Correspondence

High prevalence of iron deficiency anemia in infants attending a well-baby clinic in NorthWestern Saudi Arabia

To the Editor

I have 3 comments on the interesting study by Al Hawsawi et al[1] on the prevalence of iron deficiency anemia (IDA) in infants attending a well-baby clinic in NorthWestern Saudi Arabia.[1]

First, Al Hawsawi et al[1] addressed high prevalence (51%) of IDA in Saudi infants aged 6-24 months. I presume that such prevalence ought to be cautiously taken. This is based on the presence of an important methodological limitation. There has been a continuous improvement in the methods to detect iron deficiency (ID), a common condition in children, in the last decades or so, but it is still difficult to establish which parameters should be included in a diagnostic panel for ID and IDA.[2] New hematological parameters, such as reticulocyte hemoglobin content (CHr) have expanded the classic ones, such as mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC). A variety of hematology analyzers now provide novel parameters to assess cellular hypochromia and microcytosis in both reticulocytes and mature red blood cells. The repertoire of biochemical markers has also been expanded, with iron, transferrin, and ferritin being supplemented by circulating transferrin receptor and hepcidin. Molecular identification of functional variants of key iron metabolism determinants has provided explanations for the heritability of some iron metabolism biomarkers.[3] Al Hawsawi et al[1] mentioned in the study methodology that the diagnosis of IDA was relied upon hemoglobin (Hb) concentration of less than 11 g/dl, and serum ferritin (SF) of less than 10 μg/L. It is obvious that SF concentration, as a parameter of iron status that is commonly used in the diagnosis of IDA, often has limited values if the iron deficiency is accompanied by inflammatory disease.[4] Among the new hematological parameters recently developed to precisely diagnose IDA, CHr has recently gained ample attention in the pediatric clinical settings since it is found to be the strongest predictor of ID and IDA,[2,5,6] as well as it could be considered an affordable and widely available marker to detect early responders to oral iron therapy and to switch unresponsive children to parenteral iron supplementation or transfusion.[7] Actually, the utility of CHr in the identification of Saudi children with IDA has been evaluated and showed that a CHr cut-off level of 26 pg is considered to be a reasonable indicator of anemic states. Moreover, CHr together with a complete blood count might provide an alternative to the traditional hematologic or biochemical panel for the diagnosis of ID and IDA in young children and it is cost-effective.[8] I wonder why Al Hawsawi et al[1] did not employ the protocol of CHr estimation in the their study methodology. I presume that the implementation of that protocol could better elucidate the actual prevalence of IDA in the Saudi pediatric population.

Second, Hawsawi et al[1] mentioned that IDA was noticed in males (53%) more than females (47%). However, the difference was not statistically significant (p=0.367). I presume that such male predominance was not an incidental observation. It could be partly attributed to the gender variations in iron status that renders male infants more susceptible to IDA. An interesting Swedish study showed that at 4, 6, and 9 months, boys had significantly lower blood Hb, MCV, ferritin and higher zinc protoporphyrin (ZPP), and transferrin receptors (TfR) than girls. At 9 months, boys had a 10-fold higher risk of being classified as having IDA. The differences at 9 months in blood MCV and ZPP remained significant after controlling for iron supplementation, growth variables, and other possible confounders. The study concluded that there were substantial gender differences in Hb and other indicators of iron status during infancy The gender differences in some variables (MCV, ZPP, and ferritin) might be due to genetic or hormonal factors, whereas the differences in some other variables (Hb and TfR) probably reflected a truly increased risk of IDA in boys.[9]

Third, based on the high prevalence of IDA in Saudi infants (51%) in Hawsawi et al's study[1] together with the notion that iron supplementation for IDA in young children might improve growth and development outcomes, Al Hawsawi et al[1] recommended iron supplementation to be given at early infancy with universal screening of Hb and SF estimations to all infants at 12 months of age. I presume that such recommendation needs to be interpreted with cautions. This is based on the following 3 points: 1) The exact prevalence of IDA in Saudi children at the national level is not yet fully known to be sizable. Large scale community-based studies at the national level employing a sound methodology is needed; 2) The costs of IDA screening and iron supplementation is substantial and, hence, the required budget ought to be created. For instance, the cost-benefit profile of CHr with Hb alone and Hb as a component of the complete

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blood count for the detection and treatment of ID in 9- to 12-month-old infants has been evaluated. The incremental cost to diagnose and treat ID, compared with diagnosing and treating anemia by Hb, was only $22 per patient screened ($440 per case of anemia prevented; number needed to treat = 20). With a 10-year time horizon incorporating risks and costs of neurocognitive delays associated with untreated ID, the cost of the CHr strategy was $280 per case of anemia prevented.[10] Evaluating the cost of implementing the program of multimicronutrient supplementation containing iron revealed that the annual cost per community member was US $1.51 and the cost-effectiveness ratio was US $0.12 per 1% of prevented anemia per community member;[11] and 3) The recently published review of the evidence regarding the benefits and harms of screening and routine supplementation for IDA for the US Preventive Services Task Force has addressed that although some evidence on supplementation for IDA in young children indicates improvements in hematologic values, evidence on clinical outcomes is lacking. Moreover, no randomized controlled screening studies are available to be evaluated.[12]

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Reply from the Author

No reply was received from the Author.

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