

Original Article

Iron deficiency anemia among hospitalized children with microcytic anemia hospitalized in national children's hospital in Vientiane, Lao PDR

Bounpalisone Souvanlasy¹, Surapon Wiangnon², Arunee Jetsrisuparb², Goonnapa Fucharoen³, Khampe Phongsavath¹, Mayfong Maysay⁴, Sommanykhone Phangmanyxay¹ and Sourideth Sengchanh¹

¹Department of Pediatrics, University of Health Science, Ministry of Health, Lao PDR; ²Division of Hematology and Oncology, Department of Pediatrics, Srinagarind hospital, Faculty of Medicine, Khon Kaen University, Thailand; ³Centres for Research and Development of Medical Diagnostic Laboratory, Faculty of Associated Medical Sciences, Khon Kaen University, Thailand; ⁴Physician and Head of Field Research, the welcome trust at Mahosot

Abstract:

Background: Iron-deficiency anemia (IDA) is one of the common conditions bringing children to the hospital. The condition remains an important public health challenge that is preventable and treatable, with guidelines for early, cost-effective detection. This study aimed to determine the prevalence of IDA, confirmed by an increase in Hb >1g/dL after 4 weeks of oral iron supplementation. **Methods:** This cross-sectional prospective descriptive study enrolled children 6 months to 5 years old with a diagnosis of microcytic anemia and admitted to a pediatric ward from August 1st 2014 to January 31 2015. **Results:** Among 722 children, 38.55% were anemic, and 37.68% had microcytic anemia. The majority (85.2%) were aged 6-24 months, and of those children. Fifty percent had mild anemia, 49.3% had moderate anemia, 0.7% had severe anemia (only 2% were admitted to the hospital because of anemia), Regarding nutritional status, 7.5%, 4.1%, 5.4%, 0.7% had stunting, wasting, underweight and overweight status, retrospectively. After 4 weeks of therapeutic trial with iron, 41.2% were found to have IDA, and 21.21% had combined with thalassemia, 82.5% had thalassemia with 21.10% heterozygous Hb E, 11.3% had homozygous Hb E and 11.92% had combined alpha and Hb E. In addition, 7% had the alpha thal 1 gene (SEA), 15.49% had the alpha thal 2 gene (3.7kb deletion), 2.8% had the alpha thal 2 gene (4.2kb deletion), 2.8% had non-deletion Hb Constant Spring and 2.8% had Hb Pakse. **Conclusion:** The high prevalence of IDA found in this study suggests that routine iron supplementation for a hospitalized child with microcytic anemia is a favorable, cost-effective strategy.

Keywords : ● Iron deficiency anemia ● Children ● Microcytic anemia

J Hematol Transfus Med. 2019;29:35-46.

Received 8 December 2018 Corrected 12 December 2018 Accepted 14 February 2019

Correspondence should be addressed to Bounpalisone Souvanlasy, Department of Pediatrics, University of Health Science, Ministry of Health, Lao PDR;

Introduction

Anemia is a global public health problem with major consequences for human health. In addition, severe anemia is a significant cause of childhood mortality. The World Health Organization (WHO) estimates 1.6 billion people worldwide are affected with anemia¹. Among this affected population, approximately 47% are children under 5 years old and constitute one of the most vulnerable groups, especially those in the first 2 years of life². The etiology of childhood anemia is multifactorial³. Microcytic anemia is a disorder, frequently reported in pediatrics. Even though many children with this disorder have no symptoms or complaints⁴, iron deficiency anemia (IDA) has been linked to increased childhood morbidity and impaired cognitive development. It appears to be associated with lower intellectual performance scores among school children and leads to psychological and developmental consequences for children.⁵

Iron deficiency accounts for about one half the world's burden of anemia. WHO estimates that in 2004, IDA resulted in 273,000 deaths: 45% in Southeast Asia, 31% in Africa, 9% in the Eastern Mediterranean region, 7% in the Americas, 4% in the Western Pacific, and 3% in Europe with, overall 97% occurring in low- and middle-income countries⁶. One of these countries is Lao People's Democratic Republic (PDR), where the national prevalence of anemia among children aged 6–59 months was 40.9% in 2006. One tenth (10.8%) of these cases involved IDA. The prevalence of IDA was even higher (25.2–30.9%) among children aged 6–23 months⁷. A population-based cross-sectional study was carried out in six communities in Songkhone District, Savannakheth Province, in February 2009. The prevalence of anemia was found to be 48.9% and was higher among children aged 6–23 months and children from large families⁸. However, more recent data is lacking. Therefore, this study was conducted to acquire information on the current status of microcytic anemia in Lao PDR.

