ANEMIA IN WOMEN DURING REPRODUCTIVE YEARS IN RURAL AREA

Maida Y. Shamdeen[1] *MRCOG, FRCOG*, **Baybeen K.Alselevany** [2] *PhD*.

Abstract

Background: iron deficiency anemia **(IDA)** is a medical and public health problem of prime importance, causing few deaths, but contributing seriously to the weakness and substandard performance of millions of people**.**

Objectives: To determine the prevalence of anemia, 10 years after sanction among women, at reproductive years in rural areas.

Patients & Methods: The study was carried out in September 2002 within field application for university of Mosul on women in reproductive years in Badoosh areas, 20 Km to the North of Mosul city. The study was conducted in rural areas, where 98 women were evaluated clinically, after a questionnaire with 17 items including age, marital status, and social status, number of children, lactation, and menstrual blood loss. A blood sample was taken to evaluate hemoglobin level (Hb), Hematocrit (hct), serum iron level (SI), total iron binding capacity (TIBC), and transferrin saturation (TS).

Results: The mean age of the women with all tests available was 28.75±10.6 years (range15-50 years); the mean number of previous pregnancies in parous women was 5 pregnancies. 58 women were found to be anemic (57.14%). The mean values of their Hb, hct, SI, TIBC and TS in anemic and non anemic group were; (106.8g/l,126.79g/L),(0.32L/L,37.9L/L),(13.53µmol/dl,15.42µmol/L),(69.85µmol/L,62.55 µmol/L) and(19.37%,24.7%) respectively, while the over all results for the same values for all women were 115.4g/L, 0.34L/L, 14.34 µmol/L, 61.01µmol/L and 23.50% respectively. In the anemic group 37 women were married (66.07 %),10 women (17.3%) were lactating, 28women (48.3 %) had more than 4 children, 98 % of the sponsors of the family were workers of low socioeconomic status, 12 (12.3%) married women had heavy menstrual cycle and 84(85.7%) of the families had more than 6 persons in the house.

In the present study the level of Hb was lower and TIBC was higher in anemic as compared to non-anemic patients ($p< 0.05$), while there was no significant difference in the levels of hct, SI, and TS% in anemic patients from that of non-anemic patients ($P>0.05$).

Conclusions: Almost all the anemic women were suffering from iron deficiency (ID) ،which is mainly due to nutritional factors and low socioeconomic status, multiparity, lactation and heavy menstrual loss. This may reflect the effects of the blockade on the nutritional and social status in the rural areas.

Recommendations: For girls ages 12-18 and non-pregnant women of childbearing ages, it is recommended to screen for anemia every 5 years, and annual screen for women with risk factors for iron deficiency anemia, and more frequent in pregnant women. Give iron supplements to all women in reproductive years in rural areas.

Key words: IDA, reproductive years of women life.

IRAQI J MED SCI, 2007; VOL. 5 (2):65-70

Introduction

The WHO criterion for anemia in women is Hb less than 120 gm/L and less than 110gm/L in pregnant women due to physiological anemia [1- 7]. Anemia may be difficult to define in countries in which malnutrition, infection, high altitude, air pollution and smoke or congenital hematological disorders are common [1-3, 6-12]. The prevalence of ID is 10-15% in pregnant compared to3-4% in non-pregnant women. Flemings et al [13], found that approximately 50% of the anemic women were ID. The signs and symptoms

[1]**Dept.of Obstet & Gynecol, College of Medicine Duhok University,** [2]**Dept. Of Medical Physiology College of Medicine Mosul University Mosul – Iraq**

ddress correspondence to:Dr.BaybeenK.AlselevanyEmail: bselevany@yahoo.comMobile: 0770 161 0242

Received 21st December 2004: Accepted 19th November 2006.

of anemia are dependent upon the degree of anemia, as well as the rate at which the anemia has evolved. The history, physical examination, and simple laboratory testing are all useful in evaluating the anemic patient. One or more of the three independent mechanisms can cause anemia: decreased RBC production, increased RBC destruction, and RBC loss [1, 3, 14-15].

