

## Case Report

# Micronutrient deficiencies in obese Thai children

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We reported the cases of 3 obese children who presented variously with costochondral beading, numbness of both hands and feet, and hypochromic-microcytic anemia. Serum ascorbic acid concentration, erythrocyte thiamin pyrophosphate effect, and serum hepcidin concentration were done to investigate these symptoms, respectively, with findings of ascorbic acid insufficiency, thiamin deficiency, and iron deficiency anemia from poor iron absorption due to a hyper-inflammatory state, respectively. This report indicated that obese children not only have energy excess but may also risk being micronutrient deficiencies; therefore, weight management with special attention to an adequate intake of vitamins and minerals should be provided.

**Key Words:** ascorbic acid, hepcidin, iron deficiency anemia, obese children, Thiamin deficiency

## INTRODUCTION

Generally, obese individuals believe that they are in an “over-nutritious” state. In fact, however, while they might be consuming excess energy, a number of studies have found obese children and adolescents to be deficient in several important micronutrients. Studies have reported a high prevalence of iron deficiency in overweight and obese Swiss children,<sup>1</sup> and in Iranian overweight and obese children and adolescents.<sup>2</sup> Vitamin D deficiency was also found in obese American children and adolescents.<sup>3</sup> Lower serum  $\alpha$ -tocopherol and carotenoid concentrations were reported in American obese adolescents compared with normal weight subjects.<sup>4</sup> In South-East Asia countries, the prevalence of vitamin D deficiency was 91% in overweight and obese Malaysian children,<sup>5</sup> in contrast to 11.3% of obese Thai children.<sup>6</sup> To the authors’ knowledge, there has been no published study of ascorbic, thiamin, and iron deficiencies in obese Thai children.

## PATIENT 1

A 3-year-old boy presented with snoring and frequent arousals from sleep at night for 2 years. Past history revealed that several episodes of tonsillitis had been treated with oral antibiotics. Current dietary history revealed frequent consumption of French-fried potatoes served with tomato ketchup, fried fish, and 8-10 servings of a 240-mL box of sweetened soy ultra-high temperature (UHT) milk per day. He did not eat vegetables and fruits. On examination, his weight was 27.6 kg (>97<sup>th</sup> percentile), height 101 cm (>97<sup>th</sup> percentile), and weight-for-height 173%. Acanthosis nigricans, nasal congestion, tonsillar hypertrophy, and costochondral beading were found. No swollen gum and gum bleeding were observed. Other physical findings were unremarkable.

A complete blood count showed Hb 12.2 g/dL, white blood cells 16,700/mm<sup>3</sup>, platelets 461,000/mm<sup>3</sup>, mean corpuscular volume (MCV) 58.7 fL, mean corpuscular hemoglobin (MCH) 18.7 pg, red cell distribution width

(RDW) 14.9%, and hypochromic and microcytic red blood cells. His hemoglobin typing showed AA<sub>2</sub>. DNA analysis for 7  $\alpha$ -thalassaemia deletions and 2 common  $\alpha$ -thalassaemia non-deletions showed negative. Serum ferritin, transferrin, plasma zinc, and serum copper concentrations were 17.4 ng/mL (normal ferritin representing normal iron store and in the presence of infection:  $\geq 30$  ng/mL), 4 g/L (normal range: 2.04-3.6 g/L), 101 mcg/dL (normal range: 78-118 mcg/dL), and 167 mcg/dL (normal range: 64-156 mcg/dL), respectively. Therefore, microcytosis in this child was contributed to iron deficiency alone. Serum ascorbic acid concentration was 0.24 mg/dL (a lower limit of normal serum ascorbic acid: 0.4 mg/dL, and a cut-off point to define deficiency: <0.2 mg/dL). Roentgenography of both femurs showed no significant change. Fasting blood glucose concentration revealed 100 mg/dL and insulin 29.12  $\mu$ U/mL.