In Lao PDR, anemia is one of the common conditions bringing children to the hospital. Causes of anemia often are unknown because of limitations in diagnostic resources. Many anemic children are hospitalized due to other causes, and their anemia is neglected. IDA is a preventable, treatable disease, with guidelines for early cost-effective detection. Basic screening for IDA consists of a complete blood count (CBC) looking for hemoglobin less than the age-adjusted normal range, with a sensitivity of 86% and a specificity of 97%⁹. The diagnosis is supported by a low mean corpuscular volume (MCV) and increased red cell distribution width (RDW). However, the diagnosis will be more complicated in areas where thalassemia is also common¹⁰. Confirmation of the diagnosis is commonly obtained with a trial of iron supplementation, which is appropriate in resource-limited countries¹ like Lao PDR. Oral administration of simple ferrous salts (most often ferrous sulfate) provides inexpensive, less toxic and effective therapy¹¹. In the United States, the most cost-effective strategy for infants and young children, presenting mild microcytic anemia and a presumptive diagnosis of IDA, is a therapeutic trial of iron. The estimated cost for this approach for a 10 kg child is approximately five dollars.¹²

Screenings that can be performed on fingertip samples of blood offers an easy method for surveying large numbers of individuals at minimal costs of time and resources¹. This test identifies children with IDA without requiring iron status markers, which could help reduce cost of diagnosis and treatment, especially in resource-poor settings³. The aim of this study was to determine the prevalence of IDA as confirmed by increased Hb > 1g/dL after oral iron supplementation of ferrous sulfate 4 mg/kg/day among children 6 months to 5 years old who had microcytic anemia and had been hospitalized.

Material and Methods

This prospective descriptive study was carried out from August 1st 2014 to January 31 2015, in an inpatient ward. Purposive sampling (Figure 1) was used to enroll subjects while those who received blood transfusions or iron supplementation in the previous three months were excluded, as were children with thalassemia diagnosed by hemoglobin typing. Moreover,

children who presented twice during the study period, those with poor compliance and those with any condition causing impaired absorption were excluded.

The sample size was calculated to be 138 cases using a simple formula (Daniel, 1999), with a confidence level of 95%, increased by 30% of the total for cases expected to be lost to follow-up.