The classical presentation of IDA is, multigravid woman in her forties, presents with chronic blood loss from menometrorrhgia, weakness, headache, irritability and varying degrees of fatigue and exercise intolerance, however many patients are asymptomatic and present only with anemia. The Plummer– Vincent or Patterson– Kelly syndrome (dysphasia, esophageal web, and atrophic glossitis), koilonychias, Chlorosis and blue sclera. Pica and pagophagia are specific for ID state; an occasional manifestation of ID is beeturia [15-22]. Reduced absorption of iron and a diet deficient in iron can cause ID [17, 23-28]. Physical examination will show pallor of the palms, nail beds, face or conjunctivae. In developed countries the prevalence of anemia is stated as below 20 %, while in developing countries the prevalence is 40-70 % [3-4, 13].

The manifestations of ID occur in several stages. They are defined by the extent of depletion, first of iron stores and then of iron available for hemoglobin synthesis [14, 20, 25-26].

Laboratory evaluation: the initial testing should include Hb, hct, RBC count and RBC indices. Important discriminating features are low SF and ST, an increased TIBC and low SI, which is excellent indicator of iron store, there appears to be a direct quantitative relationship between the SF and iron stores [23-24,26,27,29-37,39-41].Pregnant women have an elevated serum transferrin in the absence of ID [24,35-37,40-43)]. In severe IDA, SI is reduced and the TIBC is elevated; the latter finding reflects the reciprocal relationship between SI and transferrin gene expression in most nonerythroid cells [35]. The low SI and high TIBC result in a low TS (often less than 10% compared to the normal value of 25-45%)(40,42-43). One problem in pregnancy and oral contraceptives users is increase in the plasma transferrin concentration; as a result, the percent saturation may be low in such patients in the absence of ID [44-45]. Once the diagnosis of anemia due to ID is established, attempts to find out the cause should follow [27, 31, 35, 41, 46-49].

Patients & Methods

A cross-sectional study was conducted in September 2002 on women in reproductive age in Badoosh area 20Km north to Mosul city. Ninety-eight women were selected randomly: almost all in the childbearing age (14-52years), with a mean age of 28.75±10.6 years. Demographic, socioeconomic, menstrual, obstetric, and medical data were collected. Clinical evaluations for symptoms and signs of anemia were done. About 5ml of venous blood was drawn from antecubital vein .The blood sample was divided into two parts: first one ml of blood was added to a tube containing EDTA for the estimation of Hb, and hct. Second, 4ml were put in a clean dry disposable plain tube and centrifuged at 3000 rpm for 15 minutes. The serum obtained was used for estimation of the SI and TIBC, SF was not available to be done .Hb (gm/L) was measured by using cyanomethemoglobinometry, and hct (L/L) was estimated by microhematocrit methods according to Dacie and Lewis (50), SI (µmol/L) and TIBC (µmol/L) were estimated by an enzymatic colorimetric assay (Giesse Diagnostics Kit -Italy), and TS (%) was calculated by the formula; TS% = SI/TIBC X 100.

Statistical analysis was performed using student-unpaired t –test. All values were expressed as mean ± SD. The accepted level of significance was at $P<0.05$.

Results. Evaluation of the results showed that 58 women had low Hb and hct, the prevalence of anemia was 57.14% .The mean age of the women in this study was 28.75± 10.6 years, peak incidence was found in the age group 25-35years as shown in the Figure 1. The non-anemic group was 40 women. The results respectively in the anemic and non- anemic group: concerning marital status, lactation, having more than four offspring or not, and presence of heavy menstrual 37(66.07%), 22(55%) were married, 10 (17.3%), 1(2.5%) were lactating, 28 (48.3%), 10(25%) had more than four children, and 12 (12.3%),6(15%)had heavy loss as shown in (Table1), The sponsor of the families in 99%,98% of cases were workers of low socioeconomic status and (92%),(90%) 0f the families had more than six person in the house (ranging between 6 -20).The distribution of anemia according to the ages is shown in (Figure 1).

