D J Med Sci 2026;12(2):105-110
doi: 10.5606/fng.btd.2026.245

Case Report

Infantile vomiting caused by vitamin B12 deficiency: A case report

Cenk Çelik[1], Ece Çetin[2], Ayça Vitrinel[3]

[1]Department of Pediatrics, Beykent University Faculty of Medicine, İstanbul, Türkiye
[2]Department of Pediatrics, Acıbadem International Hospital, İstanbul, Türkiye
[3]Department of Pediatrics, Yeditepe University Faculty of Medicine, İstanbul, Türkiye

ABSTRACT

Vitamin B12 deficiency is an important but often overlooked cause of feeding difficulties, vomiting, and neurodevelopmental delay in early infancy. Early diagnosis and treatment are essential to prevent permanent neurological damage. We report the case of a 49-day-old male infant admitted with restlessness, refusal to feed, and recurrent non-projectile vomiting after breastfeeding. The patient was exclusively breastfed. The family medical history was unremarkable. The mother was on a cow's milk elimination diet, and the patient was on anti-reflux medical treatment with approaches to prevent overfeeding and reflux. The volume and frequency of the vomiting were gradually increasing. Lethargy, hypotonia, and hypoactivity were notable on initial assessment. No abnormal hematological values were found, and serum vitamin B12 levels were low. The mother's serum vitamin B12 levels were also found to be inadequate. The infant's symptoms improved with parenteral hydroxycobalamin. In conclusion, this case underlines the importance of considering vitamin B12 deficiency in the differential diagnosis of persistent vomiting and neurodevelopmental delay in infants, particularly in settings where maternal malnutrition is prevalent. Prompt diagnosis and appropriate treatment lead to rapid clinical improvement and prevent long-term complications. Clinician awareness and early screening strategies in high-risk populations could play a crucial role in improving outcomes.

Keywords: Case report, hypotonia, vitamin B12 deficiency, vomiting infant.

Vitamin B12 is an essential water-soluble vitamin with important cellular functions. Vitamin B12, also known as cobalamin, plays an important role in nucleic acid and protein synthesis, carbohydrate and lipid metabolism, mitochondrial functions, erythroid formation, and nerve fiber myelination. Vitamin B12 is essential for humans and cannot be synthesized in the body. Infancy is a risky period for vitamin B12 deficiency. Maternal vitamin deficiency due to a vegetarian diet or low socio-economic status is the most common cause of deficiency during this period. Non-specific symptoms such as growth retardation, restlessness, hypotonia, convulsions, hyperpigmentation, and rejection of solid foods may occur in deficient infants.[1] As long-term vitamin B12 deficiency can lead to irreversible neurological damage, it is very important to diagnose and intervene at an early stage.

Unlike most previously reported cases of infantile cobalamin deficiency, which typically present between two and 12 months of age with predominantly hematological or neurological features, our patient became symptomatic as early as 49 days of life, with recurrent vomiting as the leading complaint and without anemia or macrocytosis.[1] Notably, the deficiency arose from clinically silent maternal insufficiency, the mother had no anemia or neurological symptoms, and the presentation closely mimicked gastrooesophageal reflux and cow's-milk protein allergy, resulting in a diagnostic delay. This case, therefore, highlights vitamin B12 deficiency as

Received: June 24, 2026
Accepted: July 22, 2026
Published online: August 31, 2026

Correspondence: Cenk Çelik, MD.
E-mail: cnkclk@outlook.com.tr

Cite this article as:
Çelik C, Çetin E, Vitrinel A. Infantile vomiting caused by vitamin B12 deficiency: A case report. D J Med Sci 2026;12(2):105-110. doi: 10.5606/fng.btd.2026.245.

an under-recognized cause of vomiting in early infancy that is easily missed when maternal deficiency is occult and underscores the importance of early recognition and treatment to prevent irreversible neurodevelopmental damage.

CASE REPORT

A 49-day-old male infant was admitted to the outpatient clinic with complaints of restlessness, refusal to feed, and vomiting. It was noted that the vomiting was mainly after feeding and gradually increased over the last one month. It was learned that the vomit contained no blood or bile and was not projectile. His diet consisted entirely of breastfeeding. There was no history of any trials with infant formula or other foods.

The patient had no family history of any illness. It was learned that he visited the health facility three weeks ago due to post-feeding vomiting and sudden breathlessness. Anteroposterior chest radiograph, abdominal ultrasound, urine test, and urine culture, which were performed with a pre-diagnosis of food aspiration, were normal. It was learned that anti-reflux medical treatment, including sodium alginate and potassium bicarbonate, was started along with a cow's milk elimination diet and training on how to prevent overfeeding and reflux. However, the volume and frequency of vomiting gradually increased in spite of treatment.

