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ORIGINAL ARTICLE

Prevalence of iron deficiency and its association with vitamin A deficiency in preschool children

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Abstract

Objectives: To identify the prevalence of iron deficiency in the population studied, as well as verifying if such deprivation is associated with vitamin A deficiency.

Methods: One hundred seventy-nine children, $\geq$ 24 months and < 72 months of age, with no diarrhea and/or fever at collection were studied. Vitamin A deficiency identification was carried out through serum 30-day dose-response test. Samples of peripheral blood from fasting children was obtained for hemoglobin counts, serum iron, and unsaturated iron binding capacity assays. Information about the presence of diarrhea and/or fever during the 15 days preceding the study was also obtained.

Results: 35.8% (64/179) of the children presented iron deficiency and 75.4% (135/179), vitamin A deficiency. 29.1% (52/179) of the children presented both iron and vitamin A deficiencies. Iron deficiency was not associated with vitamin A deficiency. A separate analysis for each hematimetric index also demonstrated no significant difference between children with or without vitamin A deficiency. Children aged 24 to 36 months presented significantly higher prevalence rates of iron deficiency ($p = 0.0005$) as did children with diarrhea and/or fever during the 15 days preceding the study ($p = 0.003$).

Conclusions: Although iron deficiency was not associated with vitamin A deficiency, high rates of both deficiencies were exhibited in a "healthy" population with low malnutrition indices. Such situations are known as "hidden hunger". Younger children presented a higher risk of iron deficiency as did children with diarrhea and/or fever during the 15 days preceding the study.

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Introduction

In the last few years, micronutrient deficiency (vitamins and minerals) has become an important public health problem comparatively to macronutrient deficiency (proteins, carbohydrates, and fat). This has drawn the attention of health authorities and professionals on a worldwide basis. National and international studies about the deficiency of trace elements have highlighted the occurrence of iron deficiency (ID) in infants,[1,2] especially the one expressed as iron-deficiency anemia, and vitamin deficiency, mainly vitamin A in preschool children.[3] However, ID in preschool children[4] and vitamin A deficiency (VAD) in infants[1] have also been described. Most of these deficiencies are subclinical, hence the nomenclature "hidden hunger."

ID can be present in its subclinical form – when only the erythrocyte indices which indirectly indicate micronutrient reserves, are low, without reduction of hemoglobin levels – or in the form of clinical anemia – with variable reductions

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of hemoglobin levels.[5] However, the presence of infections (even if not severe) or inflammations can interfere with the interpretation of lab tests, increasing the number of children considered to be anemic in a given population.[6]

Micronutrient deficiencies can be extremely detrimental to infant and child health. Children with ID, even those without clinical anemia, may have behavioral and cognitive disorders and have their growth affected,[7,8] in addition to having fatigue and weakness from the inadequate use of energy by the muscles. In its turn, VAD, even in its subclinical form, causes an increase in infant morbidity and mortality, due to the larger number of cases of respiratory infection and to the increased severity of cases of diarrhea. In extreme cases, VAD can cause blindness, due to irreversible damage to the cornea.[9] In addition, VAD can cause anemia as well.[10]

Many children simultaneously have deficiencies of different micronutrients.[11] Thus, it is possible to infer that when children show deficiency of one micronutrient, they may as well have concomitant deficiency of other micronutrients.

Due to the relatively common occurrence of VAD among preschool children, this study was carried out to assess the prevalence of this deficiency in a population of children treated in a general pediatric outpatient service, accredited with the Unified Health System, and located in the outskirts of Ribeirão Preto.[3]

This study also sought to determine the prevalence of ID, and to verify whether ID was associated with VAD in the same population.