Overnight pulse oximetry showed desaturation of 80-85% for less than 10 seconds but being normal after changing position. Polysomnography was not available to confirm obstructive sleep apnea. Echocardiography demonstrated no pulmonary hypertension and no chamber enlargement. Lateral skull roentgenography showed adenoid hypertrophy. A Skin prick test was positive to Bermuda grass.

Simple obesity, vitamin C insufficiency, iron deficiency, adenotonsillitis, allergic rhinitis, sinusitis and probable obstructive apnea were diagnosed. Vitamin C 100 mg twice a day, ferrous sulfate 4 mg/kg/day, a decongestant,

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an antihistamine, intranasal corticosteroid, and an oral antibiotic drug were prescribed. He was advised to avoid known allergen and to clean his nasal passage with normal saline. His diet was changed to low-calorie, protein-sparing solid foods, vegetables, some fruits, and 480 mL of non-fat milk/day.

At a 4-month follow-up visit, after medical treatment for allergic rhinitis and sinusitis, and dietary control, snoring and arousals from sleep at night markedly decreased that might be caused by a decrease in the magnitude of nasal congestion, tonsillar enlargement and body weight. His body weight and height were 21.3 kg and 103 cm, and percentage of weight-for-height had decreased substantially at 133%.

## PATIENT 2

A 10-year-old boy presented complaining of numbness of both hands and feet for about 2 weeks, but no weakness or pain at all extremities. Dietary history revealed 3-4 meals/day composed of 4-5 ladles of polished steamed rice/time, fried rice, fried meat, fried eggs, cooked fermented fish with very few vegetables consumed, but 3-4 servings of various beverages each day, most commonly sweetened chocolate-flavored whole milk, sweetened drinking yogurt, sweetened soft drinks, and 2 servings of sweetened lemon iced tea, and fried potato chips. A physical examination revealed body weight 54 kg (>97<sup>th</sup> percentile), height 145 cm (90<sup>th</sup> percentile), weight-for-height 154%, normal pin-prick sensation and vibration sensation, normal muscle strength, normal gait and no diplopia. Other findings were unremarkable.

A blood sample analyzed for thiamin pyrophosphate effect (TPPE) showed a marked deficiency (61%) (reference range: 0-15%) representing severe thiamin deficiency. The boy was treated as an outpatient with a beginning oral daily dosage of thiamin, 1 100-mg tablet per day for 2 weeks, followed by half tablet daily for 4 weeks. Therapeutic lifestyle modifications including nutritional education (low-calorie, protein sparing diets with no tea), increased exercise and physical activity, decreased sedentary habits, and weight reduction were encouraged. At a 6-week follow-up visit, the numbness had disappeared and a TPPE appeared normal (1.5%). Additionally, he had lost 1.3 kg.

## PATIENT 3

An 8-year-old boy came to the hospital with cough and rhinorrhea. He had no history of abdominal pain and blood loss e.g. hemoptysis, hematemesis, or hematochezia. Dietary history revealed 3-4 meals/day, consisting of foods such as 2 ladles of steamed rice/time, fried eggs, fried chicken drumsticks, fried pork, cheese sausage, and cream of mushroom soup, sweetened beverages, sandwich cookies, and biscuits with sweetened condensed milk. On examination, his weight was 36 kg (>97<sup>th</sup> percentile), height 123.5 cm (25<sup>th</sup>-50<sup>th</sup> percentiles), weight-for-height 156%, and mild pallor and injected pharynx were noted. No jaundice, hepatosplenomegaly and acanthosis nigricans were found. Others findings were unremarkable.

A complete blood count showed Hb 10.6 g/dL, white

blood cells 20,260/mm<sup>3</sup> with neutrophils 87.9%, platelets 292,000/mm<sup>3</sup>, MCV 57.8 fL, MCH 18.2 pg, RDW 17.0%, and hypochromic and microcytic red blood cells. Urinalysis and stool exam did not demonstrate blood loss. Hemoglobin typing showed; A 85.6% and A<sub>2</sub> 2.3%. Serum iron concentration was 2.9 μmol/L (reference range: 9-29), total iron binding capacity (TIBC) 58.3 μmol/L (reference range: 45-70), and transferrin saturation 5% (reference range: 30-50). Further history found no family history of thalassemia. Therefore, a DNA blood test was positive for α-thalassemia 1 (--<sup>SEA</sup>/αα), but his anemia still could not be adequately explained.