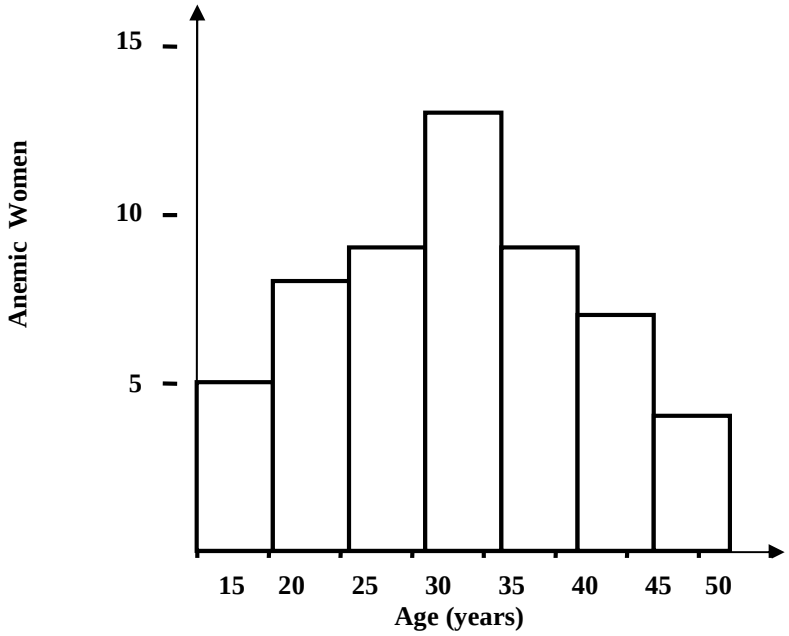

Figure 1: Distribution of anemia according to ages.

The mean number of previous pregnancies, marital status, and lactation, presence of pregnancy, and those with menorrhagia, and others are shown in (Table1).

Table 1: percentage (%) values for anemic, non anemic and mean of both group concerning marital, menstrual and pregnancy statuses.

State	Non-anemic patients n=40	Anemic patients n=58	% Of total
Married	**55%**	**66%**	**60.5%**
Single	**45%**	**34%**	**39,5%**
Lactating	**2.5%**	**17.3%**	**9.9%**

Pregnant	**2.5%**	**7.1%**	**4.8%**
Married with more than 4children	**25%**	**82.4%**	**53.7%**
More than 6person in the family	**97.5%**	**92%**	**94.8%**
Menorrhagea	**15%**	**13%**	**14%**
Low socioeconomic class	**95%**	**98%**	**96.5%**

In the present study the level of Hb is significantly lower and TIBC is significantly higher ($p<0.05$) in anemic as compared to non-anemic patients. The values of hct, SI, and TS % were lower in anemic than in non-anemic patients but these were statistically non-significant as shown in (Table 2).

Table 2: mean± SD of all variables in anemic compared to non- anemic patients.

Variables	Overall group n=98	Non-anemic group n = 40	Anemic group n = 58	p-value
Hb (gm/L)	**115.4± 14.9**	**126.79 ±5.31**	**106.8±14.2***	**0.000**
hct (L/L)	**0.345± 4.45**	**0.379±1.66**	**0.320±4.29** ^{NS}	**0. 934**
SI (µmol/L)	**14.34±6.67**	**15.42± 6.52**	**13.53±6.73** ^{NS}	**0.170**
TIBC(µmol/L)	**61.01±13.94**	**62.55±12.76**	**69.85±14.76***	**0.013**
TS (%)	**23.50±15**	**24.70±11.88**	**19.37±17.1** ^{NS}	**0.091**

NS: non-significant.

***: significant difference** * **p <0.05**

Discussion

The study showed that 57.14% of the women in this locality were anemic; this is compatible with data from other study in developing countries (1-4). The three types of factor responsible for the high prevalence of women anemia in such setting were, iron deficiencies, due to under feeding, consumption of cereal with low iron content, short intervals between pregnancies, and helminthes infestation. Poverty impairs all these factors and limited access to health care and lack of medicine. Although iron and folic acid supplementation are generally recommended, there are numerous economic, cultural and social obstacles to this simple preventive measure [2]. Logistic regression was found that anemia significantly related to the age, socioeconomic status, parity and lactation.

Conclusions

Almost all the anemic women were suffering from iron deficiency, mainly due to nutritional factors and low socioeconomic status, multi parity, lactation and heavy menstrual loss. This may reflect the effects of the sanction on the nutritional and social status in the rural areas.