Patients and methods

From September 1999 to September 2000, children aged between two years complete and six years incomplete treated in a Unified Health System unit (*Centro Médico Social Comunitário de Vila Lobato*) in the outskirts of Ribeirão Preto, were invited, at the time of their systematic evaluation visit, to participate in the study. Parents or surrogates signed an informed consent form, in which they allowed the inclusion of their children in the study. The average per capita family income was approximately US$ 120/month (median: US$ 86/month).[3]

For identification of VAD, the serum 30-day dose response test (+S30DR) was used. The +S30DR consists of the collection of a blood sample for quantification of serum retinol levels immediately before (T_0) oral supplementation with 200,000 IU of retinyl palmitate, and of the collection of a blood sample to the same quantification, 30 to 45 days after (T_1), the referred supplementation. For calculation of +S30DR, we used the formula (T_1-T_0/T_1) x 100. Individual results $\geq$ 20% indicate low hepatic reserves of vitamin A.[12] This test seeks to reduce the biases related to the classification of a child as VAD carrier or noncarrier, which may change due to different situations (e.g.: inflammatory process), by quantifying retinol levels.[13] After at least a six-hour fasting period and before vitamin A supplementation, peripheral blood samples were collected from the selected children, for quantification of plasma/serum hemoglobin levels, unsaturated iron-binding capacity (UIBC), and iron and retinol levels before supplementation. Blood samples were not collected from children who presented with diarrhea and/or fever episodes.

Blood for hemoglobin quantification was collected in Vacutainer® tubes containing EDTA and then sent to the Laboratory of Hematology of Hospital das Clínicas (affiliated with the School of Medicine of Universidade de São Paulo (HCFMRP-USP)). There, blood was processed by an automatic counter (Coulter® T890 or STKS). For serum iron and UIBC quantification, we used an atomic absorption system (Cobas Integra - Roche®). The collected blood was placed in Vacutainer® tubes without anticoagulant and, then sent to the Central Laboratory of HCFMRP-USP. The samples were centrifuged at 2,500 rpm for 15 minutes to separate the serum from other blood cell elements. Iron quantification was based on the Guanidine/FerroZine® method. For UIBC quantification, direct determination with FerroZine® was used.

ID was considered for all children aged up to 5 years with hemoglobin levels < 11 g/dl and all children aged between 5 and 6 years with hemoglobin < 11.5 g/dl or with serum iron levels < 50 µg%, or with transferrin saturation < 0.16 (16%), or with UIBC > 346 µg/dl. All children considered to have anemia or ID according to the previous definitions were treated with an oral preparation of ferrous sulfate.

Parents or surrogates were asked whether their children had had fever (axillary temperature > 37 °C checked with a thermometer) or diarrhea (three or more bowel movements with watery stools or any amount of watery stools containing blood visible to the naked eye within 24 hours) 15 days before joining the study group.

Children's weight and height was measured at the time participants joined the study. These data were used to obtain z scores for the weight/age (w/a) and height/age (h/a) ratios, compared with the National Center for Health Statistics (NCHS) growth curves.

The chi-square test (χ^2) was performed to assess the possible association of ID, of serum iron levels, and of transferrin saturation with VAD, in addition to assessing the differences between ID prevalence rates between genders, different ages, and children with episodes of fever or diarrhea in the last 15 days before the study. The test was also used to verify the difference between the number of fever and diarrhea episodes between age groups 15 days prior to the study. Fisher's exact test was used for determination of hemoglobin and UIBC levels, for the establishment of a possible association with VAD. We used Pearson's correlation coefficient to establish the correlation between the levels of hemoglobin, serum iron and UIBC, and transferrin saturation with serum retinol levels before supplementation.

An alpha value of 0.05 (5%) was set for all statistical analyses.

The study protocol was approved by the ethics committee of HCFMRP-USP (HCRP n° 4016/99).

