

Severe Megaloblastic Anemia Secondary to Folate Deficiency in a 2-Year-Old Boy with Prolonged Predominant Goat Milk Feeding: A Case Report from a Low-Resource Setting

Salad Abdullahi Farah¹ , Fartuun Abdullahi Hassan Orey¹ , Abdullahi Hassan Elmi² ,
Nima Ahmed Mohamed¹

¹Department of Pediatrics and Child Health, Dr Sumait Hospital, Faculty of Medicine and Health Sciences, SIMAD University, Mogadishu, Somalia;

²Department of Nursing and Midwifery, Dr Sumait Hospital, Faculty of Medicine and Health Sciences, SIMAD University, Mogadishu, Somalia

Correspondence: Salad Abdullahi Farah, Email saladabdullahif@simad.edu.so

Introduction: Megaloblastic anemia in young children is a significant but preventable condition, commonly caused by deficiencies in folate or vitamin B12. Prolonged predominant feeding with nutritionally inadequate milk substitutes, such as unfortified goat milk, increases the risk of severe nutritional anemia, particularly in low-resource settings.

Case Presentation: We report a 2-year-old Somali boy who presented with a 5-month history of fatigue, recurrent fever, poor appetite, abdominal distension, progressive weight loss, and cough. The child had a history of prolonged predominant goat milk feeding with limited dietary diversity. On admission, he was critically ill, severely pale, tachycardic, tachypneic, and hypoxic, with an oxygen saturation of 78% on room air. Clinical examination also revealed bilateral pedal edema, tender hepatomegaly, and a gallop rhythm, suggesting cardiac compromise secondary to profound anemia. Laboratory evaluation showed severe anemia with a hemoglobin level of 2.4 g/dL, thrombocytopenia, megaloblastic changes on peripheral smear, profoundly reduced serum folate (<0.8 ng/mL), normal vitamin B12 and ferritin levels, and positive Plasmodium antigen testing. The child was managed with oxygen support, packed red blood cell transfusions, folic acid and iron supplementation, intravenous artesunate, nutritional rehabilitation, and caregiver counseling. His clinical condition improved, and follow-up showed hematologic recovery with hemoglobin increasing to 11 g/dL and serum folate improving to 4 ng/mL.

Discussion: This case highlights the systemic impact of folate deficiency in children and the dangers of Prolonged predominant goat milk feeding can lead to life-threatening megaloblastic anemia in young children, especially in low-resource environments. The condition is preventable through early recognition, supplementation, and caregiver education. Public health strategies should promote culturally sensitive nutrition awareness programs to prevent similar cases in at-risk communities.

Conclusion: Exclusive goat milk feeding can lead to life-threatening megaloblastic anemia in young children, especially in low-resource environments. Early identification, nutritional intervention, and caregiver education are essential to prevent severe complications and ensure optimal growth and development.

Keywords: megaloblastic anemia, folate deficiency, prolonged goat milk feeding, pediatric nutritional anemia, Somalia, sub-Saharan Africa, low-resource setting, case report

Introduction

Anemia remains one of the most common and preventable public health problems among infants and young children worldwide. Recent global estimates indicate that anemia continues to affect a substantial proportion of children under five years of age, with the greatest burden reported in low- and middle-income countries, particularly across sub-Saharan Africa and South Asia.¹ In these settings, childhood anemia is often multifactorial, resulting from nutritional deficiencies, malaria and other infections, poverty, food insecurity, limited access to healthcare, and inadequate dietary diversity. In

early childhood, anemia is especially concerning because it may impair growth, neurodevelopment, immune function, and overall child survival. Therefore, early recognition of nutritional and infectious contributors is essential for preventing severe complications.¹

Megaloblastic anemia is a distinct subtype of anemia characterized by the presence of abnormally large, immature red blood cells in the bone marrow and peripheral circulation. It results primarily from impaired DNA synthesis, which occurs due to deficiencies in folate or vitamin B12.^{2,3} These vitamins are essential cofactors in nucleotide synthesis and red blood cell production. When either of them is deficient, cell division is disrupted, leading to ineffective erythropoiesis and the classic hematologic findings of megaloblastic anemia.²

Folate deficiency during infancy and early childhood is especially concerning because these periods are marked by rapid growth, increased cellular turnover, and high metabolic demands. The deficiency may arise from a variety of factors, including inadequate dietary intake, intestinal malabsorption, chronic illness, or exclusive feeding practices that fail to meet the child's nutritional requirements.⁴ Infants who are not breastfed or whose diets are restricted to non-fortified foods are at heightened risk.