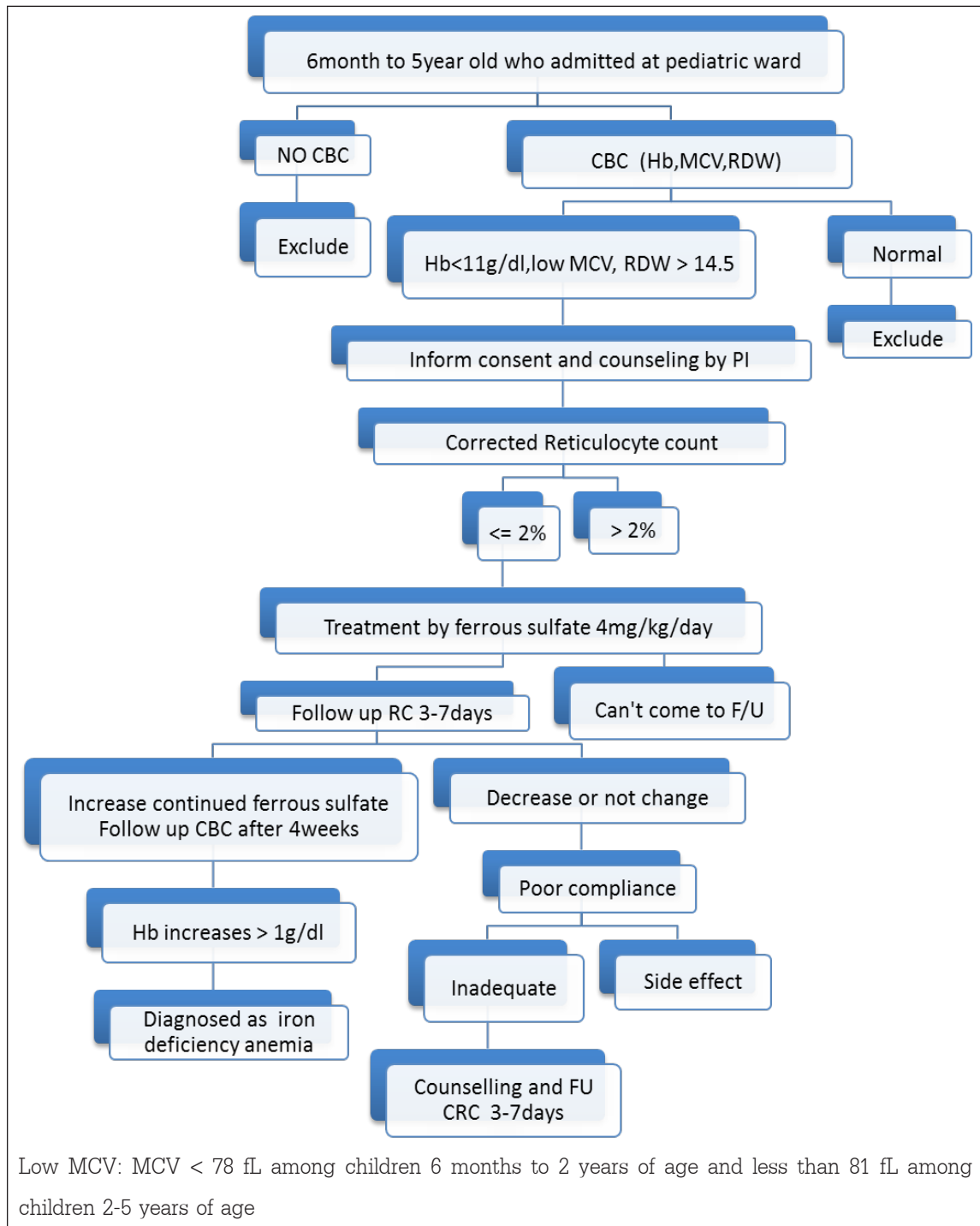


Figure 1 Purposive sampling

Ethics considerations

Ethics approval was obtained from the Lao National Ethics Committee for Health Research.

Statistical analysis

Data were entered using an Epidata 3.1 database, then were exported to Microsoft Excel. The data were then analyzed using STATA Program, using descriptive statistics to calculate frequency and percent for age, sex and severity. The Wilcoxon matched pair sign rank and the Fisher's exact test were used to compare the mean difference of hematological parameters between the two dependent groups because data did not conform to a normal distribution. The level of statistical significance was considered at $p < 0.05$

Results

A total of 722 children between the ages of 6 and 59 months were admitted to the pediatric ward. Among these cases, 32 children (4.43%) did not have CBC at the time of admission. We found 266 of 690 inpatients (38.55%) anemic. No patients were diagnosed with anemia with high MCV, but 260 patients (37.68%) had low MCV, and 6 patients (0.86%) had normal MCV.

Amongst children with low MCV, only 208 (80%) enrolled in the study, 18 patients (6.92%) had corrected reticulocyte count of more than 2%, 29 patients (11.15%) had RDW of less than 14.5%, while 5 patients (1.92%) did not give consent for the study. Furthermore, we followed up on the 208 patients that enrolled in the study 4 weeks after receiving a therapeutic trial of iron. Unfortunately, 60 patients were excluded from the study. In all, 8 patients (3.84%) were dismissed because of poor compliance after receiving counseling twice and 52 (25%) were lost to follow-up. Only the remaining 148 patients (71.15%) were we able to complete the study. Figure 2 illustrates an overview of the data collected. One important note is the randomness of the data sample, despite the 28.85% loss in the sample count. Among the differences of age, sex and hematological findings of children that were able to participate in the study, those that were lost to follow-up were similarly aligned, as shown in Table 1.

When we analyzed the 148 participants (71.15%); 101 (68.2%) were males and 47 (31.8%) were females, with a mean age of 16.67 months old (± 9.48 months), and median age of 14 months, (ranging from 6-57