Results

A total of 188 children were assessed in order to determine the prevalence of VAD using the +S30DR test. Of these, 74.5% (140/188) showed results compatible with VAD, but no children had xerophthalmia.[3] From the initial population, at least one erythrocyte index was obtained in 186 children; due to lack of material, we could not obtain these indices in two patients. Seven children were excluded from the analysis because their test results indicated they had hemoglobinopathies. Thus, 179 children were evaluated for ID. Of analyzed children, 55.3% (99/179) were male and 44.7% (80/179) were female. No difference was observed between the prevalence of ID between male and female patients (p = 0.15; Table 1).

According to the definitions used, ID was found in 35% (64/179) of the children. Such deficiency was not associated with VAD in the study population (p = 0.18; Table 2). Even when each erythrocyte index (hemoglobin, serum iron, UIBC and transferrin saturation) was analyzed separately, there was no statistically significant difference between children with or without VAD, as shown in Table 2. There was concomitant occurrence of VAD and ID in 29% (52/179) of the children.

The means, medians, standard deviations, first and third quartiles and minimum and maximum values observed in the levels of hemoglobin, serum iron, UIBC, transferrin saturation and serum retinol levels before supplementation in the study population are presented in Table 3.

In order to the effect of age on the prevalence of ID, the population was divided into four age groups, as follows: from 24 months complete to 36 months incomplete (group A), from 36 months complete to 48 months incomplete (group B), from 48 months complete to 60 months incomplete (group C) and, finally, from 60 months complete to 72 months incomplete (group D). Group A showed an ID prevalence significantly higher than the other groups (p = 0.0005; Table 4). Even when Group A is compared to older groups (groups B,C and D) together, ID prevalence remains statistically significant (p = 0.0002). There was no significant difference in the ID prevalence rates between groups B, C and D (p = 0.18). There was also no significant difference in the ID prevalence rates in the group A as for sex (p = 0.35; data not showed).

It was observed that 38.4% (58/179) of the children had episodes of fever and diarrhea during the 15 days that preceded the study. Among the children with episodes of fever and diarrhea, 51.7% (30/58) presented ID, and 28.1% (34/121) did not. This difference was statistically significant (p = 0.003; Table 5). It was also observed that 51.6% (32/64) of the children aged < 36 months had had previous episodes of fever or diarrhea, as well as 23.1% of the children aged $\geq$ 36 months; this difference was statistically significant (p < 0.001).

There was no correlation between pre-supplementation serum retinol concentrations and hemoglobin levels (r = 0.05), serum iron (r = 0.23), UIBC (r = -0.02) and transferrin saturation (r = 0.19).

Table 1 - Distribution of iron deficiency according to sex in infants from 24 months complete and 71 months and 29 days referred to the *Centro Médico Social Comunitário of Vila Lobato*, Ribeirão Preto (1999-2000)

Sex	With ID (%)	Withou ID (%)	Total (%)
Male	40 * (40.4)	59 (59.6)	99 (100.0)
Female	24 (30.0)	56 (70.0)	80 (100.0)
Total	64 (35.8)	115 (64.2)	179 (100.0)

ID = iron deficiency.
* (χ^2; p = 0.15).

Table 2 - Distribution of iron deficiency and hematimetric indexes according to vitamin A deficiency levels in infants from 24 months complete to 71 months and 29 days referred do the *Centro Médico Social Comunitário from Vila Lobato*, Ribeirão Preto (1999-2000)

Variable	With VAD	%	Without VAD	%	p
With iron deficiency	52	38.5	12	27.3	0.18 *
Without iron deficiency	83	61.5	32	72.7	
Hemoglobin † < 11 g/dl	9	6.3	1	2.4	0.45 ‡
Hemoglobin $\geq$ 11 g/dl	118	93.7	40	97.6	
Serum iron < 50 µg%	36	27.7	8	20.5	0.37 *
Serum iron $\geq$ 50 µg%	94	72.3	31	79.5	
UIBC > 346 µg/dl	24	18.3	4	9.1	0.23 †
UIBC $\leq$ 346 µg/dl	107	81.7	40	90.9	
Transferrin saturation < 16%	43	33.3	12	30.0	0.69 *
Transferrin saturation $\geq$ 16%	86	66.7	28	70.0	

VAD = vitamin A deficiency; UIBC = unsaturated iron-binding capacity.
* χ^2.
† (hemoglobin < 11.5 g/dl in children between 5 and 6 years-old).
‡ Fisher's exact test.