Goat milk is sometimes used as an alternative feeding source for infants and young children because it is perceived by some caregivers as natural, easily tolerated, or culturally acceptable. However, unfortified goat milk is nutritionally incomplete for young children and is particularly poor in folate compared with human milk and cow's milk.⁵ Goat milk contains only a very small amount of folate, while breast milk and cow's milk contain substantially higher concentrations. This marked difference helps explain why prolonged reliance on goat milk without appropriate complementary feeding or supplementation can result in severe folate depletion. Goat milk-associated megaloblastic anemia, sometimes referred to historically as "goat milk anemia," has been recognized in the medical literature for many decades. Despite being preventable, it remains clinically relevant in settings where caregivers may use unfortified animal milk because of cultural beliefs, poverty, limited nutrition education, or restricted access to appropriate infant feeding alternatives.⁵

This case report describes a 2-year-old Somali boy who developed severe folate-deficiency megaloblastic anemia in the context of prolonged predominant goat milk feeding, limited dietary diversity, concurrent malaria infection, and clinical features suggestive of high-output cardiac failure. The case is important because it illustrates how preventable nutritional deficiency may present with life-threatening hematologic and systemic complications in a low-resource setting. Although goat milk-associated megaloblastic anemia has been previously described, reports from Somalia remain limited. To our knowledge, this is among the first systematically described pediatric case reports from Somalia documenting profound folate-deficiency megaloblastic anemia associated with prolonged predominant goat milk feeding, complicated by concurrent malaria infection and features of cardiac compromise. The case highlights the need for detailed feeding history, early micronutrient assessment, infection screening in endemic settings, and culturally sensitive caregiver education.

Case Presentation

A 2-year-old boy from a rural community was admitted with a 5-month history of recurrent high-grade fever, non-bilious vomiting, poor appetite, progressive weight loss, palpitations, abdominal distension, bilateral pedal edema, and persistent cough. His caregivers reported several previous hospital visits, during which he had been treated for malaria and pneumonia without sustained clinical improvement. The feeding history revealed prolonged predominant goat milk feeding from early infancy, with limited dietary diversity and inadequate intake of folate-rich complementary foods. According to the caregivers, goat milk was used because of cultural preference, household accessibility, and limited access to infant formula or fortified alternatives. Available history did not suggest regular intake of fortified cereals, green leafy vegetables, legumes, or other folate-rich foods.

On admission, he appeared critically ill, febrile at 39°C, severely pale, dehydrated, and hypoxic, with an oxygen saturation of 78% on room air. He had bilateral pedal edema and marked conjunctival pallor, with no lymphadenopathy, jaundice, or cyanosis. He was tachypneic, with a respiratory rate of 60 breaths/min, and tachycardic, with a heart rate of 154 beats/min. Heart sounds S1 and S2 were audible, with a gallop rhythm. Abdominal examination revealed a soft, non-tender abdomen with tender hepatomegaly. Because the child had progressive weight loss, bilateral pedal edema, and hepatomegaly, the previous statement that he appeared well nourished was removed to avoid overinterpretation. Objective nutritional assessment was limited; however, the available clinical findings suggested possible nutritional compromise rather than normal nutritional status.

