Iron Status of One-Year-Old Infants in a Well Baby Clinic

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Abstract

Seventy-two healthy infants (37 males, 35 females) attending a private well baby clinic were enrolled in the study. Their mean birthweights and body weights at one year of age were 3,079 grams and 10 kilograms, respectively. Blood samples were drawn approximately on their first birthday for evaluating the iron status. Complete blood count, hemoglobin (Hb) typing and DNA analysis for common carrier status of thalassemia and hemoglobinopathis were also determined. According to the infants of serum ferritin, the patients were classified into 4 groups: group 1, iron deficiency anemia (Hb <11 g/dl and ferritin <12 ng/L) in 1 infants (1.4%); group 2, iron deficiency without anemia (Hb >11 g/dl and ferritin <12 ng/L) in 5 infants (6.9%); group 3, borderline iron depletion (ferritin 12-30 ng/L) in 39 infants (54.2%); group 4, iron sufficiency (ferritin >30 ng/L) in 27 infants (37.5%). The iron deficiency state emerged as 8.3 per cent (6/72). There was no significant difference of levels of Hb and mean corpuscular volume (MCV) among the infants with iron deficiency without anemia, borderline iron depletion and iron sufficiency.

The results also revealed that 25 out of 72 (34.7%) infants were carriers of thalassemia and hemoglobinopathies. The carrier infants had significant lower Hb and MCV than those of the non-carrier infants with the p-values of 0.004 and 0.000, respectively; while their serum ferritin levels were not significantly different. Additionally, the association of carrier and iron deficiency state was further evaluated. The Hb and MCV among carrier infants with and without iron deficiency were not significantly different. Six infants with carrier state were found to have slightly decreased levels of Hb ranging from 10.3 to 10.9 g/dl with the ferritin ranging from 18.7 to 382.9 ng/L while the remainders had Hb of >11 g/dl. Therefore, 7 out of 72 (9.2%) infants had anemia (Hb <11 g/dl) which was caused by the carrier state of thalassemia and hemoglobinopathies (n=6) and iron deficiency anemia (n=1).

The risk factors of iron deficiency status were associated with feeding regimen including continuation of breast feeding until one year of age without adequate haem iron supplement, exclusive

formula feeding, inadequacy of solid food supplement with only one meal per day and excluding haem iron from animal liver without substitution. The infants with risk factors had significantly lower levels of serum ferritin (mean 14.1 ± 1.7 ng/L) than those without risk factors (mean 31.9 ± 1.9 ng/L) with a p-value of 0.000.

In conclusion, adequate haem iron supplement in 3 meals of solid food is essential for the prevention of iron deficiency status in one-year-old infants.

Key word : Iron Deficiency Anemia, Iron Status in Infants

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Iron deficiency anemia is still the most common nutritional deficiency worldwide and affects two-thirds of children in most developing countries (1). A recent national study in England showed that 12 per cent of 2-year-old children were anemic, rising to 21 per cent in Asian immigrants(2). In Thailand, 21 per cent of 2-year-old children were found to be anemic in 1989(3) and decreased to 15 per cent among children younger than 6 years old in 1996(4).

Iron is an essential component for brain growth. It is needed during the early and rapid development of the brain and different cells within the brain continue to need iron for many ongoing processes especially in the infancy period(5). In humans, persistent cognitive impairment was reported among children 5 to 6 years of age who were anemic during the infancy period although their hematological parameters were corrected(6). Since these changes may be irreversible, the most practical prevention of iron deficiency anemia is to provide adequate iron-fortified food or iron supplement.

This study presents the iron status of one-year-old infants attending a private well baby clinic. The carrier state of thalassemia and hemoglobinopathies was also determined since they also presented with normal or subnormal levels of hemoglobin. Moreover, the prevalence of carrier state among the Thai population is extremely high at 30-40 per cent. Therefore, a complete hematologic study will be helpful in determining the cause of anemia among the studied infants.

SUBJECTS AND METHOD

Subjects

Seventy-two healthy infants, who attended a well baby clinic at Pativej Pahonyothin Private Clinic, were enrolled in the study at the age of one year. After obtaining informed consent from their parents, 5 ml of venous blood was drawn from a peripheral vein at approximately on their first birthday. All of them were in good health without concurrent infection at the time of blood sampling. The parents and caregivers also provided details of milk and solid food given to the infants. Their physical examination and developmental evaluation were performed by one pediatrician (A.C).