The patient's medical history revealed that the patient was born healthy at 36 weeks by cesarean section, and that the mode of delivery was cesarean due to preterm labor and the mother's previous cesarean section. There was no history of birth trauma or asphyxia. Birth weight was 2860 g (50-90p), height 49 cm (50-90p), and head circumference 33 cm (50-90p). It was noted that he had received phototherapy for neonatal jaundice on postnatal day three and that transcranial sonography was normal during this period. A written informed consent was obtained from the parent of the patient.

Clinical findings

At presentation, the patient's weight was 4520 g (50-90p), height 55 cm (50-90p) and head circumference 37 cm (50-90p). Lethargy, hypotonia, and hypoactivity were noted on neurological examination. Age-appropriate head control and visual tracking had not yet been attained. Other system examinations showed no pathological features.

Diagnostic assessment

Laboratory evaluation revealed hemoglobin 11.7 g/dL, hematocrit 33.2%, mean corpuscular volume 93.3 f/L, total bilirubin 4 mg/dL, and direct bilirubin 0.4 mg/dL. Other general biochemistry tests and venous blood gas analysis were unremarkable. The levels of C-reactive protein, thyroid-stimulating hormone, and free T4 were all normal. Serum vitamin B12 was low, with a value of 97 pg/mL and serum folate was normal with, a value of 15 ng/mL. Due to technical limitations, methylmalonic acid and homocysteine levels could not be measured. The serum vitamin B12 level of the mother was also found to be low, with 162 pg/mL. The mother had no neurological symptoms or hematological laboratory findings of anemia. The mother's dietary history showed that she had poor access to animal foods. Taken together, the absence of anemia or macrocytosis, normal folate, and unremarkable inflammatory and endocrine screening argued against infectious and endocrine causes; a normal venous blood gas made a decompensated organic acidemia less likely; and abdominal ultrasound had already excluded hypertrophic pyloric stenosis in an infant with non-projectile vomiting. In the presence of a compatible clinical picture, a low serum vitamin B12 concentration and a concordantly low maternal serum vitamin B12 level, cobalamin deficiency was considered the most likely diagnosis. Since methylmalonic acid and homocysteine, the functional markers that would have confirmed intracellular cobalamin deficiency, could not be measured, the diagnosis rested on the serum vitamin B12 concentration, the maternal vitamin B12 status, and the rapid clinical and biochemical response to cobalamin replacement.

Therapeutic intervention

An intramuscular injection of hydroxycobalamin was started at a dose of 500 mcg every other day for a period of 10 days. After a total of five intramuscular

injections, the patient's movements, interest in the surroundings, and desire to eat increased, and hypotonicity improved, with a significant decrease in the volume and frequency of vomiting. Treatment was continued with 500 mcg sublingual methylcobalamin every other day for two weeks and then 500 mcg sublingual methylcobalamin twice a week for two weeks.

Follow-up and outcomes

At the follow-up visit in the third postnatal month, weight was 6380 g (90-97p), height 59 cm (50-90p), and head circumference 41 cm (90-97p). The complaints of poor feeding and vomiting disappeared completely. Neurological examination showed that he had become more active and mobile compared to when last seen. Age-appropriate head control and visual tracking were present. Control serum vitamin B12 levels were found within the normal range. The plan was for the patient to continue treatment with 500 mcg of sublingual methylcobalamin once a month for six months and then to be re-evaluated. The diagnostic and therapeutic course is summarized in Figure 1.

DISCUSSION

In Istanbul, Türkiye's most populous city, vitamin B12 deficiency in pregnant women and newborns is a common health problem.[2] Mothers are at increased risk of vitamin B12 deficiency due to vegetarian diets or poor access to foods of animal origin, as well as increased requirements during pregnancy and breastfeeding.[3] Early recognition and correction of this common deficiency can prevent progressive and permanent problems in children's growth and cognitive development.[4] The long-term neurological outcome depends on the degree and duration of the deficit, and, as in the case described here, rapid recognition, diagnosis, and treatment are important. It is