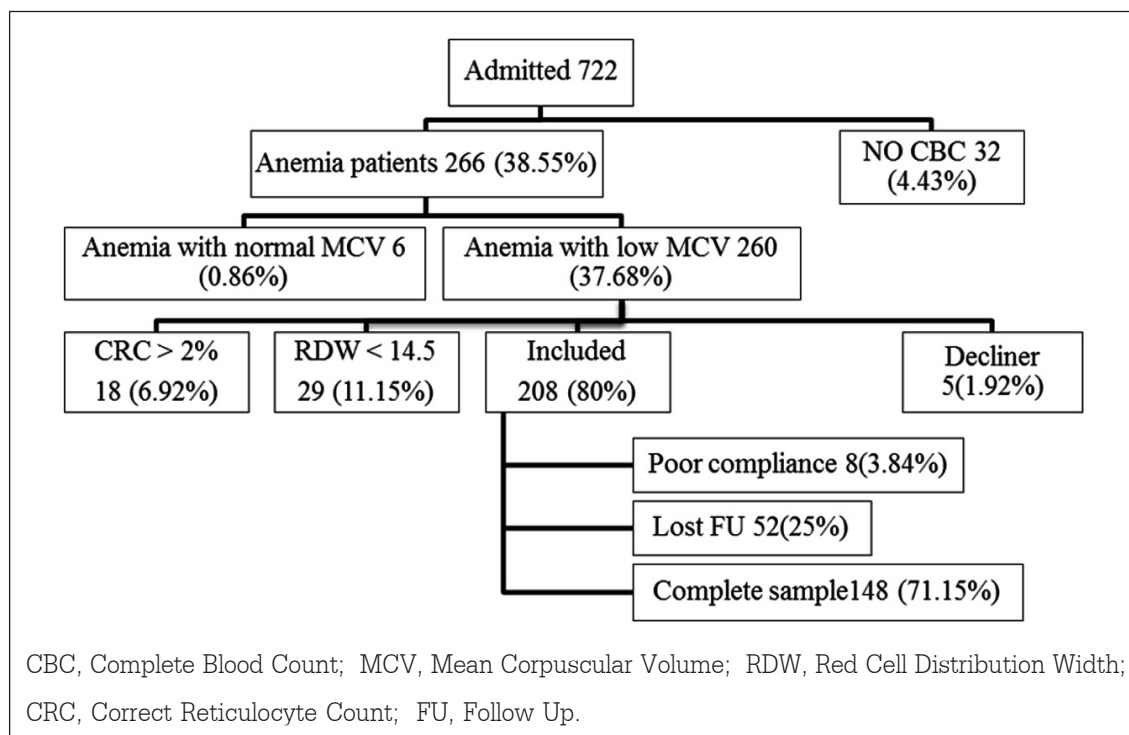


Figure 2 Overview of the data

Table 1 Compare the demographic characteristic and hematological finding data

	Complete data collection		Lost to follow-up	
	N (%) 148 (100)	Mean \pm SD	N (%) 60 (100)	Mean \pm SD
Age (months)				
6-11	51 (34.5)	16.67 \pm 9.48	18 (30)	16.67 \pm 9.67
12-24	75 (50.7)		30 (50)	
25-36	19 (12.8)		8 (13.3)	
>36	3 (2)		4 (6.7)	
Sex				
Boy	101 (68.2)		37 (61.7)	
Girl	47 (31.8)		23 (38.3)	
Hematological finding				
RBC ($\times 10^{12}/L$)	148 (100)	5.2 \pm 0.63	60 (100)	5.2 \pm 0.73
Hb (g/dL)	148 (100)	9.8 \pm 0.88	60 (100)	9.9 \pm 0.75
HCT (%)	148 (100)	31.5 \pm 3.06	60 (100)	32.5 \pm 2.74
MCV (fl)	148 (100)	61.6 \pm 8.54	60 (100)	62.2 \pm 8.47
MCH (pg)	148 (100)	19.2 \pm 2.80	60 (100)	19.0 \pm 2.51
MCHC (g/dL)	148 (100)	30.9 \pm 2.40	60 (100)	30.5 \pm 1.60
RDW (%)	148 (100)	18.2 \pm 3.10	60 (100)	18.6 \pm 3.98
WBC ($\times 10^9/L$)	148 (100)	12.1 \pm 4.68	60 (100)	11.8 \pm 5.37
PLT ($\times 10^9/L$)	148 (100)	340.4 \pm 131	60 (100)	307 \pm 108
CRC (%)	148 (100)	0.83 \pm 0.48	60 (100)	0.95 \pm 0.52

RBC, Red Blood Cell; Hb, Hemoglobin; HCT, Hematocrit; MCV, Mean Corpuscular Volume; MCH, Mean Corpuscular Hemoglobin; MCHC, Mean Corpuscular Hemoglobin Concentration; RDW, Red cell Distribution Width; WBC, White Blood Cell; PLT, Platelet; CRC, Correct Reticulocyte Count

months). Moreover, we segregated the age groups in accordance with their level of microcytic anemia. We found that the 12 to 24 months group had a microcytic anemia level of 75 (50.7%); the 6 to 11 months group had a level of 51 (34.5%), the 25-36 months had a level of 19 (12.8%); and the 36 months and older group had a level of 3 (2%).

Using the WHO Child Growth Standards median, we found that the nutritional status of more than two thirds of the children with microcytic anemia was normal. The three criteria that we used in the assessments are shown in Table 2.