Laboratory tests

The complete blood counts (CBC) were performed by Coulter based on electrical impedance. Serum ferritin was determined by microplate enzyme immunoassay described by the manufacturer(7). Hemoglobin (Hb) typing was assayed by high performance liquid chromatography using Variant TM from Biorad. DNA analysis of four common carriers of thalassemia and hemoglobinopathies including

alpha thalassemia-1 of Southeast Asian type ($--^{SEA}$), alpha thalassemia-2 of rightward ($\alpha^{3.7}$-) deletion and leftward ($\alpha^{4.2}$-) deletion and Hb Constant Spring ($\alpha^{cs}\alpha$), was determined.

Anemia was diagnosed by a level of hemoglobin of less than 11 g/dl. The iron statuses[8] were divided into 4 stages based on the levels of serum ferritin and hemoglobin including iron deficiency anemia (Hb <11 g/dl and ferritin <12 ng/L), iron deficiency without anemia (Hb >11 g/dl and ferritin <12 ng/L), borderline iron depletion (ferritin 12-30 ng/L) and iron sufficiency (ferritin >30 ng/L).

Statistics

The levels of serum ferritin were computed to be log 10 and expressed as geometric mean. The comparison between the mean of the groups of patients was calculated by unpaired *t*-test or one way ANOVA. A p-value of less than 0.05 was considered significant.

RESULTS

Seven-two healthy infants (37 males, 35 females) were enrolled in the study from 1999 to 2001. Fifty-nine infants were the first-born child while 10 and 3 infants were the second and third child, respectively. The route of delivery included normal labor, 45 infants; cesarean section, 17 infants; forceps extraction and vacuum extraction, one each and 8 infants with missing data. Their birthweight, age at enrollment, body weight, height and head circumference at one year of age are shown in Table 1. Sixty-five were normal term infants except for 3 with low birthweights of 2,160, 2,060 and 2,410 grams, 2 monozygotic twins of 2,160 grams and 2 large for gestational age of 4,040 and 4,170 grams. All of them had normal growth and development.

The infants were classified into 4 groups based upon the levels of serum ferritin as shown in Table 2: group 1, iron deficiency anemia (Hb <11 g/dl and ferritin <12 ng/L) included 1 infant (1.4%); group 2, iron deficiency without anemia (Hb >11 g/dl and ferritin <12 ng/L) included 5 infants (6.9%); group 3, borderline iron depletion (ferritin 12-30 ng/L) included 39 infants (54.2%); group 4, iron sufficiency (ferritin >30 ng/L) included 27 infants (37.5%). The iron deficiency state emerged as 8.3 per cent (6/72). The infant with iron deficiency anemia showed low MCV of 66.3 fl and hypochromic micorcytic red blood cells on the peripheral smear while the infants with iron deficiency without anemia showed a rather normal MCV and very few hypochromic microcytic red blood cells. Their clinical and laboratory findings are shown in Table 3. There was no significant difference of the levels of Hb and mean corpuscular volume (MCV) among the infants with iron deficiency without anemia, borderline iron depletion and iron sufficiency.

Twenty-five out of 72 (34.7%) infants were carriers of thalassemia and hemoglobinopathies which included alpha thalassemia-1 trait (n=2), alpha thalassemia-2 trait (n=9), Hb E trait (n=8), Hb CS trait (n=2), combined Hb E and alpha thalassemia-2 trait (n=3) and beta thalassemia trait (n=1). The mean ± SD of Hb and MCV of 25 carrier infants were significantly less than those of 47 non-carrier infants with the p-values of 0.004 and 0.000, respectively (Table 2). The low MCV and hypochromic microcytic red blood cells on the peripheral blood smear were commonly found in infants with beta thalassemia trait and alpha thalassemia-1 trait while infants with Hb E trait, Hb CS trait and alpha thalassemia-2 trait had a near normal levels of MCV and very few hypochromic microcytic red blood cells. The association of carrier and iron deficiency status was further evaluated. The Hb and MCV of carrier infants with and without iron deficiency were not significantly different. Six infants, 3 with borderline iron depletion and 3 with iron sufficiency, were found to have slightly decreased levels of Hb ranging from 10.3 to

Table 1. The descriptive data of the 72 studied infants.