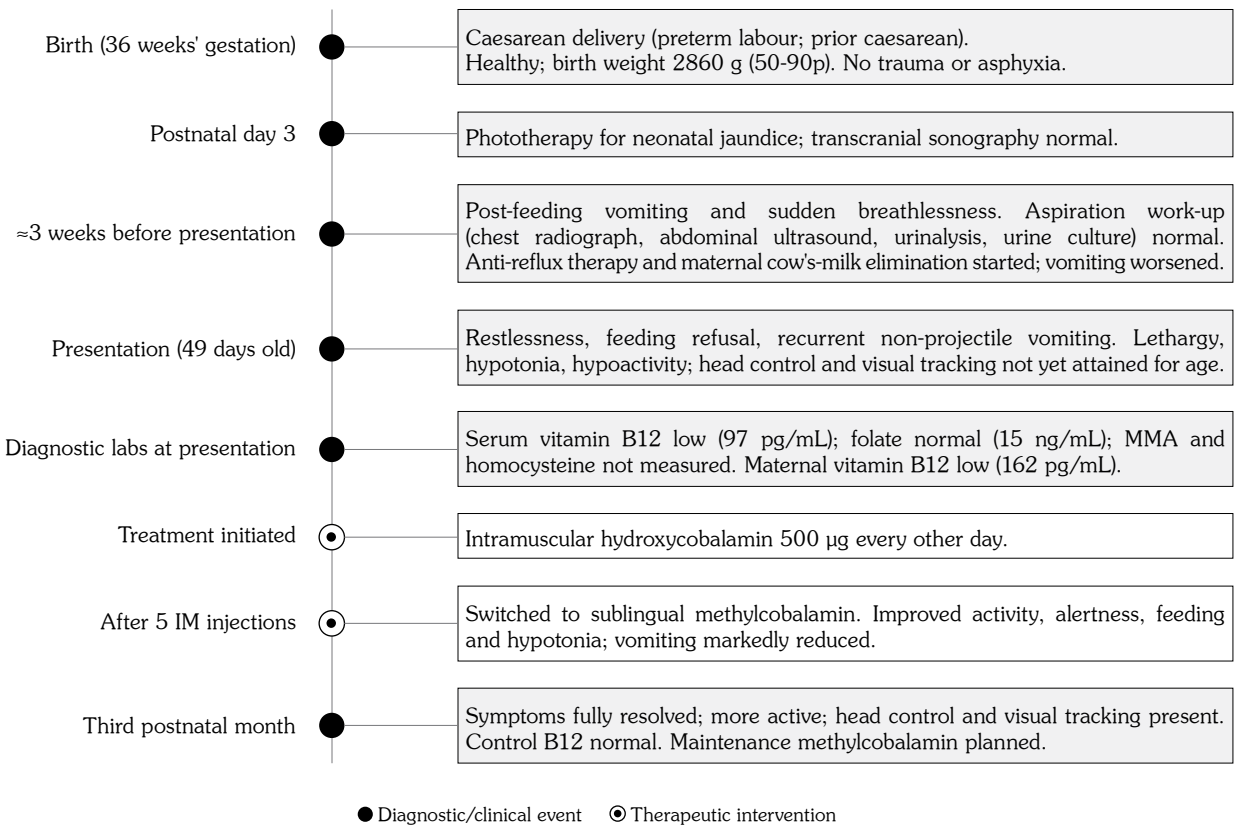

Figure 1. Diagnostic and therapeutic timeline of the case, presented in accordance with the CARE reporting guidelines. Filled circles denote diagnostic/clinical events; open circles denote therapeutic interventions.

B12, vitamin B12; MMA, methylmalonic acid; IM, intramuscular.

thought that the implementation of screening methods for vitamin B12 deficiency, which have been recommended in many studies, will be even more effective in regions where the problem is very common, as in this case.[5]

The signs and symptoms of vitamin B12 deficiency usually appear between two and 12 months after birth.[6] During this period, lethargy, retardation in anthropometric measurements, hypotonia, cessation of developmental skills, and regurgitant vomiting may be observed. These metabolic abnormalities improve very rapidly within a few days of treatment with vitamin B12.[7] Although there is insufficient literature to explain the mechanism of vomiting in vitamin B12 deficiency, it is thought to be due to the neurological effects of deficiency on the enteric nervous system or to methylmalonic acidemia.[8] In the following months, when complementary feeding is started, vomiting in the form of refusal of solid food may be observed in these patients, and developmental delay is often present. It is natural for clinicians to have diagnostic and therapeutic approaches towards more common causes such as overfeeding and infantile reflux in early infants presenting with regurgitated vomiting as in the described case. Persistent clinical symptoms are a warning sign of an underlying metabolic deficiency in this case. In the prenatal history, detailed questioning about pathologies that can cause vitamin B12 deficiency, such as maternal nutrition during pregnancy, pernicious anemia, inflammatory bowel disease, celiac disease, or a history of previous gastrointestinal surgery, will also help in the diagnosis. As in this case, developmental concerns detected on clinical examination, such as hypotonia and the age-appropriate milestones of head control and visual tracking not yet being attained, can serve as a warning sign that the cause of regurgitant vomiting is vitamin B12 deficiency.