Table 3 - Mean, median, standard deviation, first and third quartiles and minimum and maximum levels of hemoglobin, serum iron, total iron binding capacity, transferrin and serum retinol saturation (before supplementation) in infants from 24 months complete to 71 months and 29 days referred to the *Centro Médico Social Comunitário from Vila Lobato*, Ribeirão Preto (1999-2000)

Hematimetric index	Mean/median values (standard deviation) (First quartile - third quartile) [minimum value - maximum value]
Hemoglobin (g/dl) n = 168	12.3/12.3 (1.02) (11.7 – 12.9) [8.1 – 15.0]
Serum iron (µg%) n = 172	80.5/71 (47.2) (49 – 97) [4 – 255]
UIBC (µg%) n = 175	299.3/294 (61.6) (256.5 – 333.5) [160 – 523]
Transferrin saturation (%) n =171	21.1/19.1 (10.8) (13.4 – 27.8) [1.2 – 55.3]
Serum retinol before supplementation (µmol/l) n = 179	0.91/0.88 (0.42) (0.61 – 1.16) [0.13 – 2.92]

UIBC = unsaturated iron binding capacity.

In general, seven children presented some degree of malnutrition. Three children presented z scores < -2 standard deviation (SD) from NCHS reference values for the W/H ratio (wasting), 2 for the W/A ratio (underweight), one child for H/A and W/A ratios (stunting/underweight) and, finally, one child for W/H and W/A ratios (wasting/underweight).

Discussion

This study aimed at establishing the prevalence of ID and its association with VAD in a group of preschool children treated in a general pediatric outpatient service. Only 3.9% (7/179) of the children had malnutrition. However, a large prevalence of children with ID (35.8%; 64/179) was observed. High prevalence rates for micronutrient deficiency in population with a small rate of malnutrition had already been observed in other studies,[1] characterizing what is known as hidden hunger. On the other hand, some studies observed higher prevalence rates of malnutrition in populations with multiple micronutrient deficiencies. Recently, Castejón et al.,[14] after studying Venezuelan children aged between 2 to 7 years, found a prevalence rate of 38.1% for anemia and 21.8% for VAD; the same study showed a prevalence of 14.4% for stunting and 9.4% for underweight. The mean hemoglobin levels (12.3 g/dl) in our population were

Table 4 - Distribuition of iron deficiency in different age ranges in infants from 24 months complete to 71 months and 29 days referred to the *Centro Médico Social Comunitário from Vila Lobato*, Ribeirão Preto (1999-2000)

Age range (months)	With ID (%)	Without ID (%)	Total (%)
≥ 24 and < 36	34 (54.8) *	28 (45.2)	62 (100.0)
≥ 36 and < 48	15 (34.9)	28 (65.1)	43 (100.0)
≥ 48 and < 60	7 (17.5)	33 (82.5)	40 (100.0)
≥ 60 and < 72	8 (23.5)	26 (76.5)	34 (100.0)
Total (%)	64 (35.8)	115 (64.2)	179 (100.0)

ID = iron deficiency.
* (χ^2; p = 0.0005).