The diagnoses of rhinopharyngitis, simple obesity, iron deficiency anemia, α-thalassemia-1 trait were made and the patient was put on ferrous sulfate at a daily dose of 130 mg elemental iron and vitamin C. Therapeutic lifestyle modifications were also suggested.

After 3.5 months of this treatment, he gained 2.6 kg, but his anemia had not improved much; tests at that time found Hb 11 g/dL, white blood cells 12,550/mm<sup>3</sup>, platelets 263,000/mm<sup>3</sup>, MCV 59.5 fL, MCH 19.5 pg, RDW 16.6%, and reticulocyte count 0.52%.

At 5.5 months of treatment, the patient's weight had increased to 39.2 kg. The iron deficiency anemia was not responding to the oral iron therapy, and after a 2-month cessation of iron treatment, a serum hepcidin sample was sent to the Netherlands to be measured by using a combination of weak cation exchange chromatography and time-of-flight mass spectrometry (<http://www.hepcidinanalysis.com>).<sup>16</sup> The result showed his serum hepcidin concentration was 16.2 nmol/L.

## DISCUSSION

In the present report, all three pediatric patients were obese and were diagnosed with different micronutrient deficiencies. Two of them had vitamin problems, ascorbic acid insufficiency and thiamin deficiency, and the other had iron deficiency anemia.

In regards to patient 1, the ascorbic acid insufficiency could have been the result of several factors. The patient had a very high consumption of UHT milk and French-fried potatoes in his diet and these foods contain no ascorbic acid, as it is destroyed by heat. Secondly, the boy had no consumption of vegetables and fruits, which are ascorbic acid-rich foods and the main source of vitamin C in the normal diet. Ketchup does contain a low concentration of ascorbic acid, but is also high in energy from sugar and high-fructose corn syrup, and high in sodium. Ascorbic acid deficiency can cause anorexia, leading to avoidance of other foods except for ascorbic acid-poor foods that aggravates an imbalance of food intake. This implies that poorly selective food choices occur not only to malnourished subjects but also to obese subjects. Mah *et al*<sup>7</sup> reported that male obese adults had 51% lower vitamin C intake and 38% lower plasma vitamin C concentration than male lean adults. They also found a significant association between low vitamin C status and pro-inflammatory response and impaired vascular function. Harnroongroj *et al*<sup>8</sup> reported higher prevalence of vitamin C deficiency in overweight and obese subjects than in normal weight subjects (51.5% vs 41.7%).