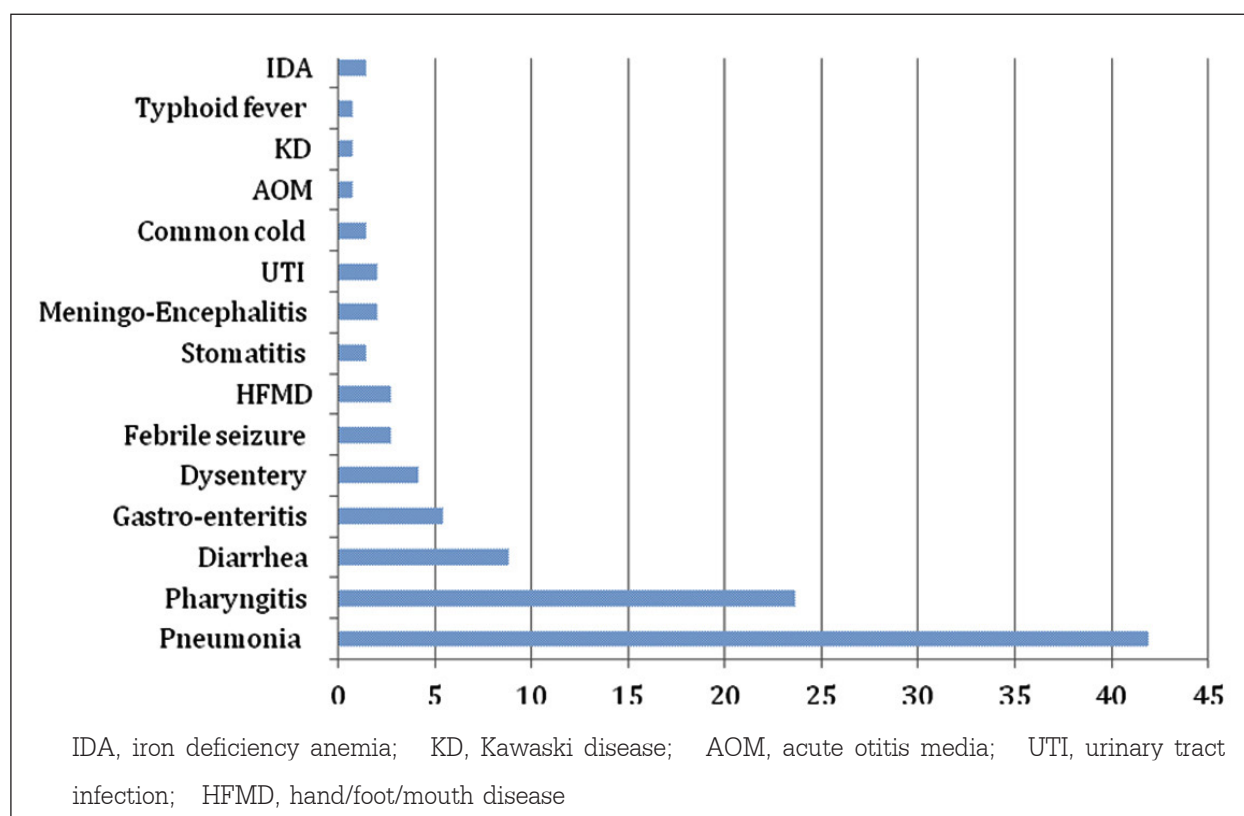
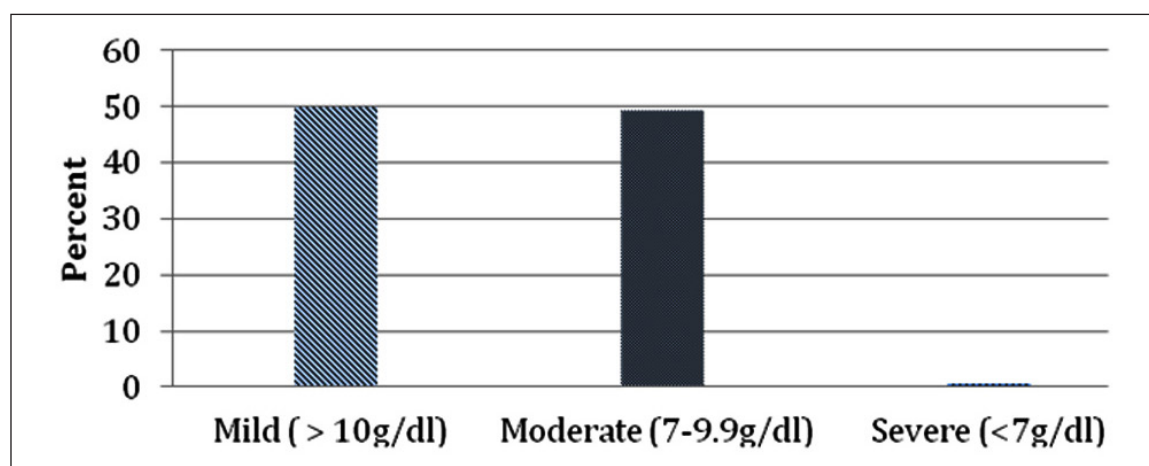
Of the 148 children who were enrolled in the study, 145 (98%) were admitted with conditions other than anemia, while only 3 children (2%) had only anemia.

Furthermore our diagnosis revealed 62 children (41.9%) with pneumonia, 35 (23.6%) with pharyngitis, 13 (8.8%) with diarrhea, 8 (5.4%) with gastro-enteritis, 6 (4.1%) with dysentery, 4 (2.7%) with febrile seizures, 4 (2.7%) with hand/foot/mouth disease, 2 (1.4%) with IDA, 2 (1.4%) with stomatitis, 3 (2%) with meningo-encephalitis, 3 (2%) with urinary tract infection, 2 (1.4%) with common cold, 1 (0.7%) with acute otitis media, 1 (0.7%) with Kawasaki disease, and lastly 1 (0.7%) with typhoid fever. Figure 3 illustrates the diagnosis of all patients upon admission.