	Mean ± SD	Range
Birthweight (g)	3,079 ± 459	2,060-4,170
Age (month)	12 ± 0.8	11.5-15
Body weight (kg) at 1 year old	10 ± 1.2	8.1-13.3
Height (cm) at 1 year old	75.6 ± 2.7	70-83.5
Head circumference (cm) at 1 year old	45.7 ± 1.4	42-48.5

Table 2. The levels of Hb, MCV and serum ferritin among 72 studied infants with different associated conditions.

Infants	Number	Hb (g/dl)		MCV (fl)		Ferritin (ng/L)	
		Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range
Total	72	12.2 ± 0.9	10.3-13.9	76.2 ± 6.1	53.8-91.3	26.9 ± 2.0	13.2-54.9
Iron status							
1) iron deficiency with anemia	1	10.5*		66.3*		4.7*	
2) iron deficiency without anemia	5	12.2 ± 0.9	11.1-13.2	74.3 ± 4.6	71.6-82.4	9.6 ± 1.2	7.9-11.7
3) borderline iron depletion	39	12.2 ± 0.9	10.5-13.5	75.8 ± 6.8	53.8-91.3	24.4 ± 1.3	16.0-25.9
4) iron sufficiency	27	12.2 ± 0.7	10.3-13.5	77.5 ± 5.0	61.8-85.8	52.2 ± 1.9	27.6-98.6
Carrier of thalassemia and hemoglobinopatheis							
1) carrier	25	11.8 ± 0.9**	10.3-13.6	71.5 ± 6.4**	53.8-82.7	29.4 ± 2.4	12.1-71.2
2) non-carrier	47	12.4 ± 0.8	10.5-13.9	78.7 ± 4.3	66.3-91.3	25.7 ± 1.8	13.9-47.3
Carrier of thalassemia and hemoglobinopatheis							
1) with iron deficiency but without anemia	2	12.8*, 11.6*		71.6*, 72.3*		9.1*, 10.8*	
2) with borderline iron depletion	16	11.7 ± 0.2	10.3-13.6	70.3 ± 1.3	53.8-80.7	21.1 ± 1.1	19.7-22.5
3) with iron sufficiency	7	11.8 ± 0.5	10.5-13.5	71.1 ± 2.2	61.8-76.8	91.9 ± 1.5	59.5-142.1
Feeding							
1) inadequate feeding	15	12.2 ± 0.9	10.5-13.9	76.9 ± 5.4	66.3-83.7	14.1 ± 1.7**	8.3-23.7
2) formula plus 2 meals/day	10	12.2 ± 1.1	10.5-13.9	72.2 ± 6.2	61.8-80.7	31.2 ± 1.7	18.3-53.2
3) formula plus 3 meals/day	47	12.2 ± 0.8	10.3-13.7	76.8 ± 6.1	53.8-91.3	32.1 ± 1.9	16.2-73.7

* individual value.

** statistically significant different from the comparable groups.

Table 3. Clinical and laboratory findings of iron deficiency anemia (case 1) and iron deficiency without anemia (cases 2-6).

Case	Sex	Birthweight (g)	Body weight at one year old (kg)	Milk	Solid food (meals)	Hb (g/dl)	MCV (fl)	Serum ferritin (ng/L)	Hb typing	DNA for alpha thalassemia & Hb Constant Spring
1	M	2,900	10.3	Breast	3[a]	10.5	66.3	4.7	Normal	αα/αα
2	M	2,890	10	Formula	3[b]	13.2	82.4	7.1	Normal	αα/αα
3	M	3,030	11.5	Formula	3[b]	11.8	72.9	11.9	Normal	αα/αα
4	F	3,000	9.85	Breast	1[a]	11.6	72.3	10.8	Hb E trait	αα/αα
5	F	2,160	10.3	Formula	1	12.8	71.6	9.1	Normal	$\alpha^{3.7}$-/αα
6	M	3,500	10	Breast	3[a]	11.1	72.1	9.8	Normal	αα/αα

a : little haem iron of pork, meat and liver were given.
b : no animal liver was given.