In Patient 2, dry beriberi was diagnosed due to the presence of subjective numbness, in spite of no sensory deficit detected during the medical examination. He had several risk factors which can contribute to thiamin deficiency. First, excessive consumption of starch (polished rice and potatoes) and sugar result in an increased requirement for thiamin, which functions as a coenzyme for carbohydrate metabolism. Second, polished rice is very low in thiamin because the bran is removed. Third, drinking green tea enhances a low thiamin status due to a thiamin antagonist in tea. Due to these risk factors, and our suspicions, a thiamin pyrophosphate effect blood test was done, which showed a significantly low thiamin status. The method for TPPE measurement was published in detail elsewhere.<sup>9</sup> Briefly, TPPE is *in vitro* assessment of thiamin status on the basis of activity of thiamin pyrophosphate on transketolase enzyme. Transketolase enzyme catalyzes the conversion of ribose-5-phosphate into sedoheptulose-7-phosphate, which requires thiamin pyrophosphate as a cofactor. The blood sample is preincubated in a condition, which use ribose-5-phosphate as a substrate with and without thiamin pyrophosphate. Erythrocyte transketolase activity is studied by measuring the number of sedoheptulose-7-phosphate formed in the reaction per minute per litre. The TPPE is calculated from the percentage of increase in sedoheptulose-7-phosphate after adding thiamin pyrophosphate. In thiamin deficiency, erythrocyte transketolase activity decreases, but the TPPE increases. The TPPE of greater than 15% suggests a thiamin deficiency. Nerve studies, electromyography and nerve conduction velocity test can show peripheral neuropathy, but electrodiagnostic studies were not performed in this case. The patient responded well to the thiamin treatment. This result also suggests poor food choices of obese children. In Patient 3, the boy's anemia was mostly caused by a decrease in intestinal iron absorption, which was indirectly explained by a very high concentration of plasma hepcidin. The reference concentrations of plasma hepcidin of normal weight Thai children are not available, but the boy's plasma hepcidin concentration of 16.2 nmol/L was markedly higher than the average concentrations of healthy non-obese Egyptian children ( $1.61 \pm 0.7$  nmol/L),<sup>10</sup> lean Italian children ( $1.9 \pm 1.6$  nmol/L),<sup>11</sup> and Swiss normal weight children (median 1.4, min-max: 0.4-6.1 nmol/L).<sup>1</sup> A number of studies reported that overweight and obese children had a higher prevalence of iron deficiency and a higher serum hepcidin concentration compared with non-obese children in spite of no difference in dietary iron intake.<sup>1,2,10,12,13</sup> Compared to healthy, non-obese children, serum hepcidin was significantly greater in obese children with iron deficiency anemia (IDA) and significantly lower in non-obese children with IDA.<sup>10</sup> Sanad *et al*<sup>10</sup> reported that after iron treatment serum hepcidin increased in non-obese children with IDA but no changes in obese children with IDA were observed. A mechanistic link between obesity and poor iron status has been proposed.<sup>14</sup> Obesity is a chronic inflammatory state because macrophages in adipose tissues synthesize pro-inflammatory cytokines including interleukine-6 and tumor necrosis factor- $\alpha$ . These cytokines lead to increased hepcidin release/synthesis from the liver and adipocytes. Hepcidin functions by degrading a ferroportin,

which is a cellular iron exporter at basolateral membrane of duodenal enterocytes and macrophages, resulting in inhibition of iron flow into plasma from dietary iron-absorbing enterocytes and iron-recycling macrophages.<sup>15</sup> This leads to impaired dietary iron repletion and reduced iron stores causing the anemia of chronic inflammation condition. Sanad *et al*<sup>10</sup> reported increased serum hepcidin concentration at baseline in obese children with IDA, which suggested that it was not suppressed in iron deficiency. Furthermore, there was no significant change in serum hepcidin after iron therapy in obese children with IDA that reflected no normal negative feedback to regulate hepcidin synthesis from iron treatment in contrast to a response in non-obese children with IDA. In addition, Amato *et al*<sup>16</sup> showed that after weight reduction obese children had significantly reduced serum hepcidin concentration and increase in iron absorption and status. Therefore the significance of weight reduction in addition to iron treatment would be emphasized for management for obese children with iron deficiency. In this study we did not measure serum interleukine-6 or tumor necrosis factor- $\alpha$  levels of Patient 3 owing to unavailability of the tests; we did, however, explain to the patient and his parents that we suspected the pathogenesis of his anemia was his obesity, and we gave them information concerning an importance of therapeutic lifestyle changes for his weight management and consequent anemia.

Besides the problems of ascorbic acid, thiamin, and iron deficiency, there were other micronutrient deficiencies in obese subjects e.g. carotenoids, vitamin D, vitamin E, and calcium. We summarized published studies of micronutrient deficiencies in obese children and adults in Table 1.

Vitamin and mineral deficiencies, due to either an unbalanced diet or adiposity-associated inflammation, are commonly overlooked in obese children and adolescents. Although weight management strategy is emphasized, implications of these micronutrient deficiencies would be considered in the treatment of obesity in children. The details of dietary intake including quality, quantity and frequency of meals, beverages, and snack are important to be assessed as a part of routine health care in children.