In an infant presenting with recurrent vomiting, feeding refusal and hypotonia, a broad differential must be considered before the picture is attributed to cobalamin deficiency. Hypertrophic pyloric stenosis was unlikely given the non-projectile vomiting and a normal abdominal ultrasound; gastroesophageal reflux and overfeeding were initially favored but were excluded by the lack of response to anti-reflux measures; and cow's-milk protein allergy became improbable after a maternal elimination diet failed to relieve the symptoms. Sepsis and urinary tract infection were argued against by normal inflammatory markers and a normal urinalysis and culture, and an endocrine cause was excluded by normal thyroid function. The coexistence of neurological findings with feeding intolerance then directed attention towards a metabolic or nutritional etiology, for which vitamin B12 deficiency, supported by the low infant and maternal serum concentrations, was the most consistent explanation.

Clinical improvement is rapid and beneficial in patients diagnosed with vitamin B12 deficiency in the first few months of life and treated early. Literature shows that oral cyanocobalamin and sublingual methylcobalamin are as effective as parenteral treatment.[9] However, in cases of severe deficiency with neurological sequelae, parenteral vitamin B12 replacement is recommended in the first instance.[10,11] In this case, in which regurgitant vomiting occurred and neurological symptoms became prominent, intramuscular treatment was preferred until clinical improvement began. Treatment was then continued with sublingual methylcobalamin, a less invasive and more comfortable option for the patient.

Data on optimal vitamin B12 dose and schedule in children are limited. The B12 formulation and dosage regimens used also vary widely depending on the etiology of the deficiency and the regions where treatment is administered.[10] In a study conducted in our country, dosing 100 mcg daily for one week, followed by 1000 mcg every other day for one week, then twice a week and then once a week has been shown to be safe and effective in vitamin B12 supplementation in symptomatic children.[12] However, there is also a view that infants with vitamin levels below 150 pg/mL should be treated with a higher loading dose than the conventional treatment dose (100 µg or less).[13] In our patient, the presence of prominent neurological signs guided the choice of parenteral therapy as the first-line option, and the every-other-day schedule is consistent with the alternate-day parenteral regimen generally recommended for cobalamin deficiency with neurological involvement until

clinical improvement is achieved. A dose of 500 µg per injection was considered appropriate for a young infant, lower than the 1000 µg dose used in adults but within the range reported to be safe and effective in symptomatic children. Once clinical improvement became evident, treatment was stepped down to sublingual methylcobalamin, a less invasive route shown to be as effective as parenteral administration. The rapid clinical and biochemical response, without adverse effects, supports the adequacy of the selected regimen. Although the toxicity of vitamin B12 is known to be low, there are case reports showing that it causes transient movement disorders that may be confused with convulsions, especially in infants.[14] To avoid the adverse effects that may be associated with vitamin B12 supplementation in infants, the most appropriate treatment program and dosage need to be determined. Further studies are needed to determine this.

This report is subject to several limitations. First, as a single-case report, its findings cannot be generalized, and no causal inference can be drawn beyond the temporal association between hydroxycobalamin treatment and clinical improvement. Second, methylmalonic acid and homocysteine could not be measured due to technical limitations; these metabolites would have provided functional confirmation of intracellular cobalamin deficiency and strengthened the biochemical diagnosis. Third, developmental status was assessed clinically (age-appropriate head control and visual tracking) rather than with standardized instruments such as the Denver Developmental Screening Test or the Bayley Scales of Infant Development, so the developmental findings should be interpreted with caution. Fourth, the maternal work-up was incomplete: folate, ferritin, mean corpuscular volume, homocysteine, and methylmalonic acid were not obtained, which limits characterization of the underlying mechanism of maternal deficiency. Finally, long-term neurodevelopmental follow-up was not available, so the durability of the observed recovery and the absence of late sequelae could not be confirmed. Prospective studies with standardized developmental assessment and longer follow-up are needed to address these gaps.

In conclusion, in many communities, vitamin B12 deficiency is a public health problem, particularly among pregnant women and infants. In infants, the risk of neurodevelopmental delay increases with the duration of detection and replacement of the deficiency. The hematological and neurological complications of deficiency usually occur later. It is important to increase clinicians' awareness of vitamin B12 deficiency, as deficiency findings may be subtle and non-specific in early infancy. When evaluating infants with symptoms of regurgitation and refusal to feed, vitamin B12 deficiency should be considered along with clinical conditions such as infantile reflux and cow's milk protein allergy. In patients who have had early diagnosis and treatment, clinical improvement will be rapid. Extensive clinical trials are needed to determine the appropriate treatment program with the least potential for side effects, which should be preferred in the infant period.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: C.Ç.: Contributed to the patient's clinical management, study concept and design, data collection, literature review, and drafting of the manuscript; E.Ç.: Contributed to the data collection and literature review; A.V.: Contributed to the study supervision, critical revision, and final revision of the manuscript. All authors read and approved the final manuscript.