10.9 g/dl with the ferritin ranging from 18.7 to 382.9 ng/L. They comprised of alpha thalassemia-2 trait (n=2) and one each of beta thalassemia trait, Hb E trait, Hb CS trait and alpha thalassemia-1 trait. The remainders of carrier infants had Hb of >11 g/dl. Therefore, the total number of infants with anemia defined by Hb less than 11 g/dl was found in 7 out of 72 infants which reflected the anemia rate of 9.2 per cent. Six were caused by the carrier state of thalassemia and hemoglobinopathies and one by iron deficiency.

The infants with iron deficiency and borderline iron depletion were supplemented with iron at the dose of 3 mg/kg/day for 3 months. Repeated blood sampling was performed in the infant with iron deficiency anemia. Although his hematological parameters were completely corrected, long-term follow-up is needed.

The association of low levels of serum ferritin with the infant's sex, birthweight, bodyweight, height and head circumference at one year of age, and dietary intake, was evaluated. The significant risk factor was the dietary intake, which included continuation of breast feeding until one year of age without adequate haem iron supplement, exclusive formula feeding, inadequacy of solid food supplement with only one meal per day and excluding haem iron of animal liver without substitution as shown in Table 4. The formula was iron fortified with an average of 0.8-1 mg/100 kcal. The mean ± SD of serum ferritin in 57 infants without the risk factors who received formula and 2-3 meals per day was 31.9 ± 1.9 (range 16.5-61.8), which was significantly higher than those of 15 infants with at least one of the above risk factors (mean ± SD 14.1 ± 1.7, range 8.3-23.7) (p=0.000). But the Hb and MCV in infants with inadequate dietary intake were not significantly different from those of infants with adequate dietary intake. Fourteen out of 15 infants at risk were associated with iron deficiency anemia (n=1), iron deficiency without anemia (n=5), and borderline iron depletion (n=8). Therefore, the parents and caregivers of children with associated risk factors were counseled for the bioavailable haem iron supplement such as meat, chicken, pork, animal liver and well-cooked animal blood.

After excluding infants with iron deficiency, borderline iron depletion and carrier state of thalassemia and hemoglobinopathies, the mean ± SD of Hb was 12.3 ± 0.6 g/dl (range 11.3-13), MCV 79 ± 3 fl (range 73.8-85.8) and serum ferritin 44.1 ± 1.4

Table 4. The levels of serum ferritin among 15 infants with inadequate dietary intake.

Type of feeding	Number of infants	Individual level of serum ferritin (ng/L)
Breast feeding plus 3 meals/day	5	4.7, 9.8, 10.8*, 18.9, 23.1
Formula alone	3	13.7, 14.2, 34
Formula plus 1 meal/day	3	9.1, 17.1, 25.8
Formula plus 3 meals/day but excluding animal liver	4	7.1, 11.9, 16.6, 21.2

* only one meal of solid food was given daily

ng/L (range 31.0-62.7) among 20 normal infants. Their peripheral blood smears showed normochromic normocytic red blood cells.

DISCUSSION

Iron is essential for human growth and development. The iron deficiency state affects the developmental milestones[9,10]. Between 4 and 12 months of age, the total body iron increases by approximately 130 mg. The sources of iron other than milk are necessary. Therefore, if they do not receive sufficient iron-containing foods, iron deficiency may occur and usually develops after the first birthday[11]. Therefore, evaluation of iron status at the first birthday is cost-effective for the prevention of iron deficiency in children.

In the present study, only a single blood sampling was drawn when the infants were in good health without concurrent infection. Mainly, hemoglobin and serum ferritin were used for the assessment of iron status. The serum iron and total iron binding capacity for determining transferrin saturation were not included because of the technical difficulty in preparing iron free equipment during blood samplings and laboratory testings. Therefore, the status of iron depleted and iron-deficient erythropoiesis could not be distinguished by the low serum ferritin alone. However, both statuses, which were classified as iron deficiency without anemia, require iron supplements for at least 3 months.