The mean values of RBC, Hb, HCT, MCV, MCH, MCHC, RDW, WBC, platelet and CRC before receiving the therapeutic trial of iron are shown in Table 1. Figure 4 illustrates the severity of anemia. It shows

Table 2 Nutrition statuses among children with microcytic anemia

Nutrition status	Weight/Height (wasted) N (%)	Height/Age (Stunted) N (%)	Weight/Age (Underweight) N (%)
Standard median	114 (77)	90 (60.8)	85 (57.4)
≤ -1sd	21 (14.2)	24 (16.2)	41 (27.7)
≤ -2sd	6 (4.1)	23 (15.5)	14 (9.5)
≤ -3sd	4 (2.7)	10 (6.8)	7 (4.7)
≤ -4sd	2 (1.4)	1 (0.7)	1 (0.7)
> 2sd	1 (0.7)	N	N

**Figure 3** Diagnosis of all patients upon admission**Figure 4** Severity of microcytic anemia on admission

that 74 children (50%) had mild anemia, 73 children (49.3%) had moderate anemia, and only 1 child (0.7%) had severe anemia. In addition, we followed up the reticulocytosis between 3 to 7 days after the iron therapeutic trial, with the mean duration of 4.18 (\pm 1.27) days. We found a definite increase of reticulocytes with the mean value of 1.56% (\pm 0.59), and median of 1.5% (ranging from 0.3-3.6%). Furthermore, we had relatively good compliance from an overwhelming number of cases, 144 (97.3%), while only 4 cases (2.75%) had poor compliance. However, the 4 cases that showed poor compliance were re-counseled and received complete medication. Figure 3 illustrates the diagnoses patients received upon admission.

Based on the results of the patients who responded to the 4-week iron therapeutic trial (ferrous sulfate, 4 mg/kg/dose), we observed how hemoglobin levels changed. We found no increase in hemoglobin level of $> 1\text{g/dL}$ among 87 children (58.8%), while 61 children (41.2%) exhibited an increase of $> 1\text{g/dL}$. Interestingly, we found 49 children (80.3%) less than 2 years of age diagnosed with IDA. The mean Hb level with

IDA was $9.45 (\pm 1.01)$. Next, we compared the mean hematological findings before and after receiving iron therapeutic trial between children who had IDA with those who hadn't. The results are shown in Table 3.

Of the 148 patients with microcytic anemia, we diagnosed IDA among 61 patients (IDA group), and 87 patients with no response to the iron therapeutic trial (NIDA group). We continually checked both groups for beta thalassemia and alpha thalassemia. However with a limited budget, we could only work with 132 patients (89.1%). High performance liquid chromatography (HPLC) or capillary zone electrophoresis (CE) was used to analyze hemoglobin level in the 51 patients with IDA, and 81 patients with NIDA. Additionally, polymerase chain reaction (PCR) was used to analyze alpha thalassemia among 71 patients (47.97%), of which 51 were categorized as IDA group and 20 patients as NIDA group.

After evaluation, 109 children (82.57%) were found to have thalassemia. In total, 21 children (19.26%) were found to have thalassemia disease, while the other 88 children (80.73%) were trait carriers. We evaluated 28

Table 3 Comparing the mean hematologic findings between children with IDA and NIDA diagnosis before and after receiving iron therapeutic trial

Group	Iron deficiency anemia (n = 61)		Not iron deficiency anemia (n = 87)		p-value
Laboratory findings	Before	Change after	Before	Change after	
	Mean ± SD	treatment	Mean ± SD	treatment	
RBC (x 10 ¹² /L)	5.13 ± 0.69	+0.44	5.25 ± 0.59	+0.00	<0.05
Hb (g/dL)	9.45 ± 1.01	+1.85	10.09 ± 0.67	+0.11	<0.05
HCT (%)	31.06 ± 3.17	+4.04	31.82 ± 2.95	+0.91	<0.05
MCV (fl)	61.51 ± 9.17	+2.68	61.67 ± 8.12	+1.33	0.80
MCH (pg)	19.72 ± 2.80	+0.69	19.58 ± 2.66	+0.05	<0.05
MCHC (g/dL)	30 ± 3.07	+1.89	31.59 ± 1.49	-0.40	<0.05
RDW (%)	18.53 ± 3.32	+1.15	18.09 ± 2.94	+0.51	0.09
WBC (x10 ⁹ /L)	12.2 ± 5.01	-1.98	12.04 ± 4.46	-2.06	0.29
PLT (x10 ⁹ /L)	355 ± 139	-15.2	329.6 ± 125	+22.5	0.07
CRC (%)	0.83 ± 0.49	+1.17	0.83 ± 0.48	+0.37	<0.05

RBC, Red Blood Cell; Hb, Hemoglobin; HCT, Hematocrit; MCV, Mean Corpuscular Volume; MCH, Mean Corpuscular Hemoglobin; MCHC, Mean Corpuscular Hemoglobin Concentration; RDW, Red Cell Distribution Width; WBC, White Blood Cell; PLT, Platelet; CRC, Correct Reticulocyte Count

Table 4 Various thalassemia genotypes among children presenting microcytic anemia