Anthropometric assessment on admission showed a weight of 12.1 kg and a length of 86 cm. The calculated weight-for-age Z-score (WAZ) was approximately -0.2 , and the height-for-age Z-score (HAZ) was -0.1 , both within the normal range for age. The weight-for-height Z-score (WHZ) was $+0.1$, indicating appropriate weight relative to height. The mid-upper arm circumference (MUAC) measured 13.5 cm, consistent with normal nutritional status. The head circumference was 48 cm, appropriate for age. Overall, these findings are consistent with adequate nutritional status, with no anthropometric evidence of acute or chronic malnutrition. Available caregiver history did not report previous developmental regression, seizures, or known neurodevelopmental delay; however, a formal motor and cognitive developmental assessment was not performed. Perinatal history, including gestational age, birth weight, and maternal folate status during pregnancy, was not fully documented. There was no known family history of similar illness, hereditary hematologic disease, or affected siblings. Consanguinity was not assessed.

Initial laboratory investigations revealed profound anemia, with a hemoglobin concentration of 2.4 g/dL, and thrombocytopenia, with a platelet count of $48 \times 10^9/L$. The white blood cell count was $8.42 \times 10^9/L$, and C-reactive protein was <2.5 mg/L. Peripheral blood smear demonstrated megaloblastic changes, supporting a diagnosis of megaloblastic anemia. Serum folate was profoundly reduced at <0.8 ng/mL, while serum vitamin B12 was within the reference range at 282.23 pg/mL and serum ferritin was 113.53 ng/mL. Malaria antigen testing was positive, while serologic tests for tuberculosis and dengue were negative. Renal and hepatic function tests were largely unremarkable. Serum creatinine was slightly below the reference range at 0.39 mg/dL; this was not interpreted as renal impairment and may reflect age-related variation or reduced muscle mass in the context of prolonged nutritional illness and weight loss. White blood cell count and C-reactive protein were within the reference range. Available laboratory findings are summarized in Table 1. Additional hematologic indices

Table 1 Laboratory Investigations at Admission, Ward Discharge, and Outpatient Follow-Up

Test	Reference Range	On Admission	On Ward Discharge	On Follow-Up
White cell count, WBC ($\times 10^9/L$)	4.00–12.00	8.42	8.34	Not performed
Hemoglobin, Hb (g/dL)	12.0–16.0	2.4	8.0	11.0
Platelet count, PLT ($\times 10^9/L$)	100–300	48	148	
Mean corpuscular volume, MCV (fL)	75–87	112		
Mean corpuscular hemoglobin, MCH (pg)	24–30	36		
Mean corpuscular hemoglobin concentration, MCHC (g/dL)	32–36	32		
Red cell distribution width, RDW (%)	11.5–14.5	18.5		
Reticulocyte count (%)	0.5–2.5	0.4		
C-reactive protein, CRP (mg/L)	2.5–10	<2.5		
Alanine transaminase, ALT (U/L)	6–40	11.8		
Creatinine, Crt (mg/dL)	0.4–1.4	0.39		
Sodium, Na^+ (mmol/L)	135–145	135.2		
Potassium, K^+ (mmol/L)	3.5–5.5	4.81		
Vitamin B12 (pg/mL)	180–916	282.23		
Folic acid (ng/mL)	2.0–15.0	<0.8	1.8	4.0
Ferritin (ng/mL)	30–350	113.53		
Blood group	ABO/Rhesus	O positive		

Abbreviations: ALT, alanine transaminase; CRP, C-reactive protein; Crt, creatinine; Hb, hemoglobin; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; PLT, platelet count; RDW, red cell distribution width; WBC, white blood cell count.

showed an MCV of 112 fL, MCH of 36 pg, MCHC of 32 g/dL, and RDW of 18.5%, supporting macrocytic/megaloblastic anemia. Reticulocyte count was 0.4% on admission, indicating inadequate marrow response, which is typical in untreated megaloblastic anemia.

Overall, the clinical and laboratory findings were most consistent with severe folate-deficiency megaloblastic anemia associated with prolonged predominant goat milk feeding, with concurrent malaria infection and clinical features suggestive of high-output cardiac failure secondary to profound anemia. The child was initially stabilized with supplemental oxygen for hypoxia and careful intravenous fluid therapy for dehydration. Because of profound anemia and clinical features of cardiac compromise, packed red blood cell transfusion was administered cautiously at 10 mL/kg per transfusion, with close monitoring for respiratory distress and volume overload. His hemoglobin increased from 2.4 g/dL on admission to 8 g/dL after one week of inpatient management. Oral folic acid supplementation was initiated during admission at a dose of 1 mg once daily and continued after discharge. Oral ferrous sulfate at 3 mg/kg/day and multivitamins, including B-complex vitamins, were also prescribed to support hematologic recovery and nutritional rehabilitation. Although ferritin was within the reference range, iron supplementation was provided to support increased erythropoietic demand during marrow recovery after correction of folate deficiency and transfusion.