Recommendations

For girls ages 12-18 and non-pregnant women of child bearing ages, it is recommended to screen for anemia every 5years, and annual screen for women with

risk factors for ID anemia. Supply Iron supplementations for all women in reproductive years of live.

References

1. Word Health Organization, nutritional anemia's; Reports of a WHO scientific group. Geneva, Switzerland WHO; 1968.

2. The prevalence of anemia in women; a tabulation of available information, Geneva, WHO 1992 WHO/ MCH/ MSM/ 92.2

3. Hercherg S, et al. Nutritional anemia in pregnant Beninese women; consequences on the hematological profile of the newborn B. Journal of nutrition, 1987, 57; 185-193.

4. Dop MC, et al. Anemia during pregnancy in Lome (Togo); prevalence, risk factors and repercussions for the neonates Revue d epidemiologic et de sante publique 1992.4; 259-267.

5. Fleming AF. Hematological disease in the tropics In: Cook GC ed. Manson's tropical disease 20th ed, London, Saunders, 1995: 101-173.

6. Nutritional anemia, Report of a WHO group of experts. Geneva, WHO 1972 (WHO technical report series No 501

7. Gjorup T, Bugge PM, Hendrickson C, Jensen AM. A critical evaluation of the clinical diagnosis of anemia Am J epidemiol 1986; 124-657 Stone JE et al. An evaluation of methods for screening for anemia. Bulletin WHO 1989; 62; 115-120.

8. Perry GS, Byers T, and Yip R et al. Iron nutrition does not account for the hemoglobin differences between blacks and whites. J Nutr 1992; 12:1417.

9. Hillman R S, Ault K A. Clinical approach to anemia. In: Hematology in clinical practice, Mc Graw-Hill, New York p.29.

10. Hillman RS, Ault KA. Normal erythropoiesis. In: Hematology in clinical practice. Mc Graw – Hill, New York, p.3. Steensma DP, Hoyer, JD, Fairbanks. Hereditary RBC disorder in middle Easter patients Mayo Clinic Proc 2001; 76; 285.

11. Morris MW, Williams WL, Nelson DN. Automated blood cell counting. In: Williams Hematology, 5th ed, Beutler E, Lichtman MA, Coller BS, et al. Mc Graw-Hill New York 1995: p. 13.

12. Van den Broek NR. Anemia in pregnancy in developing countries Reviews.British Jour of obst and gyn, 1998, 105: 385-390.

13. Fleming AF. Anemia in pregnancy in tropical Africa Transactions of the royal Soc of Trop Med and Hyg 1989; 83: 441-448.

14. Nardore DA, Roth KM, Mazur DJ, et al. Usefulness of physical examination in detecting the presence or absence of anemia. Arch Intern Med 1990; 150 –201.

15. Mohanads N, Schrier SL. Mechanism of red cell destruction in hemolytic anemia. In: the hereditary hemolytic anemia. Mentzer WC, Wagner GM (Edn), Churchill Living stone, New York, 1955: p; 13.

16. Howell JT, Monto RW.Syndrome of anemia, dysphagia and glossitis. N Engle L Med 1953; 249; 1009.

17. Crosby WH. Physiology & pathology of the iron metabolism Hosp. Pract 1990: 2627

18. Osaki T, Ueta E, Arisawa K et al. The pathophysiology of glossal pain in-patient with ID and anemia Am J Med Sick 1999; 318- 324 16.

19. Crosby WH, Whatever's become of chlorosis JAMA 1987; 257; 2799.

20. Reynolds RD, Binder HJ, Miller MB et al. Pagophagia and ID anemia. Ann Intern Med 1968; 69: 435.

21. Rector WG. Pica its frequency and significance in patients with IDA due to gastrointestinal blood loss J Gen Intern Med 1989; 4: 512.

22. Tunnessea WW, Smith C, Oski FA. Beeturia; a sign of ID. Am J Dis Child 1969; 117: 424.

23. Bridges KR, Seligman PA.Disorders of iron metabolism. In; Blood; principle& practice of hematology, Hardin RL, Luk SE, Stossel TP. (Eds) 1995 chap 49.

