International Journal of Contemporary Pediatrics
*Parikh R et al. Int J Contemp Pediatr. 2023 Mar;10(3):376-379*
http://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291

DOI: https://dx.doi.org/10.18203/2349-3291.ijcp20230440

**Case Series**

# Role of nutrition in curing dysphagia: a case series of five rare cases of severe acute malnutrition with severe megaloblastic anaemia

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**Received:** 08 January 2023
**Revised:** 04 February 2023
**Accepted:** 09 February 2023

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## ABSTRACT

Severe acute malnutrition (SAM) is associated with multiple vitamin deficiencies, including vitamin B12. Vitamin B12 deficiency is commonly found in the Indian subcontinent, and has devastating clinical consequences. It leads to megaloblastic anaemia and several other complications. Dysphagia is an unusual symptom of the same. The present study was undertaken to evaluate the reversal of dysphagia with vitamin B12 supplements. A hospital-based retrospective case series was conducted at the B. J. Wadia Hospital for Children. Cases were collected from 19 November 2021, to 25 August 2022. Five children less than two years of age, who presented with complaint of dysphagia, presence of megaloblastic anaemia and concurrent nutritional compromise were evaluated for their clinical profiles, and the outcomes were analysed. We found that dysphagia was a reversible symptom with medical nutrition therapy and injectable B12 therapy in children with SAM who had severe megaloblastic anaemia. The present study has identified the prevalence of dysphagia in cases of SAM with megaloblastic anaemia. This dysphagia is reversible and curable with nutritional rehabilitation and injectable B12 therapy without any further invasive intervention.

**Keywords:** SAM, Megaloblastic anaemia, B12 therapy, Dysphagia, Medical nutrition therapy

## INTRODUCTION

Megaloblastic anaemia (MA) incorporates a heterogeneous group of macrocytic anaemias characterised by the presence of large red blood cell precursors called megaloblasts in the bone marrow.[1] It is the hematologic result of any of a variety of etiological factors causing improper nucleoprotein synthesis.[2] Of these factors, the most common are deficiencies of vitamin B12, folic acid, or both. Vitamin B12 is reported to contribute to more than one-third of anaemia prevalent in India, and it is right next to iron deficiency anaemia among children and adolescents.[3]

Other causes of B12 deficiency in children are ileal resections, inflammatory bowel disease and pernicious anaemia.

In megaloblastic anaemia, there is an asynchrony between the maturation of cytoplasm and that of nuclei, leading to macrocytosis, immature nuclei, and hyper-segmentation in granulocytes in the peripheral blood. The deficiency is critical to recognise and treat as it is a reversible cause of bone marrow failure, demyelinating nervous system disease, and, as suspected in this case series, neurogenic dysphagia. Vitamin B12 is necessary for the development and initial myelination of the central nervous system as well as for the maintenance of its normal function. Demyelination of the cervical and thoracic dorsal and lateral columns of the spinal cord occurs due to vitamin B12 deficiency.

Here we present five cases, younger than 2 years of age, with megaloblastic anaemia, concurrent nutritional compromise, and significant dysphagia. Common

symptoms include-pallor, hyperpigmentation of the skin, coarse, sparse, easily pluckable brown hair, developmental delay or regression, feeding difficulties, hypotonia, lethargy, and low anthropometric measurements for age. The blood indices were suggestive of macrocytic anaemia which was confirmed on peripheral smear by our hematologist and further corroborated by deficient B12 levels. Feeding difficulties among these children resulted in severe acute malnutrition requiring hospitalization. These children underwent parenteral vitamin B12 therapy to ensure better neurological and nutritional outcomes. They were started on F75 formulations and oro-motor stimulation as per the severe acute malnourishment protocol, which resulted in appropriate weight gain, an overall positive outcome, and recovery from dysphagia by the eighth day of treatment.

## CASE SERIES

### *Case 1*

A 1-year-old male child was admitted with complaints of vomiting, cough, cold, and a 7-day fever. On examination, the child had delayed milestones, anthropometric measurements (weight for length less than 3 standard deviation), hyperpigmentation of the skin, sparse brown hair, severe pallor, and the inability to swallow feeds. The child was breastfed since birth and was taking inappropriate complementary feeds after 6 months of age. Investigations were suggestive of vitamin B12 deficiency with urinary tract infection. The child was started on intravenous vitamin B12 injections after two test doses along with third-generation cephalosporins. He was nutritionally rehabilitated by starting F-75 feeds via nasogastric tube as per the SAM protocol at 130 ml/kg/day. Sensory stimulation, in conjunction with oro-motor stimulation, was also simultaneously initiated. On the first two days of intravenous vitamin B12 and sensory stimulation therapy, the child tolerated only tube feeds. Trial with oral feeds failed. On day 4 of intravenous vitamin B12, the child gradually started tolerating liquid feeds orally. Subsequently on day 5, semisolid food was tolerated, and by day 7 of intravenous B12 therapy, the child started taking full diet. There was a weight gain of 14.5 grams per kilogramme per day at the time of discharge.