Concurrent malaria infection was treated with intravenous artesunate according to local pediatric malaria management practice; however, the exact dose and duration were not fully documented in the available clinical record. During hospitalization, the child's clinical condition gradually improved; within four days, oxygenation normalized, fever subsided, appetite improved, and pedal edema decreased. Concurrent malaria infection was treated with intravenous artesunate according to local pediatric malaria management practice. During hospitalization, the child's clinical condition gradually improved; within four days, oxygenation normalized, fever subsided, appetite improved, and pedal edema decreased. Oral folic acid supplementation was initiated during admission after confirmation of profound folate deficiency and was continued after discharge at a dose of 1 mg once daily. Serum folate improved from <0.8 ng/mL on admission to 1.8 ng/mL at ward discharge and 4 ng/mL at outpatient follow-up. During the same period, hemoglobin increased from 2.4 g/dL on admission to 8 g/dL at discharge and 11 g/dL at follow-up. Caregivers received counseling on the risks of prolonged goat milk–dominant feeding and practical guidance on introducing a balanced, age-appropriate, folate-rich diet. He was discharged in stable condition with oral folic acid, ferrous sulfate, multivitamins, nutritional counseling, and close outpatient follow-up.

Discussion

This case highlights a severe and preventable form of nutritional megaloblastic anemia in a young child with prolonged predominant goat milk feeding in a low-resource setting. Although goat milk–associated megaloblastic anemia has been described previously, this case is clinically important because of the profound anemia, concurrent malaria infection, thrombocytopenia, hypoxemia, and clinical features suggestive of high-output cardiac failure.^{5–7} Reports from Somalia remain limited, and this case adds context-specific evidence on how nutritional deficiency, infectious disease, and delayed access to diagnostic services may overlap in pediatric practice.

Folate is essential for one-carbon metabolism and DNA synthesis. In its active forms, including tetrahydrofolate and 5-methyltetrahydrofolate, folate supports purine synthesis, thymidylate formation, and methylation reactions. Deficiency impairs nuclear maturation in rapidly dividing cells, particularly hematopoietic precursors, resulting in ineffective erythropoiesis and megaloblastic changes in the peripheral blood and bone marrow.⁸ This mechanism explains the patient's profound anemia and thrombocytopenia. In young children, prolonged folate deficiency may also affect growth, immune function, and neurodevelopment, making early recognition and treatment essential.⁸

The differential diagnosis in this child included severe iron deficiency anemia, vitamin B12 deficiency, malaria-associated anemia, hemolytic anemia, aplastic anemia, leukemia, hereditary hemoglobinopathies, and inherited or malabsorptive causes of megaloblastic anemia. Normal ferritin and vitamin B12 levels, profoundly reduced serum folate, megaloblastic changes on peripheral smear, and hematologic improvement after folic acid supplementation supported folate deficiency as the main diagnosis.⁷ However, in a sub-Saharan African setting, hemoglobinopathies such as sickle cell disease and thalassemia, G6PD deficiency, chronic hemolysis, and malaria-related anemia remain important considerations. Hereditary folate malabsorption, thiamine-responsive megaloblastic anemia, congenital transcobalamin disorders, and celiac disease were also considered theoretically, but advanced testing was not available. These diagnostic constraints are acknowledged in the Limitations section.⁷

Concurrent malaria infection likely contributed to the severity of anemia. *Plasmodium* infection can worsen anemia through hemolysis of infected and uninfected red blood cells, inflammatory suppression of erythropoiesis, splenic clearance, and dyserythropoiesis.^{4,6} In this patient, the profound folate deficiency was considered the primary driver of megaloblastic anemia, but malaria may have acted as an additional stressor by increasing red blood cell destruction and impairing marrow recovery. The coexistence of folate deficiency and malaria is clinically important in endemic settings because both conditions may interact and produce more severe anemia than either condition alone.^{4,6}

