The cutoff value of serum ferritin for iron deficiency anemia in determining treatment termination

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Introduction: Iron deficiency anemia (IDA) is the most common hematologic problem and easily treated with oral ferrous sulfate. In diagnosis of IDA, the serum ferritin level is the most reliable factor. However, there is no definite cutoff value of serum ferritin for the termination of iron therapy. A small-scale retrospective study was therefore carried out to determine an appropriate level of serum ferritin. Methods: Forty-seven patients who were diagnosed with IDA and treated with oral ferrous sulfate at Korea University Guro and Ansan Hospital from January 1, 2006 to January 1, 2009 were retrospectively analyzed by using the hospital’s medical records on their treatment and hemoglobin levels. Results Thirty-nine patients were enrolled in the treatment success group and eight patients were in the treatment failure group. The cutoff value of serum ferritin for the termination of treatment was 22.95 ng/mL (sensitivity 57.1%, specificity 93.3%). The period that the hemoglobin level was maintained after termination of treatment was not significantly different between the group with a serum ferritin level over 23 ng/mL and the group with a serum ferritin level below 23 ng/mL. (Kaplan-Meier curve, p=0.42). The hemoglobin level at the time of treatment termination was a significant prognostic factor for treatment response (p=0.04) in multivariate analysis. Conclusion: The cutoff value of serum ferritin for treatment termination was 22.95 ng/dL, and hemoglobin at the time of treatment termination was a significant prognostic factor (p=0.04). The hazard ratio of the hemoglobin level at the time of treatment termination was 0.27. Further large-scale studies should be undertaken. Keywords: Iron deficiency anemia, Serum ferritin, Ferrous sulfate, Treatment termination

Dapsone-induced hemolytic anemia

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Dapsone has been used for various cutaneous, infectious, and rheumatologic diseases due to its potent anti-bacterial and anti-inflammatory effects. However, the clinical use of dapsone may be limited by its potential various side effects including skin, nervous, gastrointestinal, hepatic, renal, and hematologic toxicities. In hematologic toxicities, dapsone usually causes non-immune hemolytic anemia and methemoglobinemia. We herein report four cases of non-immune hemolytic anemia induced by dapsone. These patients had taken dapsone for one month to one year duration and presented with normocytic- or macrocytic anemia. Laboratory findings revealed hemolytic features such as increased reticulocyte production index (RPI), decreased serum haptoglobin, indirect bilirubinemia, and elevated lactase dehydrogenase (LDH) levels. Peripheral blood smear (PBS) showed mild to moderate degree of spherocytosis. Other possible causes of hemolytic anemia were excluded, hence, they were considered as dapsone-induced non-immune hemolytic anemia. After holding of dapsone administration, all patients were recovered from anemia and had no feature of hemolysis. Since early stage of dapsone-induced hemolytic anemia is usually overlooked, severe degree of hemolytic anemia may be occasionally provoked. Common but overlooked mild hemolytic anemia due to dapsone is known to be only cured by discontinuation of the drug. Intensive investigation and the discontinuation of caustic drug including dapsone should be warranted for the patients with non-immune hemolytic anemia.