The risk of anemia was 9.2 per cent (7/72). Only one infant had iron deficiency anemia while 6 infants had anemia without iron deficiency. Their anemia was caused by the carrier state of thalassemia and hemoglobinopatheis. Additionally, the risk of iron deficiency state was 8.3 per cent (6/72) which included one infant with anemia and 5 infants without anemia. The risk of anemia and iron deficiency state were similar to the study of Picciano et al[12] in a private practice reporting the risk of 8 per cent and 11 per cent, respectively. Although the rates were lower than those in the general population of 15 per cent, the infants with borderline iron depletion verified by serum ferrintin between 12 and 30 ng/L were as high as 54.2 per cent. Iron depletion or deficiency is mainly caused by an inadequate iron supplement combined with an increasing iron requirement in infants because of high growth rate. Knowledgeable parents and caregivers in preparing adequate haem iron supplement are essential. A misunderstanding about excluding haem iron from animal liver without substitution was found in four parents in the present study. Therefore, the awareness of parents and caregivers in providing adequate haem iron supplement (1 mg/kg/day) of approximately 15 mg per day is important for the prevention of iron deficiency in infants.

It is suggested that the determination of Hb, MCV, peripheral blood smear and serum ferritin by using 2 ml of whole blood, should be included in the screening for iron status. However, they are insufficient for identifying the cause of anemia since there is a high prevalence (30-40%) of carrier states of thalassemia and hemoglobinopathies in the Thai population, which also manifests a slightly low Hb and MCV, and microcytic red blood cells on the peripheral blood smear. Repeated blood sampling from one-year-old infants is technically difficult and also induces psychological trauma. Therefore, an additional 3 ml of EDTA blood should be stored for further hemoglobin typing and DNA analysis for the common carrier states of thalassemia and hemoglobinopathies.

From a practical viewpoint, slightly low levels of Hb and MCV are commonly found in the carrier state of thalassemia and hemoglobinopathis. But a normal or slightly low level of Hb and MCV cannot distinguish iron deficiency or iron depleted state. Also, there is no significant difference in the level of Hb and MCV among infants with or without

iron deficiency state. A low level of Hb will be found in the late stage of iron deficiency as shown in the present study and only one out of 45 infants with iron deficiency or iron depletion had Hb of less than 11 g/dl indicating anemia. An additional determination of serum ferritin is essential for the accurate diagnosis of the iron deficiency state. However, if, CBC is the only available test, and the infant is anemic with hemoglobin less than 11 g/dl, elemental iron of 3 mg/kg/d should be given. One month later, CBC and peripheral blood smear should be repeated. If the level of hemoglobin rises more than 0.5 to 1 g/dl, 2 more complete months of iron therapy for the presumptive diagnosis of iron deficiency anemia are needed. If the hemoglobin is still less than 11 g/dl after 3 months of iron supplementation, the status of carriers of thalassemia or hemoglobinopathis is suspected. A further investigation of hemoglobin typing combined with DNA analysis for alpha thalassemia trait is suggested according to the laboratory facilities.

In conclusion, the adequate haem iron supplement in 3 meals of solid food for one-year-old infants should be routinely emphasized in the child-health supervision in order to prevent iron deficiency status.

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ภาวะธาตุเหล็กในทารกอายุ 1 ปีที่คลินิกเด็กดี

