Rheumatoid anemia

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Résumé en anglais	Rheumatoid anemia is a typical example of anemia of chronic disease. It differs from other forms of anemia, such as iron deficiency anemia or iatrogenic anemia. Rheumatoid anemia is normochromic, normocytic or, less often, microcytic, aregenerative, and accompanied with thrombocytosis. Serum transferrin levels are normal or low, transferrin saturation is decreased, serum ferritin levels are normal or high, the soluble transferrin receptor (sTfR) is not increased (a distinguishing feature with iron deficiency anemia), and the sTfR/log ferritin ratio is lower than 1. This review discusses the prevalence and impact of rheumatoid anemia based on a review of the literature. Iron metabolism, absorption, diffusion, storage, and use by the bone marrow are described using published data on transferrin, ferritin, and hepcidin. Hepcidin is now recognized as a key factor in rheumatoid anemia, in conjunction with the cytokine interleukin-6 (IL-6). Hepcidin is a hormone that lowers serum iron levels and regulates iron transport across membranes, preventing iron from exiting the enterocytes, macrophages, and hepatocytes. In addition, hepcidin inhibits intestinal iron absorption and iron release from macrophages and hepatocytes. The action of hepcidin is mediated by binding to the iron exporter ferroportin. Hepcidin expression in the liver is dependent on the protein hemojuvelin. Inflammation leads to increased hepcidin production via IL-6, whereas iron deficiency and factors associated with increased erythropoiesis (hypoxia, bleeding, hemolysis, dyserythropoiesis) suppress the production of hepcidin. Data from oncology studies and the effects of recombinant human IL-6 support a causal link between IL-6 production and the development of anemia in patients with chronic disease. IL-6 diminishes the proportion of nucleated erythroid cells in the bone marrow and lowers the serum iron level, and these abnormalities can be corrected by administering an IL-6 antagonist. IL-6 stimulates hepcidin gene transcription, most notably in the hepatocytes. Studies involving human hepatocyte exposure to a panel of cytokines showed that IL-6, but not TNFα or IL-1, induced the production of hepcidin mRNA. Recent data on hepcidin level variations in patients with rheumatoid arthritis are reviewed. Rheumatoid anemia is best corrected by ensuring optimal control of systemic disease activity. The role for iron supplementation (per os or intravenously) and erythropoietin in the treatment of rheumatoid anemia is discussed. Given the cascade of interactions linking IL-6, hepcidin, and anemia, IL-6 antagonists hold considerable promise for the management of rheumatoid anemia.
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