Genotype	IDA	NIDA	Total	%
β -Thalassemia				41.28
Heterozygous E	5	18	23	21.10
Heterozygous β -thalassemia	1	5	6	5.50
Homozygous E	2	13	15	13.76
Compound heterozygous β -thalassemia / Hb E	0	1	1	0.91
α -Thalassemia				19.26
Heterozygous α -thal 2 gene 3.7kb deletion	6	4	10	9.17
Heterozygous α -thal 2 gene 4.2kb deletion	1	1	2	1.83
Homozygous α -thal 2 gene 3.7kb deletion	1	0	1	0.91
Heterozygous α -thal 1 gene (SEA)	3	1	4	3.66
Heterozygous Hb Constant Spring (Hb CS)	1	1	2	1.83
Heterozygous Hb Pakse (Hb PS)	2	0	2	1.83
α - β Thalassemia				17.43
Heterozygous Hb E with heterozygous α -thal 1 *	3	10	13	11.92
Heterozygous Hb E with Heterozygous α -thal 2 gene 3.7kb deletion	1	0	1	0.91
Homozygous Hb E with heterozygous α -thal 1 gene (SEA)	0	1	1	0.91
Homozygous Hb E with Hb Constant Spring (Hb CS)	1	0	1	0.91
Homozygous β +thalassemia with heterozygous α -thal 1	0	1	1	0.91
CSEABart's disease	1	1	2	1.83
Non-Thalassemia **	23		23	
Normal Hb typing can not rule out Heterozygous α -thal 1 or Homozygous α -thal 2-thalassemia***	0	24	24	22.01
Total	51	81	132	

*3 cases had the alpha thal 1 gene deletion (SEA) and in 10 cases, we were unable to perform PCR for alpha thalassemia and so only performed Hb typing (%E less than 25%)

**Diagnosed by Hb typing and PCR for alpha thalassemia deletion and nondeletion

***Hb typing (A2A) but A2 range 1.7-3% mean (2.15%) and still showed low MCV, which did not respond to iron therapeutic trial, no PCR for alpha thalassemia deletion and, non-deletion

cases (21.21%) with the combination of thalassemia carriers and IDA. Of the 4 cases with thalassemia disease, 3 were homozygous E cases, and 1 was CSEA Bart's disease. Various thalassemia genotypes among patients are shown in Table 4. The most common form of thalassemia amongst the carriers was heterozygous E with 23 patients (21.10%), followed by homozygous E with 15 patients (11.36%), heterozygous E with heterozygous alpha thalassemia 1 with 13 patients (11.92%). Moreover, both deletion and nondeletion alpha thalas-

semia carriers (diagnosed through PCR interpretation) were found among 17 patients (15.59%) and 4 patients (3.66%), respectively. We looked for the proportion of patients, who were nonresponsive to the iron therapeutic trial with normal Hb typing, but did not perform PCR because of financial limitations. However, we suspected that 24 patients (20%) might be carriers of alpha thalassemia 1, or homozygous alpha thalassemia 2, due to low Hb, MCV, and MCH.

Discussion

This is the first prospective descriptive study evaluating microcytic anemia among children between the ages of 6 and 59 months in Lao PDR. This group was chosen because the overwhelming majority was checked for CBC during admission, which made the study more cost-effective and convenient. We found that anemia was a common finding among these patients. In fact, 266 of 690 inpatients (38.55%) that had CBC were found to have anemia. However, the current anemia prevalence in Lao PDR is lower than the 2011 national survey, which found a prevalence of 42% at that time. The difference with our study was that it involved a population study. The most common of all types of anemia was microcytic anemia, which was found in 260 (97.74%) cases. Comparatively, only 6 cases were normocytic anemia, 3 cases were leukemia, 1 case was neuroblastoma, 1 case was G6PD deficiency, and 1 case was AIHA. When we screened for the criteria for microcytic anemia, 6.92% had a corrected reticulocyte count of more than 2%. After diagnosis through Hb typing, 2 Hb H disease cases were found along with 3 Hb H with CS cases, 3 heterozygous E cases, 2 heterozygous alpha thal 1 (SEA) cases, 2 homozygous E cases, 1 homozygous Constant Spring case, 2 heterozygous beta thalassemia cases, and 2 EABart's disease cases. In all, 11.15% had RDW < 14.5%, but were not screened for hemoglobin typing. A total of 1.92% refused to give informed consent, because the parents did not want to have their children's blood checked several times, and were not able to make follow-up visits. For the 208 cases, 25% were lost to follow-up due to various reasons, such as hospital distance, improvement in patient's condition, financial problems and other causes of missed appointments. A total of 5% had poor compliance due complications after taking ferrous sulfate, including nausea, vomiting and diarrhea. Some had difficulties with taking ferrous sulfate in pill form, because of the lack of access to liquid form. In conclusion, we

were able to analyze 148 children in the study. The sample size was large enough to represent the entire population, because the minimum calculated sample was 138. The patients' general characteristics, such as age, sex, severity of anemia, cause of admission, diagnosis on admission, and complete blood count, did not differ from the 30% of children that had loss to follow-up and poor compliance. Therefore, we conclude that the data would be reliable.