24. Vanden Brook NR, Letsky EA, White SA, Shenkin A. Iron status in pregnant anemia; which measurement is valid! Br J Haematol 1998; 103:817.

25. Guyatt GH, Oxman AD, Ali M, et al. Laboratory diagnosis of iron-deficiency anemia; an overview. J Gen Intern Med 1992; 7:145.

26. Hansen TM, Hansen NE. Serum ferritin as indicator of iron responsive anemia in patients with rheumatoid arthritis. Ann Rheum Dis 1986; 103:817.

27. Brittenham GM. Disorder of iron metabolisms; IDand over load. In; hematology basic principle and practice, 2nd, Hoffman, R, Benz EJ Jr Shattil SJ et al Eds Churchill Livingstone. New York 1995: 29.

28. Mc-Mahon LF Jr, Ryan MJ, Larson D, Fisher RL. Occult gastrointestinal blood loss in marathon runners. Ann Intern Med 1984; 100:846..

29. Cook JD, Skikne BS. Iron deficiency, definition and diagnosis. J. Intern Med 1989; 226: 349.

30. Finch CA, Bellotti V, Stray S, et al. Plasma ferritin determination as a diagnostic tool. West J Med 1986; 145:657.

31.Zanella A, Gruidelli L, Berzuini A, et al. Sensitivity and predictive values of serum ferritin and free erythrocyte protoporphyrin for ID. J Lab Clin Med. 1989:113-73.

32. Hallberg L, Bengtsson C, Lapidus L, et al. screening for iron deficiency: An analysis based on bone-marrow examinations and serum ferritin determination in population sample of women. Br J Haematol 1993; 85: 787.

33. Van Lupberger, W et al Hemoglobin measurement; the reliability of some simple

technique for use in a primary health care setting Bulletin of the WHO 1983; 61:957-65.
34. Kegels G, et al. Hemoglobin and packed cell volume measurement; the reliability of some simple techniques for use in surveys on rural hospitals. Annals des societies Belges de medicine tropical 1984; 64: 413-714.
35. Ghosh S, Mohan M. Screening for anemia. L lancet, 1989; 1: 823
36. Neville RG Evaluation of portable hemoglobin meter in general practice. BMJ 1987; 294: 1263-65.
37. Stott GJ, Lewis SM A simple & reliable method for estimating hemoglobin. Bulletin of the WHO 1995; 73: 369-77
38. Fairbanks VE. Laboratory testing for iron status. Hospi Pract. 1990; 20: 17.
39. Strochbach et al The value of the physical examination in the diagnosis of anemia. Archives of internal medicine. 1988; 148: 831-32.
40. Lok CN, Loh TT. Regulation of transferring function and expression: Review and update. Boil Signals recept 1998; 7:157.
41. Cook, JD. Clinical evaluation of iron deficiency. Semin Haematol 1982; 19: 6.
42. Finch CA, Huebers H. Perspectives in iron metabolism. N Engl J Med 1982; 19:6.
43. Guyatt GH, Patterson C, Ali M, et al. Diagnosis of IDA in the elderly. Am J Med 1990; 88:205.
44. Moda N, Cousens S, Kanki B. Anemia among women of reproductive age in Burkina Faso Word health foram 1996; 17: 369-72.
45. Steer P et al Relation between maternal hemoglobin concentration and birth weight in deferent ethnic groups B M J 1993; 310: 489-91.
46. Williams WJ, Morris MW, Nelson DA. Examination of the blood. In' Williams hematology, 5th ed Beutler E, Liechtman MA, Coller BS et al (Eds) Mc Graww-Hill, New York, 1995; p: 8.
47. Sanchez– Carrillo CL, et al Test on anon-invasive instrument for measuring hemoglobin concentration. International Journal of technical assessment in health care, 1989; 5: 659-67.
48. Looker, AC, Dolmen, PR, Carroll, MD et al. Prevalence of IDA in the united state JAMA 1997; 227-973.
49. Rockey DC, Cello JP. Evaluation of the gastrointestinal tract in patients with ID anemia. N Engl J Med 1993; 329:1691.
50. Dacie JV, Lewis SM. Practical hematology. Ninth edition.p115-127 by Churchill Livingstone, London 2001