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Title

Diagnosis of hemochromatosis [4] (multiple letters)

Permalink

<https://escholarship.org/uc/item/8jf017xf>

Journal

Annals of Internal Medicine, 131(4)

ISSN

0003-4819

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Publication Date

1999-08-17

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Peer reviewed

disease (ESRD), the relation between tissue iron stores and serum ferritin levels is altered. A relative increase in serum ferritin levels unrelated to iron stores or acute phase reactants is frequently observed (2, 3). Serum ferritin levels of 1000 $\mu\text{g/L}$ or greater in patients with ESRD are not uncommon, and iron overload is extremely rare (3, 4). In one of our dialysis units, the mean serum ferritin level is 630 $\mu\text{g/L}$, with a median value of 601 (SD, 358 $\mu\text{g/L}$). By using bone marrow iron stores, we showed that a serum ferritin level less than 200 $\mu\text{g/L}$ is highly specific for iron deficiency in patients with ESRD (2). Moreover, serum ferritin levels as high as 1200 $\mu\text{g/L}$ may still be consistent with low to normal iron stores in patients with ESRD.

In a study on nutritional assessment (5), we showed that severely malnourished patients undergoing dialysis had the highest serum ferritin levels, and well-nourished patients undergoing dialysis had lower values. We also found an inverse correlation between serum ferritin and transferrin levels ($r = -0.61$) in patients with ESRD and showed that low serum transferrin levels due to malnutrition may cause an erroneously normal to high transferrin saturation ratio, even in the presence of iron deficiency (2, 5). We suggest that the hemochromatosis criteria be modified for patients with ESRD.

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In response: Our discussion of the laboratory criteria for hemochromatosis was clearly in the context of an otherwise healthy person or one with clinical features suggestive of the disease. We stated that in this situation "the serum ferritin level defines the point at which hemochromatosis is expressing iron overload and treatment should be initiated." It is then that the serum ferritin level exquisitely reflects body iron stores. Other conditions can indeed cause an elevation in serum ferritin level out of proportion to iron stores, and we listed inflammation, infection, and cancer because they can complicate hemochromatosis. Renal disease is rare in hemochromatosis, and end-stage renal disease would be entirely unrelated. It is also noteworthy that most studies of chronic renal disease assess body iron stores by bone marrow iron. This is not applicable to hemochromatosis, in which hepatic iron concentration is increased while bone marrow iron stores are normal.

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Diagnosis of Hemochromatosis

To the Editor: We take issue with the hemochromatosis laboratory criteria by Powell and colleagues (1). The authors concluded that a serum transferrin saturation greater than 45%, a serum ferritin level greater than 200 $\mu\text{g/L}$ in premenopausal women, and a serum ferritin level greater than 300 $\mu\text{g/L}$ in men and postmenopausal women are indicators for primary iron overload. Although the authors explain that elevated serum ferritin levels may be due to inflammation, infection, or cancer and suggest that acute-phase reactants be obtained to exclude these entities, they ignore the fact that in patients with end-stage renal