Conflict of Interest: The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding: The authors received no financial support for the research and/or authorship of this article.

AI Disclosure: The authors declare that artificial intelligence (AI) tools were not used, or were used solely for language editing, and had no role in data analysis, interpretation, or the formulation of conclusions. All scientific content, data interpretation, and conclusions are the sole responsibility of the authors. The authors further confirm that AI tools were not used to generate, fabricate, or 'hallucinate' references, and that all references have been carefully verified for accuracy.

REFERENCES

1. Akcaboy M, Malbora B, Zorlu P, Altınel E, Oguz MM, Senel S. Vitamin B12 deficiency in infants. Indian J Pediatr 2015;82:619-24. doi: 10.1007/s12098-015-1725-3.

2. Yetim A, Aygün E, Yetim Ç, Ucar A, Karakaş Z, Gökçay G, et al. Measurement of serum vitamin B12-related metabolites in newborns: Implications for new cutoff values to detect B12 deficiency. J Matern Fetal Neonatal Med 2021;34:1260-8. doi: 10.1080/14767058.2019.1633301.
3. Tosi M, Tagi VM, Colombo A, Cecchini A, Zobele M, Montanari C, et al. Dietary pattern and nutritional assessment in a cohort of mothers identified by neonatal screening for cobalamin deficiency in offspring: An Italian single center experience. Front Nutr 2025;12:1604336. doi: 10.3389/fnut.2025.1604336.
4. Banka S, Roberts R, Plews D, Newman WG. Early diagnosis and treatment of cobalamin deficiency of infancy owing to occult maternal pernicious anemia. J Pediatr Hematol Oncol 2010;32:319-22. doi: 10.1097/MPH.0b013e3181d74719.
5. Gramer G, Fang-Hoffmann J, Feyh P, Klinke G, Monostori P, Mütze U, et al. Newborn screening for vitamin B12 deficiency in Germany-strategies, results, and public health implications. J Pediatr 2020;216:165-72.e4. doi: 10.1016/j.jpeds.2019.07.052.
6. Hasbaoui BE, Mebrouk N, Saghir S, Yajouri AE, Abilkassem R, Agadr A. Vitamin B12 deficiency: Case report and review of literature. Pan Afr Med J 2021;38:237. doi: 10.11604/pamj.2021.38.237.20967.
7. Wirthensohn M, Wehrli S, Ljungblad UW, Huemer M. Biochemical, nutritional, and clinical parameters of vitamin B12 deficiency in infants: A systematic review and analysis of 292 cases published between 1962 and 2022. Nutrients 2023;15:4960. doi: 10.3390/nu15234960.
8. Wong S, Ahmad N, Rossetti AL. Vomiting as a presenting symptom of infantile vitamin B12 deficiency. Cureus 2022;14:e25134. doi: 10.7759/cureus.25134.
9. Varkal MA, Karabocuoglu M. Efficiency of the sublingual route in treating B12 deficiency in infants. Int J Vitam Nutr Res 2023;93:226-32. doi: 10.1024/0300-9831/a000724.
10. Elangovan R, Baruteau J. Inherited and acquired vitamin B12 deficiencies: Which administration route to choose for supplementation? Front Pharmacol 2022;13:972468. doi: 10.3389/fphar.2022.972468.
11. Bjørke-Monsen AL. Defining optimal cobalamin status for neonates and infants. Food Nutr Bull 2024;45:S16-22. doi: 10.1177/03795721241227782.
12. Sezer RG, Akoğlu HA, Bozaykut A, Özdemir GN. Comparison of the efficacy of parenteral and oral treatment for nutritional vitamin B12 deficiency in children. Hematology 2018;23:653-7. doi: 10.1080/10245332.2018.1456023.
13. Kılcı C, Olcay L. A common problem in infants: Vitamin B12 deficiency. Turk J Pediatr 2023;65:931-8. doi: 10.24953/turkjped.2022.399.
14. Dilber B, Reis GP. Infantile tremor syndrome secondary to peroral vitamin B12 replacement therapy: A report of two cases with myoclonus. Turk J Pediatr 2021;63:510-5. doi: 10.24953/turkjped.2021.03.020.