#### AUTHOR DISCLOSURES

The authors declare that there was no conflict of interest. This study was supported by a "Chalermphrakiat" Grant, from the Faculty of Medicine Siriraj Hospital, Mahidol University.

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**Table 1.** Published studies of micronutrient deficiencies in obese subjects

Reference/Year	Study group	Study design	Micronutrient deficiencies	Results
Mah <i>et al</i> /2011 <sup>7</sup>	Male, age 18-35 years, obese (n=8) vs lean (n=8)	Cross-sectional study	Vitamin C	- 51% lower vitamin C intake and 38% lower plasma vitamin C concentration in obese men than in lean men - 21% lower FMD response but greater plasma concentrations of CRP, MPO, and inflammatory cytokines in obese men than in lean men - Significant association between low vitamin C status and pro-inflammatory response and impaired vascular function
Harnroongroj <i>et al</i> /2002 <sup>8</sup>	Both sex, age range 18-60 years, overweight and obese (n=270) vs normal weight (n=175)	Cross-sectional study	Vitamin C	- Higher prevalence of vitamin C deficiency in overweight and obese subjects than in normal weight subjects (51.5% vs 41.7%)
Aeberli <i>et al</i> /2009 <sup>1</sup>	Both sex, age 6-14 years, overweight (n=85) vs normal weight (n=33)	Cross-sectional study	Iron	- No difference in dietary iron intake - Higher prevalence of iron-deficient erythropoiesis (20% vs 6%) and significantly higher serum hepcidin levels in the overweight children than normal weight children
Moayeri <i>et al</i> /2006 <sup>2</sup>	Both sex, age 11-17 years, overweight and obese (n=526) vs normal weight (n=200)	Cross-sectional study	Iron	- Significant increase in prevalence of iron deficiency from normal to overweight and to obese children (2.5%, 5.3%, and 6.9%, respectively)
Sanad <i>et al</i> /2011 <sup>10</sup>	Both sex, IDA (35 obese aged 6.96±2.2 years, 35 non-obese aged 7.11±2.57 years) vs 30 healthy non-obese aged 7.11±2.57 years	Prospective case control study	Iron	- Higher serum hepcidin in obese children with IDA but lower in non-obese children with IDA compared to the healthy non-obese children - Increase in hepcidin in non-obese children with IDA but no change in obese children after iron treatment
Tussing-Humphreys <i>et al</i> /2009 <sup>12</sup>	Female, age 12-17 years, heavier weight (n=81) vs normal weight (n=129)	Cross-sectional study	Iron	- Higher prevalence of iron deficiency in heavier weight girls than those with normal weight (30.8% vs 14%) - Increasing BMI and CRP predicted increased OR for iron deficiency more than doubled
Baumgartner <i>et al</i> /2012 <sup>13</sup>	Both sex with iron deficiency, age 6-11 years, normal weight (n=230), overweight and obese (n=91)	Randomized placebo-controlled, double-blind study, iron vs placebo	Iron	- Significant associations between BAZ and CRP at baseline, and between baseline CRP and hepcidin - Significant correlation between baseline hepcidin and endpoint sTfR (poorer iron status) only in the overweight and obese group - A two-fold higher risk of remaining iron deficiency after iron supplementation in subjects with high BAZ
Amato <i>et al</i> /2010 <sup>16</sup>	Both sex, age 9-16 years, 15 obese children	Intervention study (before and after)	Iron	- After a 6-month weight loss program, there were a reduction in BMI-SDS, serum hepcidin, and serum leptin levels, but an increase in iron absorption
Neuhouser <i>et al</i> /2001 <sup>4</sup>	Both sex, age 12-17 years, normal weight (n=190) vs obese (n=39)	Cross-sectional study	Carotenoids	- 2-9% lower in serum $\alpha$ -carotene and $\beta$ -carotene concentrations among obese adolescents compared to normal weight adolescents

FMD: flow-mediated dilation, CRP: C-reactive protein, MPO: myeloperoxidase, HOMA-IR: homeostasis model assessment of insulin resistance, BMI: body mass index, OR: odds ratio, IDA: iron deficiency anemia, BAZ: BMI-for-age Z-scores, sTfR: soluble transferrin receptor, BMI-SDS: body mass index standard deviation score.