A total of 10% of the access to did differ conclude that as we observed the routine practices in a pediatric ward, we found that many patients that were admitted for other illnesses were not aware that they had anemia. Thus, anemia was never diagnosed and treated unless the condition was severe. Sometimes, severe anemia was treated by blood transfusion without a preliminary diagnosis, which became one of the main the points of interest in the study. Neglected diagnoses of anemia were the primary reason the study was conducted. IDA is the only disease that we could diagnose, and treat with a cost-effective iron therapeutic trial. This trial also helped us differentiate mild thalassemia from trait carriers.

The primary objective in this study was to determine the prevalence of IDA among children with microcytic anemia admitted with reference to the purposive sampling previously mentioned. Of all cases of microcytic anemia in this study, the male to female ratio was 2.1:1. The difference in sex distribution could perhaps be explained by higher numbers of boys being admitted to the hospital, where the majority in the 12-24 months age group was 50.7% followed by infants 6-12 months, 34.5%. For their nutrition status, we found 7.5% exhibited stunting, 4.1%, wasting, 5.4%, underweight and 0.7%, overweight. These figures differed in comparison to those of developed countries where as much as 20-25% were overweight, and 8% were underweight²⁶ due to the difference in food intake. When we screened for the severity of anemia, we found 50% were mild and 49.3%, moderate.

Because most cases were mild or moderate, anemia was often neglected due to the lack of signs and symptoms. Although this research study was conducted during the monsoon season, we had 41.9% of children diagnosed with pneumonia, and 23.6% had pharyngitis, independently of their hemoglobin level. Diarrhea, gastro-enteritis and dysentery were diagnosed in 8.8%, 5.4%, and 4.1%, respectively. However, ferrous sulfate was not given until these diseases were resolved, to optimize iron absorption. Only three cases (2%) were admitted because of anemia. One child had severe anemia, and two children had moderate anemia. Both were diagnosed with IDA, with a hemoglobin level between 7 and 7.5 mg/dl. Both cases responded to the iron therapeutic trial without needing red blood cell transfusion. We deduced from this research that 97.3% of Laotian children had good compliance with ferrous sulfate, even though the medication existed predominantly in pill form and produced a few side effects. However, parents often requested iron solution because they found it difficult to administer a portion of the pill according to their child's weight.

IDA continues to be the leading cause of anemia among preschool children, and remains a global public health challenge in Lao PDR. From this study, we found a very high prevalence of IDA at 41.2%. When divided in groups, IDA was highest in the 12-24 months group (50.8%), followed by the 6-12 months group (29.5%), and the 24-36 months group (19.7%). The disturbingly high percentage can be explained by a lack of knowledge about IDA, the effectiveness of preventive iron supplementation, and 'nutrition counseling outreach in rural areas.

The second objective of the study was to examine the prevalence and genotype of thalassemia. We found over 82.57% of patients with microcytic anemia had thalassemia (80.73%), and were also trait carriers (19.26%). The most common was β chain variant Hb E. We could observe heterozygous E (21.10%), homozygous E (11.3%) and the combination of alpha

and HbE (11.92%). Likewise, the percentage of hemoglobinopathies among children was similar to that of pregnant Laotian women with Hb E of 30.2%³⁵ and other neighboring countries like Thailand, Vietnam, and Cambodia. For all alpha thalassemia cases with PCR performed for alpha thal 1 gene deletion, we found positive results for 7% for the alpha thal 1 gene (SEA), 15.49% for alpha thal 2 gene 3.7kb deletion, 2.8% for alpha thal 2 gene 4.2kb deletion, 2.8% for alpha thalassemia carrier nondeletion Hb Constant Spring and 2.8% for hemoglobin Pakse. When thalassemia cases were examined, we found 21% of thalassemia cases with IDA. We need to realize that patients with nontransfusion dependent thalassemia (NTDT) and trait carriers could have IDA concurrently. The response to an iron therapeutic trial for children with microcytic anemia that had a corrected reticulocyte count of less than 2% was positive for both IDA and thalassemia disease or trait carriers without any side effects. Among children with IDA, an RDW variation was found from 14.6-29.5% with a mean of 18.53 (\pm 3.32) compared with children with thalassemia having 14.5-26.5%. The mean of 18.09% (\pm 2.94) was similar in comparison. Therefore, we cannot use RDW as a guide to differentiate the diagnosis between IDA and thalassemia.

Recommendations

Another important concern that emerged from this research was the high prevalence of microcytic anemia, especially IDA, among children between the ages of 12 and 24 months old (the high-risk group). The high prevalence of IDA found in our study may suggest that prophylactic iron supplementation might be more a more cost-effective solution without side effects. Practical guidelines from our research would make treating the disorder more effective. Alternatively, routine iron supplementation could be recommended for hospitalized children aged 6-59 months, and especially those 6-24 months old with microcytic anemia and low

CRC, to prevent IDA. We should urgently pay closer attention to improving iron supplementation programs for children under the ages of 5 years.

IDA among children should be considered one of the biggest health care problems in Lao PDR. It has long-term effects on vital human functions, such as mental and cognitive skills, as well as on immunity and general physical wellbeing.

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