Serum Ferritin Level in Hemodialysis Patients

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Dear Sir,

It has been recognized for some time that, in hemodialysis patients, there may be a disparity between the serum ferritin level and the serum iron/total iron binding capacity (TIBC) ratio [1]. Namely, the serum ferritin may be high despite a low iron/TIBC ratio. Various studies, with conflicting results, have attempted to determine which parameter most closely represents bone marrow iron stores in hemodialysis patients [2, 3]. One study showed that serum ferritin levels correlated well with the degree of hepa-tosplenic siderosis but not with bone marrow iron stores in hemodialysis patients treated with intravenous iron-dextran [3]. In our dialysis unit, standard practice is to use supplemental iron therapy when the iron/TIBC ratio is < 0.2 regardless of the serum ferritin. In order to determine how frequently these two values are, in fact, disparate, we conducted a retrospective review of our chronic hemodialysis patients.

We identified all our current hemodialysis patients who had been on dialysis for at least 6 months. We reviewed their charts to identify if they had any iron/TIBC ratio < 0.2 in the past 2 years. If so, we then examined, if at any of these times, they concurrently had a serum ferritin above normal. We also attempted to determine if there was any acute illness at the time that could account for a high serum ferritin as an acute phase reactant.

26 patients were identified with a low iron/TIBC ratio at least once in the last 2 years. 19/26 (73%) of these patients concurrently had a serum ferritin above normal. Of these, 6 patients had an acute illness at the time and 2 were antihepatitis C positive. 3/26 (12%) had a serum ferritin $> 1,000$ ng/ml. Of these, 2 were the patients who were antihepatitis C positive; 1 had no known liver disease and had no acute illness at the time.

Our results show that serum ferritin and iron/TIBC ratio are frequently disparate in hemodialysis patients. In some patients this might be accounted for by an acute illness or liver disease, both known to elevate the serum ferritin. However, there are clearly many patients with disparate values in whom no obvious cause can be found. Possible explanations for a high serum ferritin in hemodialysis patients could include (1) unrecognized acute illness, (2) activation by hemodialysis membranes or (3) the state of uremia itself. Alternatively, it is possible that these patients have high total body iron stores but decreased functional availability of iron for erythropoiesis. If this is the case, are we contributing to hepa-tosplenic hemo-siderosis by treating these patients with supplemental iron? Studies, in the future, should attempt to further clarify these issues and their relation to erythropoietin, intravenous iron therapy, and dialysis membrane composition in hemodialysis patients.

References

Kooistra MP, van Es A, Struyvenberg A, Marx JJ: Iron metabolism in patients with the anaemia of end-stage renal disease during treatment with recombinant human erythropoietin. *Br J Haematol* 1991;79:634-639. Van de Vyver FL, Vanheule AA, Majelyne WM, D'Haese P, Blockx PP, Bekaert AB, Buysens N, de Keersmaecker W, De Broe ME: Serum ferritin as a guide for iron stores in chronic hemodialysis patients. *Kidney Int* 1984;26:451-458. Ali M, Rigolosi R, Fayemi AO, Braun EV, Frascino J, Singer R: Failure of serum ferritin levels to predict bone-marrow iron content after intravenous iron-dextran therapy. *Lancet* 1982;i:652-655.

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