The child's hypoxemia, tachycardia, gallop rhythm, bilateral pedal edema, and hepatomegaly were consistent with cardiac strain, most likely high-output cardiac failure secondary to profound anemia. In severe anemia, reduced oxygen-carrying capacity leads to compensatory increases in cardiac output, which may eventually result in decompensation, especially in young children.^{2,6} This clinical picture also has important management implications because transfusion must be given cautiously to avoid volume overload. In this case, packed red blood cells were administered in small-volume transfusions with clinical monitoring, and the child's respiratory status and edema improved during hospitalization.^{2,6,7}

Previous reports of goat milk-associated megaloblastic anemia have similarly described young children presenting with severe anemia after prolonged reliance on unfortified goat milk. These cases consistently show that the condition is preventable when caregivers receive early feeding guidance, micronutrient supplementation, and access to appropriate complementary foods.^{1,2,9,10} The present case differs by documenting an extremely low hemoglobin level, concurrent malaria infection, and clinical features of cardiac compromise in a Somali child. These features strengthen the educational value of the report and highlight the need for clinicians in low-resource and malaria-endemic settings to assess both nutritional and infectious causes when evaluating severe pediatric anemia.^{9,10}

From a public health perspective, this case reinforces the need for culturally sensitive nutrition education in communities where unfortified animal milk is commonly used for young children. Counseling should not only discourage prolonged dependence on goat milk as a main food source but should also provide practical alternatives, including breastfeeding support, age-appropriate complementary feeding, folate-rich foods, and micronutrient supplementation where needed. Integrating feeding assessment into routine pediatric visits may help identify at-risk children before severe anemia develops.^{3,8,9}

This case report has several limitations. First, complete long-term follow-up data were not available, which limited assessment of sustained growth, hematologic, and neurodevelopmental outcomes. Second, although basic hematologic indices were obtained, several investigations that could have further strengthened diagnostic confirmation, including LDH, bilirubin fractions, haptoglobin, direct Coombs test, G6PD activity, hemoglobin electrophoresis, homocysteine, methylmalonic acid, echocardiography, chest radiography, thyroid function testing, and genetic testing, were not available because of resource limitations. Third, a peripheral blood smear photomicrograph was not available for inclusion. Despite these limitations, the clinical history, prolonged predominant goat milk feeding, profoundly low serum folate, normal vitamin B12 level, macrocytic indices, compatible peripheral smear findings, low reticulocyte response, and hematologic recovery after folic acid supplementation strongly support folate-deficiency megaloblastic anemia.

Conclusion

This case emphasizes that prolonged predominant goat milk feeding can lead to life-threatening folate-deficiency megaloblastic anemia in young children, particularly in low-resource settings where nutritional counseling and laboratory access may be limited. In children presenting with severe anemia or pancytopenia, clinicians should obtain a detailed feeding history, assess serum folate and vitamin B12 where available, and screen for concurrent infections such as malaria in endemic regions. Early folate replacement, cautious transfusion support, nutritional rehabilitation, and caregiver counseling can result in substantial clinical and hematologic recovery. Public health efforts should promote culturally sensitive education on infant and young-child feeding, safe alternatives to unfortified animal milk, and micronutrient supplementation for vulnerable children.

Abbreviations

Hb, hemoglobin; CV, mean corpuscular volume; RBC, red blood cell; WBC, white blood cell; CRP, C-reactive protein; WAZ, weight-for-age Z-score; HAZ, height-for-age Z-score; WHZ, weight-for-height Z-score; MUAC, mid-upper arm

circumference; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; RDW, red cell distribution width.

Ethics and Consent

Written informed consent for publication of this case report and the accompanying images was obtained from the patient's parent because the patient was younger than 18 years. In accordance with Dr. Sumait Hospital/SIMAD University institutional policy, ethical approval was not required for this single case report because it involved anonymized clinical information and written parental consent for publication was obtained.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors declare that they have no conflicts of interest related to this study.

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