]. Viruses more commonly associated with aplastic anemia include hepatitis viruses, parvoviruses, and HIV [4,5]. While there have been reports of cases

of aplastic anemia associated with infections caused by chickenpox virus (VZV), Epstein – Barr virus (EBV), and HSV-6 [4,6-11], no cases of HCV-1-Associated aplastic anemia have been reported in modern literature. Depending on the severity of the cytopenia, this condition can be classified as moderate, severe, or very severe. This patient met all criteria for very severe aplastic anemia (Hb < 8 g/dl, reticulocyte count <20 k/mkl, ANC <0.2 k/mgl, and platelets <20 k/mkl) [12]. As for treatment, in mild to moderate cases, active control is carried out. Most adults in the category of severe or acute aplastic anemia require immunosuppressive therapy in the form of an antitimocytic globulin, usually in combination with cyclosporine, or if the patient is under 40 years of age, the patient is advised to undergo a bone marrow transplant [2,12].

Conclusions

Therapists exposed to the clinical condition of rapidly developing pantsitopenia consistent with aplastic anemia should consider viral infection a possible etiology. In most cases of severe aplastic anemia, immunosuppressive therapy or bone marrow transplantation is required. In the treatment of aplastic anemia with HSV-1 etiology, anti-viral therapy facilitates the course of the disease.

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