Short Communication

Should a Search for Iron Deficiency be Part of the Regular Screening in All Patients, whether Anemic or not?

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Introduction

In the Symphonie Fantastique written in 1830 by Hector Berlioz, in all five movements, a certain obsessional theme - a leitmotif - keeps coming back again and again in different guises. But even though it may sound different in each of the five movements, and is played by different instruments, it is basically the same.

There is a striking resemblance between this and a leitmotif that runs through many medical conditions- iron deficiency (ID). It may cause different signs and symptoms in different people [1,2], including anemia, weakness, frailty, headache, irritability, shortness of breath, tiredness, reduced exercise tolerance, a tendency to fall, lack of cognitive function, restless legs syndrome, depression, worsening of Quality of Life and increased hospitalizations and morbidity. And it may worsen Congestive Heart Failure (CHF) [3-6]. It may also present with or without anemia.

There are two different types of ID-absolute ID with low serum iron, low% Transferrin Saturation and low serum ferritin (associated with low total iron stores), or functional iron deficiency with low serum iron, low% Transferrin Saturation and elevated serum ferritin (this combination suggesting normal or high body iron stores- the so-called Anemia of Chronic Disease [7,8].

In absolute iron deficiency there is an actual reduction in body stores of iron, and the serum ferritin levels are generally less than 30 ug/l. It can be caused by poor nutrition with reduced iron intake, malnutrition, a % Transferrin Saturation (serum iron divided by serum transferrin and this multiplied by 100%) <20%. It is caused by the iron deficiency [36,37]. In absolute iron deficiency there is an actual reduction in body stores of iron, and the serum ferritin levels are generally less than 30 ug/l. It can be caused by poor nutrition with reduced iron intake, malnutrition, a % Transferrin Saturation (serum iron divided by serum transferrin and this multiplied by 100%) <20%. It is caused by the iron deficiency [36,37].

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concluded that ID is extremely common in hospitalized CHF patients whether anemic or not but is usually not sought after by most physicians at the time of admission. The fact that, in our study, about 3/4 of those with a complete iron workup received oral or IV iron therapy at discharge suggests that doctors who investigate for iron deficiency and find it are usually willing to treat it if it is found—an optimistic note and the motivation for this paper.

In another recent study of COPD patients [39] we examined the hospital records of all patients hospitalized in our hospital with a primary diagnosis of exacerbation of COPD to assess the investigation, prevalence, and treatment of ID. In this same study we also examined retrospectively the records of 12 anemic COPD outpatients (anemia: Hb <12 g/dl) who had been treated in our nephrology clinic with the combination of ESAs and IV iron given once weekly for 5 weeks. Initially and one week after treatment we measured the hematological response and the severity of dyspnea by Visual Analogue Scale (VAS). Of 107 consecutive patients hospitalized with exacerbation of COPD examined, 47 (43.9%) were found to be anemic on admission. Two (3.3%) of the 60 non-anemic patients and 18 (38.3%) of the 47 anemic patients had a full iron workup with serum iron, %Transferrin Saturation (%TSat) and serum ferritin measured. All 18 (100%) anemic patients had ID by our criteria (the same criteria as in the previous study), yet none had oral or IV iron subscribed before or during hospitalization, or at discharge.

In the intervention outpatient study, ID was found in 11 (91.7%) of the 12 anemic ambulatory COPD patients. With Erythropoiesis Stimulating Agents and IV iron treatment the mean Hb increased from 9.72 ± 1.16 to 12.29 ± 1.09 g/dl and the VAS scale increased from 2.20 ± 1.13 to 8.45 ± 0.92 p = 0.002. The VAS was highly correlated with the change in Hb.

It is important to stress that many patients with iron deficiency are not anemic and physicians will often not measure iron parameters unless anemia is present. This in our opinion is a serious error. Many patients with severe symptomatic iron deficiency will be missed.

**Conclusion**

Iron deficiency, whether absolute or functional is exceedingly common, yet it is often not sought after and, if it is found, it is often not treated. But treatment with oral or intravenous iron can improve the anemia, fatigue, exercise tolerance and endurance, cardiac function, cognitive function and quality of life in a great many conditions. It is an unmet challenge for physicians. Measurement of serum iron, Transferrin, percent Transferrin Saturation, and serum Ferritin should, in our opinion, be part of the standard workup of all patients—whether or without anemia, since many iron deficient patients are not anemic. This includes patients in both family practice and on admission to hospital for any cause. If iron deficiency is discovered its cause should be looked for and, when discovered, should be treated. The improvement of the patient with ID with oral or IV iron will often be rapid, striking and gratifying both for the patient and for the treating physician. Few conditions in medicine are as common, as easily diagnosed and treated, and can improve the quality of life as much as iron deficiency.

**References**


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