Table 5 - Distribution of iron deficiency in infants from 24 months complete to 71 months and 29 days, with or without episodes of fever and diarrhea during the 15 days that preceded the study, referred to the *Centro Médico Social Comunitário from Vila Lobato*, Ribeirão Preto (1999-2000)

Fever and diarrhea during the 15 days that preceded the study	With ID (%)	Without ID (%)	Total (%)
Yes	30 * (51.7)	28 (48.3)	58 (100.0)
No	34 (28.1)	87 (71.9)	121 (100.0)
Total	64 (35.8)	115 (64.2)	179 (100.0)

ID = iron deficiency.
* (χ^2; p = 0.003)

higher than those found in similarly-aged children from the State of Pernambuco (11.4 g/dl) in a recent study.[15]

The prevalence of ID observed in our study was much smaller than the previous one found for healthy infants in the same population (75.6%).[1] Lower iron requirements in the studied age group, in this study, can help explain such a difference.[16] Such phenomenon also helps explain the significantly higher prevalence in groups of children aged between 24 and 36 months in our study, as De Almeida et al.[4] observed in preschool children in Pontal. Furthermore, children aged between 24 and 36 months significantly presented episodes of fever or diarrhea at a higher frequency than children of other ages. As previously mentioned, the presence of recent infections, even if not severe, produces abnormal laboratory findings that may take up to one month to normalize, and may overestimate the prevalence of anemia.[6] Thus, other data that could partly explain the high ID prevalence are that 32.4% (58/179) of the children had episodes of fever and diarrhea up to 15 days before joining the study,[3] although they were healthy at the moment they joined the study. As previously mentioned in other studies,[17] – especially on fever - , it was observed that children with episodes of fever or diarrhea significantly have a higher frequency of ID. Moreover, in our study, almost 50% of the children with ID (46.9% - 30/64) had episodes of fever or diarrhea 15 days before joining the study.

In addition, VAD was found in 75.4% (135/179) of the population; the concomitant occurrence of ID and VAD amounted to 29.1% (52/179), a very similar rate found by Palafox et al.[11] in a study carried out in the Marshall Islands (33.2%). Looking only at the group with ID, it is possible to notice that 81.2% (52/64) also presented VAD. These observations contribute to the hypothesis that micronutrient deficiency does not occur in isolation, but along with several other deficiencies – especially vitamin A deficiency and iron deficiency – as shown in some studies, especially in developing countries.[11,18,19].

Despite the high prevalence of ID and VAD in our population, we did not find any correlation between these deficiencies. One possible explanation to this finding it that, whereas younger children showed a significantly higher prevalence of ID, VAD was evenly distributed in all age groups (data not shown).[3]

Of 168 children, hemoglobin levels were lower than the values established as appropriate for age in only 10 (6.0%) cases, which shows that anemia was found in only a small number of children. We may thus infer that most of these children may not be anemic on clinical examination, for having subclinical ID. The same occurred with VAD: all the cases identified in the population were subclinical, because no children showed signs of xerophthalmia.[3] However, several studies involving populations with high ID prevalence rates also found high prevalence of anemia.[4,11,20,21] The probable explanation to this finding lies in the fact that the analyzed children are at preschool age, and are therefore at a lower risk for iron-deficiency anemia compared to infants. This hypothesis is strengthened by the fact that 60% of the children (6/10) who had hemoglobin levels lower than normal for their age fall between the ages of 24 months complete and 36 months incomplete.

Although some studies show a correlation between some erythrocyte indices (especially hemoglobin levels) and serum retinol levels,[22,23] this was not observed in our study. This probably occurred due to the fact that there were few children with hemoglobin levels lower than normal, whereas 32.4% of the children had low serum retinol levels before supplementation.[3]

Our study has a limitation as far as the data described above are concerned. All children who had at least one erythrocyte index were included in the study; thus, children with normal hemoglobin levels, but whose serum iron or UBIC was not quantified due to lack of material, were classified as normal, and then prevalence rates may have been underestimated.

