KLINIK LABORATOR DIAGNOSTIKADA INNOVATSION TEXNOLOGIYALARDAN FOYDALANISH, MUAMMOLAR VA YECHIMLAR

xalqaro ilmiy-amaliy anjuman

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Mielogrammada yetilgan granulositlar kamayishi, eritrositar va megakariositar qator hujayralar kamayishi, blast hujayralar oshishi.

Surunkali mieloleykozda umumiy qon tahlili:: gemoglobin - 75 g/l; eritrotsitlar — $2,6\times10^{12}$/ l, rang ko‘rsatkichi 0,86, gematokrit — 22%, eritrositlar hajmi (MCV) — 100 fl, eritrositdagi gemoglobin miqdori (MCH) — 29 pg, trombotsitlar — $645\times9x10^{9}$/l, retikulotsitlar — 0,1%, leykotsitlar — 278×10^{9}/l. Leykotsitlar formulasi: eozinofil - 7%, bazofil - 5%, blastlar - 2%, promielotsitlar - 9%, miyelotsitlar - 19%, metamiyelotsitlar - 25%, neytrofillar - 21%, limfotsitlar - 12%. ECHT — 65 mm/soat.

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HEMATOLOGICAL CHANGES IN PATIENTS WITH COVID-19

Kurbonova Z.Ch., Khushbokova G.U.
Tashkent medical academy, Tashkent, Uzbekistan

COVID-19 is a systemic infection with significant effects on the hematopoietic system. Coronavirus disease (COVID-19) was first reported during an outbreak in Wuhan, which quickly developed into a pandemic and infected billions of people who were forced to observe social distancing measures. Although COVID-19 is primarily a respiratory infection, recent evidence suggests that it should be considered a systemic disease affecting the cardiovascular, respiratory, gastrointestinal, neurological, hematopoietic, and immune systems.

Objective. To study the relationship between the number of leukocytes, leukocyte count and platelet count in the general blood test for Covid-19 with the severity of the coronavirus infection and the level of lung damage.

Materials and methods. As an object of study, the anamnesis of 100 patients who were treated with a diagnosis of Covid-19 in 2020-2021 in a multidisciplinary clinic of the Tashkent Medical Academy was studied. All subjects are divided into three groups:

Group 1. 30 patients with mild Covid-19.

Group 2. 36 patients with moderate Covid-19.

Group 3. 34 patients with severe Covid-19.

Result. During the incubation period and at the early stage of coronavirus infection, peripheral blood values were normal. In the 1st group of patients with mild severity, lung damage was not detected in 10 (5%) patients, leukocytosis was more than $12.0x10^9$/l and neutrophilia was more than 72%, in 8 (4%) patients, lymphocytopenia was less than 15%, in 4 (2%) patients thrombocytosis 400x109/l and in 6 (3%) patients thrombocytopenia less than $150x10^9$/l. In the 2nd group, patients infected with coronovirus in the moderate stage had respiratory failure and 20% of patients had lung damage, 68 (34%) patients had leukocytosis more than $12.0x10^9$/l, neutrophilia more than 72%,) patients had lymphocytopenia 15% less than 6 (3%) patients had thrombocytosis and 16 (8%) patients had thrombocytopenia. In 26 (13%) patients with severe coronavirus infection of the 3rd group with more than 50% lung damage, leukocytosis above $20x10^9$/l and neutrophilia in 78% with an admixture of myelocytes and metamyelocytes were detected, in 10 (5%) patients - leukocytosis $12x10^9$/l and neutrophilia 72, in 14 (7%) patients leukocytopenia was less than $4.0x10^9$/l. In addition, 32 (16%) patients had less than 10% lymphocytopenia, 6 (3%) had thrombocytosis, and 18 (9%) had thrombocytopenia. In the control group, the number of leukocytes was $6.5\pm1.2x10^9$/l, platelets $212\pm41.3x10^9$/l, the leukocyte formula did not change.

Summary. The development of leukocytosis and neutrophilia in the general blood test in patients with Covid-19, their level, lymphocytopenia and thrombocytopenia depend on the severity of the coronavirus infection and the degree of lung damage.

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APLASTIK ANEMIYA KLINIK LABORATOR DIAGNOSTIKASI

Kurbonova Z.Ch., Babadjanova Sh.A.
Toshkent tibbiyot akademiyasi

Aplastik anemiya gematologik sindrom bo’lib, bunda o’zak hujayra va uning mikromuhitidagi sifat va miqdoriy o’zgarishlar natijasida periferik qonda pansitopeniya va qizil suyak ko’migi to’qimalarining yog’li degeneratsiyasi rivojlanadi.

Aplastik anemiya kamdan-kam uchraydigan kasallik bo’lib, yiliga 1 million kishiga 2-3 holatni tashkil qiladi. Aplastik anemiya barcha yosh guruhlarida uchraydi, ammo ikkita cho’qqi qayd etiladi - 10-25 yoshda va 60 yosh va undan katta yoshda, ko’pincha ayollarda.

Bemorlarning taxminan yarmida aplastik anemiya sabablari noma’lum bo’lib qolmoqda, hatto diqqat bilan to’plangan anamnezda ham. Aplastik anemiyaning rivojlanishiga sabab bo’ladigan sabablar kimyoviy moddalar (benzol va uning hosilalari, nitroemallar, laklar, pestitsidlar va boshqalar), ionlashtiruvchi nurlanish, dorilar (antibiotiklar, sulfanilamidlar, tutqanoqqa qarshi preparatlar va boshqalar), bakterial va virusli infektsiyalar bo’lishi mumkin.

Hozirgi vaqtda aplastik anemiyaning shakllanishi va rivojlanishining bir necha mexanizmlari mavjud: gematopoetik o’zak hujayraning proliferativ faolligining buzilishi bilan funksional va anatomik nuqsoni, o’zak hujayra mikromuhiti elementlarining shikastlanishi va uning funktsiyasini bilvosita pasayishi yoki buzilishi, immunopatologik holat tufayli gematopoezning disregulyatsiyasi yoki uning bostirilishi, gematopoezni rag’batlantiruvchi omillarning yetishmasligi.

Aplastik anemiya patogenezining turli mexanizmlarining kombinatsiyasi ham mumkin. Aplastik anemiyaning og’irligini aniqlashda, davolanish boshlanishidan oldin kasallik tashxisi qo’yilganda kamida uchta periferik qon tekshiruvi natijalari hisobga olinadi. Refrakter aplastik anemiya davolash