**Table 1.** Published studies of micronutrient deficiencies in obese subjects (cont.)

Reference/Year	Study group	Study design	Micronutrient deficiencies	Results
Olson <i>et al</i> /2012 <sup>3</sup>	Both sex, age 6-16 years, obese (n=411) vs non-overweight (n=87)	Cross-sectional study	Vitamin D	<ul style="list-style-type: none"> <li>- Higher prevalence of vitamin D deficiency in obese children than non-overweight children (50% vs 22%)</li> <li>- Serum 25(OH)D level was negatively associated with soda intake, skipping breakfast, and juice intake</li> <li>- Negative correlation between serum 25(OH)D level and HOMA-IR, and between serum 25(OH)D level and 2-h glucose level in obese group</li> </ul>
Khor <i>et al</i> /2011 <sup>5</sup>	Both sex, age 7-12 years, n=402	Cross-sectional study	Vitamin D	Inverse association between serum 25(OH)D status and BMI-for-age among the boys
Poomthavorn <i>et al</i> /2012 <sup>6</sup>	Both sex, obese (n=150, age 11.2±2.6 years) vs healthy non-obese (n=29, age 8.7±1.5 years)	Cross-sectional study	Vitamin D	<ul style="list-style-type: none"> <li>- Prevalence of vitamin D deficiency was 11.3% and 10.3% in obese and healthy non-obese children, respectively.</li> <li>- No association between serum 25(OH)D status and body weight, height, BMI standard deviation score, or insulin sensitivity index</li> </ul>
Al-Musharaf <i>et al</i> /2012 <sup>17</sup>	Both sex, age 6-17 years, normal weight group (n=207) vs overweight/obese (n=124)	Cross-sectional study	Vitamin D & Calcium	<ul style="list-style-type: none"> <li>- No difference in serum vitamin D concentrations between normal weight group and overweight/obese group in both children and adolescents</li> <li>- Highest vitamin D status among children who consumed calcium &gt; 800 mg/day, compared to those who consumed calcium &lt; 250 mg/day</li> <li>- Tendency of being highest BMI in adolescents who consumed calcium &lt; 250 mg/day and lowest BMI in who consumed calcium &gt; 800 mg/day</li> </ul>
Neuhouser <i>et al</i> /2001 <sup>4</sup>	Both sex, age 12-17 years, normal (n=190) vs obese (n=39)	Cross-sectional study	Vitamin E	- 10% lower in serum $\alpha$ -tocopherol concentration among obese adolescents compared to normal weight adolescents

FMD: flow-mediated dilation, CRP: C-reactive protein, MPO: myeloperoxidase, HOMA-IR: homeostasis model assessment of insulin resistance, BMI: body mass index, OR: odds ratio, IDA: iron deficiency anemia, BAZ: BMI-for-age Z-scores, sTfR: soluble transferrin receptor, BMI-SDS: body mass index standard deviation score.

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## 微量營養素缺乏的肥胖泰國兒童

這篇個案報告三位呈現佝僂型串珠、手腳麻木，以及小球性低色素貧血之不同程度症狀的肥胖兒童。檢測血清抗壞血酸濃度、紅血球硫胺焦磷酸鹽作用及血清鐵調節素濃度，分別顯示抗壞血酸不足、硫胺缺乏以及高發炎狀態致使鐵吸收下降造成之缺鐵性貧血。這份報告指出肥胖兒童，不僅熱量攝取過多，還可能有微量營養素缺乏的風險。因此，體重管理時需特別注意維生素及礦物質的給予是否足夠。

**關鍵字：**抗壞血酸、鐵調節素、缺鐵性貧血、肥胖兒童、硫胺缺乏