Some data may help explain the observed prevalence rates. Several studies showed that nutritional factors are the major risk factors for ID.[16,24] Among these factors, the low intake of iron-rich foods with high bioavailability (e.g.: red meats) and the high intake of cow’s milk were associated with ID.[25,26] Although there are no nutritional surveys in this study, we may infer that the diet of the analyzed population is not as required, especially because of the high prevalence of VAD observed.[3] With regard to VAD, the most probable hypothesis to explain such prevalence in the analyzed population is that children of this age have a low intake of vitamin-A-rich foods, as observed in other populations with VAD.[27] Additionally, the large number of children with episodes of fever or diarrhea prior to the study may have also contributed to the high prevalence of VAD.[3,13]

Although this is not within the scope of our study, we considered that, despite the fact that ID does not occur exclusively in underprivileged social classes,[16] low socioeconomic conditions of the studied group (per capita income of US$ 120/month)[3] helped to explain such a deficiency.[5,16,28,29] Low educational levels and, consequently, low wages and poor living conditions expose children to an unfavorable environment, preventing them from having access to information and from having a balanced diet, in addition to increasing the risks for infections that may aggravate ID. Iron deficiency can be managed with efficient measures in the short, medium and long term.[5,16,30] In the short-term, regular supplementation with iron salts, either as medication or as fortified food – as successfully done in some countries – constitutes an efficient measure to eliminate this problem. Measures that may have a medium to long-term effect include training and reskilling programs for health professionals, allowing for the early identification of these problems, reduction of fever or diarrhea through good vaccination coverage and the use of anthelmintics, whenever necessary, breastfeeding promotion, improvement of child care follow up and, finally, improvement of the quality of life of the population.[28,30] Such measures may also have an effect on the deficiency of other micronutrients, including VAD.[3]