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ทารกที่มีสุขภาพแข็งแรงอายุ 1 ปี จำนวน 72 ราย (ชาย 37, หญิง 35) ที่คลินิกเด็กดีของคลินิกเอกชน ได้รับการตรวจเลือดเกี่ยวกับภาวะธาตุเหล็กในร่างกาย รวมทั้งได้ตรวจ CBC, hemoglobin typing และภาวะที่มียีนแฝงโรคธาลัสซีเมียและฮีโมโกลบินผิดปกติ น้ำหนักเฉลี่ยของทารกแรกเกิดและเมื่ออายุ 1 ปี เท่ากับ 3079 กรัม และ 10 กก ตามลำดับ ได้ศึกษาภาวะธาตุเหล็กในทารกโดยแบ่งกลุ่มตามระดับซีรั่มเฟอไรทินได้เป็น 4 กลุ่ม คือ กลุ่มที่ 1 ภาวะซีดจากการขาดธาตุเหล็ก (Hb <11 กรัม/ดล และเฟอไรทิน <12 นก/ล) จำนวน 1 ราย (1.4%) กลุ่มที่ 2 ภาวะพร่องธาตุเหล็กแต่ไม่ซีด (Hb >11 กรัม/ดล และเฟอไรทิน <12 นก/ล) จำนวน 5 ราย (6.9%) กลุ่มที่ 3 ภาวะพร่องธาตุเหล็กสะสม (เฟอไรทิน 12–30 มคก/ล) จำนวน 39 ราย (54.2%) กลุ่มที่ 4 ภาวะธาตุเหล็กปกติ (เฟอไรทิน >30 นก/ล) จำนวน 27 ราย (37.5%) ดังนั้น มีภาวะพร่องธาตุเหล็ก 6 รายคิดเป็นร้อยละ 8.3 แต่ Hb และ MCV ของทารกที่มีภาวะพร่องธาตุเหล็กแต่ไม่ซีด ภาวะพร่องธาตุเหล็กสะสมหรือภาวะธาตุเหล็กปกติไม่แตกต่างกัน

ทารก 25 ราย คิดเป็นร้อยละ 34.7 มีภาวะยีนแฝงของโรคธาลัสซีเมียและฮีโมโกลบินผิดปกติ ทารกเหล่านี้มีระดับ Hb และ MCV ต่ำกว่าทารกที่ไม่มียีนโรคธาลัสซีเมียแฝง แต่ระดับเฟอไรทินไม่แตกต่างกัน นอกจากนี้ได้ศึกษาในทารกที่มีภาวะยีนแฝงร่วมกับภาวะพร่องธาตุเหล็ก ปรากฏว่าไม่มีความแตกต่างกันในระดับ Hb และ MCV ในทารกที่มียีนแฝง ที่มีหรือไม่มีภาวะพร่องธาตุเหล็กร่วมด้วยก็ตาม แต่พบทารกที่มียีนแฝง 6 รายที่มีระดับ Hb ต่ำกว่าปกติเล็กน้อย ระหว่าง 10.3–10.9 กรัม/ดล แต่มีระดับเฟอไรทินระหว่าง 18.7–382.9 นก/ล ทารกที่มียีนแฝงรายอื่น ๆ มีระดับ Hb >11 กรัม/ดล ดังนั้น การศึกษาครั้งนี้พบทารก 7 รายคิดเป็นร้อยละ 9.2 มีภาวะซีด (ฮีโมโกลบินต่ำกว่า 11 กรัม/ดล) เนื่องจากมีภาวะที่ยีนแฝงของโรคธาลัสซีเมียและฮีโมโกลบินผิดปกติจำนวน 6 ราย และภาวะซีดจากการขาดธาตุเหล็ก (ฮีโมโกลบินต่ำกว่า 11 กรัม/ดล และซีรั่มเฟอไรทินต่ำกว่า 12 นก/ล) จำนวน 1 ราย

ความเสี่ยงต่อภาวะขาดหรือพร่องธาตุเหล็กเกิดจากการให้นมแม่จนอายุ 1 ปี โดยให้อาหารเสริมที่มีธาตุเหล็กไม่เพียงพอ, การให้นมผสมอย่างเดียว, ให้อาหารเสริมเพียงมื้อเดียว รวมทั้งการไม่ให้ตับจากสัตว์ชนิดต่าง ๆ เลย ทารกที่ได้รับสารอาหารไม่เพียงพอมีระดับซีรั่มเฟอไรทิน (เฉลี่ย 14.1 $\pm$ 1.7 มคก/ล) ต่ำกว่าทารกที่ได้รับสารอาหารเพียงพอ (เฉลี่ย 31.9 $\pm$ 1.9 นก/ล) อย่างมีนัยสำคัญ ($p = 0.000$)

ดังนั้น การให้อาหารเสริมที่มีธาตุเหล็กอย่างเพียงพอในอาหาร 3 มื้อแก่ทารกอายุ 1 ปี จะช่วยป้องกันภาวะขาดและพร่องธาตุเหล็กในทารก

คำสำคัญ : ภาวะซีดจากขาดธาตุเหล็ก, ภาวะธาตุเหล็กในทารกอายุ 1 ปี

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