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THE PARADOX OF HYPERCHROMIC ANEMIA IN CHRONIC INFLAMMATION: A CASE STUDY CHALLENGING THE CLASSIC PARADIGM OF ANEMIA OF CHRONIC DISEASE

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Relevance. Anemia of Chronic Disease (ACD) is the second most common anemia worldwide, affecting 30–50% of chronic illness patients, resulting from inflammation-driven hepcidin excess, causing iron sequestration, erythropoietic suppression, and shortened erythrocyte survival. Clinically, ACD presents as normocytic/normochromic anemia. Our case is centered around a patient with chronic multimorbidities presenting all classic risk factors for ACD but instead presents with macrocytic hyperchromic anemia, defying the expected normocytic/normochromic phenotype.

Aim: studying the case of coexisting ACD with other anemia type.

Materials and methods. An organized search was conducted to study the pathogenesis, incidence and patterns presented by ACD. Sources from 2018 were used to build the theoretical background of our study. Case reports from Clinical Hospital No.6, Minsk were reviewed and after careful assessment, we chose this case which exhibited this paradox.

Case presentation: 76y/o male with history of coronary heart disease, two episodes of myocardial infarctions, Chronic Obstructive Pulmonary Disease, silicosis, 1st Degree of esophageal varices, gastropathy due to portal hypertension, erythematous gastropathy of 2nd degree and acquired kidney cyst. Laboratory tests showed decreased values of – Erythrocytes- $1.7x10^{12}$, Hemoglobin-79g/L, Hematocrit-22.3%, Thrombocytes-$86x10^{9}$, Albumin-30.4g/L, Cholesterol-2.43mmol/L and increased values of Mean Corpuscular Volume (MCV)-129.7fL, Red-cell Distribution Width-Standard Deviation (RDW-SD)-72.5fL, Macrocytic Red cell population (Macro-R)-52.6%, Platelet Distribution Width (PDW)-23.7fL, Mean Platelet Volume (MPV)-13.5fL. Additionally, there was a rise in total-35.7mmol/L, indirect-23.3mmol/L & direct bilirubin-12.4mmol/L, Very Low Density Lipoprotein (VLDL)-0.47mmol/L, and Lactate Dehydrogenase (LDH) showed a 5-fold increase of 1207 E/L compared to the upper limit. Serum iron was 5.0mcmol/L (normal range is 11.6-31). All other parameters mentioned were normal. On physical examination, no pathological symptoms related to the liver or kidney were found. No hepatomegaly or splenomegaly were found.

Results and their discussion. ACD creates a baseline of iron restriction and erythropoietic suppression, but secondary triggers can potentially dominate the phenotype. 1) Elevated LDH and indirect bilirubin suggest intravascular hemolysis, 2) Thrombocytopenia & high MPV, PDW supports peripheral destruction, 3) Macrocytosis & thrombocytopenia raises suspicion for myelodysplastic syndrome, B12 deficiency or folic acid deficiency, 4) Gastropathy potentially results in B12 malabsorption, 5) Mixed hyperbilirubinemia suggests congestive hepatopathy due to mainly heart failure while elevated VLDL and low cholesterol may reflect statins intake or impaired hepatic lipid metabolism which tends to cause macrocytosis due to altered erythrocyte membrane lipids.

Conclusion. Chronic inflammation should suppress erythropoiesis and cause normocytic ACD. Instead, macrocytosis and hemolysis markers suggest competing pathologies superseding ACD. This case is a “perfect storm” of chronic inflammation & ACD mechanisms compounded by hemolysis, nutritional deficits, liver dysfunction and B12 deficiency. The multimorbidity in this case creates a “layered anemia” building upon ACD. However, further studies are required to be conducted to assess this deviation and to establish definitive correlation.