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References

1. Ferraz IS, Daneluzzi JC, Vannucchi H. Vitamin A deficiency in children aged 6 to 24 months in São Paulo State, Brazil. Nut Res. 2000;20:757-68.
2. Hadler MCCM, Juliano Y, Sigulem DM. Anemia do lactente: etiologia e prevalência. J Pediatr (Rio J). 2002;78:321-6.
3. Ferraz IS, Daneluzzi JC, Vannucchi H, Jordão Jr. AA, Ricco RG, Del Ciampo LA, et al Detection of vitamin A deficiency in Brazilian preschool children using the serum 30-day dose-response test. Eur J Clin Nutr. 2004;58:1372-7.
4. De Almeida CA, Ricco RG, Del Ciampo LA, Souza AM, Pinho AP, Dutra-de-Oliveira JE. Factors associated with iron deficiency anemia in Brazilian preschool children. J Pediatr (Rio J). 2004;80:229-34.
5. Centers for Disease Control and Prevention. Recommendations to prevent and control iron deficiency in the United States. MMWR. 1998;47(RR-3):1-29.
6. Dallman PR, Yip R. Changing characteristics of childhood anemia. J Pediatr. 1989;114:161-4.
7. Walter T, De Andraca I, Chahud P, Perales CG. Iron deficiency anemia: adverse effects on infant psychomotor development. Pediatrics. 1989;84:7-17.
8. Lozoff B, Jimenez E, Hagen J, Mollen E, Wolf AW. Poorer behavioral and developmental outcome more 10 years after treatment for iron deficiency in infancy. Pediatrics. 2000;105:E51.
9. Sommer A. Xerophtalmia and vitamin A status. Prog Retinal and Eye Res. 1998;17:9-31.
10. Semba RD, Bloem MW. The anemia of vitamin A deficiency: epidemiology and pathogenesis. Eur J Clin Nutr. 2002;56:271-81.
11. Palafox NA, Gamble MV, Dancheck B, Ricks MO, Briand K, Semba RD. Vitamin A deficiency, iron deficiency, and anemia among preschool children in the Republic of the Marshall Islands. Nutrition. 2003;19:405-8.
12. World Health Organization (1996): Indicators for assessing Vitamin A Deficiency and their application in monitoring and evaluating intervention programmes: report of a joint. Geneva, WHO/UNICEF, (Micronutrient series). WHO/NUT/96.10.
13. Turham DI, McCabe GP, Northrop-Clewes CA, Nestel P. Effects of subclinical infection on plasma retinol concentrations and assessment of prevalence of vitamin A deficiency: meta-analysis. Lancet. 2003;362:2052-8.
14. Castejón HV, Ortega P, Amaya D, Gómez G, Leal J, Castejón OJ. Co-existence of anemia, vitamin A deficiency and growth retardation among children 24-84 months old in Maracaibo, Venezuela. Nutr Neurosci. 2004;7:113-9.
15. Osório MM, Lira PI, Batista-Filho M, Ashworth A. Prevalence of anemia in children 6-59 months old in the state of Pernambuco, Brazil. Rev Panam Salu Publica. 2001;10:101-7.
16. Osório MM. Fatores determinanntes da anemia em crianças. J Pediatr (Rio J). 2002;78:269-78.
17. Jansson LT, Kling S, Dallman PR. Anemia in children with acute infections seen in a primary care pediatric outpatient clinic. Pediatr Infect Dis. 1986;5:424-7.
18. Bloem MW. Interdependence of vitamin A and iron: an important association for programmes of anaemia control. Proc Nutr Soc. 1995;54:501-8.
19. Yamamura CM, Sullivan KM, van der Haar F, Auerbach SB, Iohp KK. Risk factors for vitamin A deficiency among preschool children in Pohnpei, Federated States of Micronesia. J Trop Pediatr. 2004;50:16-9.
20. Kemmer TM, Bovill ME, Kongsomboon W, Hansch SJ, Geisler KL, Cheney C, et al. Iron deficiency is unacceptably high in refugee children from Burma. J Nutr. 2003;133:4143-9.
21. Hashizume M, Kunii O, Sasaki S, Shimoda T, Wakai S, Mazhitova Z, et al. Anemia and iron deficiency among schoolchildren in the Aral sea region, Kazakhstan. J Trop Pediatr. 2003;49:172-7.
22. Majia LA, Hodges RE, Arroyave G, Viteri F, Torun B. Vitamin A deficiency and anemia in Central American children. Am J Clin Nutr. 1977;30:1175-84.
23. Molla A, Khurshid M, Molla AM, Badruddin SH, Hendricks K, Snyder JD. Is anemia an accurate predictor of vitamin A status in Pakistani children? Am J Trop Med Hyg. 1993;49:276-9.
24. Szarfarc SC, de Souza SB. Prevalence and risk factors in iron deficiency and anemia. Arch Latinoam Nutr. 1997;47(2 Suppl 1):35-8.
25. Thane CW, Walmsley CM, Bates CJ, Prentice A, Cole TJ. Risk factors for poor iron status in British toddlers: further analysis of data from the National Diet and Nutrition Survey of children aged 1.5-4.5 years. Public Health Nutr. 2000;3:433-40.
26. Sandoval C, Berger E, Ozkaynak MF, Tugal O, Jayabose S. Severe iron deficiency anemia in 42 pediatric patients. Pediatr Hematol Oncol. 2002;19:157-61.
27. Miller M, Humphrey J, Johnson E, Marinda E, Brookmeyer R, Katz J. Why do children become vitamin A deficient? J Nutr. 2002;132 Suppl:S2867-80.
28. Adish AA, Esrey AS, Gyorkos TW, Johns T. Risk factors for iron deficiency anaemia in preschool children in northern Ethiopia. Public Health Nutr. 1999;2:243-52.
29. Frith-Terhune AL, Cogswell ME, Khan LK, Will JC, Ramakrishnan U. Iron deficiency anemia: higher prevalence in Mexican American than in non-Hispanic white females in the third National Health and Nutrition Examination Survey, 1988-1994. Am J Clin Nutr. 2000;72:963-8.
30. Ahluwalia N. Intervention strategies for improving iron status of young children and adolescents in India. Nutr Rev. 2002;60(5 pt 2 Suppl):S115-117.

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