### *Case 2*

A 1-year-old female child presented with decreased activity since 15 days and cough and cold since 8 days. On assessing the child, we found an edematous face, hyperpigmented skin, easily pluckable sparse hair, and dry scaly skin. She was also apathetic, with her anthropometric measures suggesting severe acute malnutrition with nutritional edema and her developmental age comparable to about 5 months. She was on breast milk from birth, and adjuvant complementary feeds were introduced at the age of 6 months, but she refused feeding as she had swallowing difficulty. After confirmation of a vitamin B12 deficiency on blood tests and peripheral smear, she was started on intravenous vitamin B12 therapy as per protocol. Simultaneously, enteral feed rehabilitation was initiated at 100 ml/kg/day with F-75, sensory stimulation therapy along with oro-motor stimulation was also started. On day 3 of her persistent dysphagia post-hospitalization, the paediatric surgical team was called to intervene and a barium swallow was performed with normal results. On day 4, the child started tolerating liquids orally, and within a week, she started tolerating solid meals. There was a weight gain of 7.5 grams per kilogramme per day after loss of her edema.

### *Case 3*

A 1-year-old female child came with a month-long history of cough, 1-day history of fever, and difficulty in breathing. In view of her increased work of breathing and subcostal intercostal retractions along with nasal flaring, the child was given oxygen support by nasal prongs in the intensive care unit. Nebulizations were given as per protocol. An X-ray of the chest and a gene expert test was done on a gastric lavage to rule out tuberculosis, all of which were normal (Figure 1 for normal chest X-ray). Antibiotics were started in view of her sepsis. Therapeutic milk formulation along with ongoing breast feeding was initiated orally in the intensive care unit. After 48 hours, oxygen was tapered and weaned off, and the child was shifted to the nutritional rehabilitation centre for further management. In view of the classically positive findings of vitamin B12 deficiency on peripheral smear along with severe acute malnutrition, the child was started on vitamin B12 injectable therapy. Oro-motor as well as sensory stimulatory therapy was executed. A complementary feeding trial was started, which was not accepted by the child until day 4 of vitamin B12 therapy. On day 5, there was improvement in feed tolerance, and the child started eating the full complementary diet as advised. The weight gain of the child was 13 grams per kilogramme per day at the time of discharge.

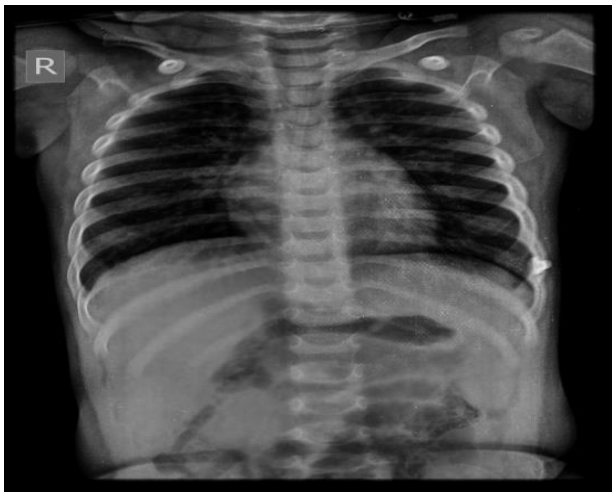


**Figure 1: Normal chest X-ray of 1-year-old female child with suspected tuberculosis.**

***Case 4***

A term baby boy, appropriate for gestational age, presented at 18 months of age with complaints of delayed milestones, loss of appetite, and not gaining weight. The child was investigated and confirmed to have severe megaloblastic anaemia. Intravenous B12 therapy as per protocol was begun along with a starter diet, oro-motor stimulation, and sensory stimulation therapy. On examination, the child was developmentally delayed with stereotypical features of B12 deficiency and had decreased head control (truncal hypotonia). Occupational therapy and speech therapy were initiated concomitantly. A milk-based transition diet was started along with a trial of complementary feeding, but the child had severe dysphagia and intolerance to solid meals. Vitamin B12 treatment was continued. Over a period of 4-5 days, activity of the child improved and reversal of dysphagia was noticed. The child was then shifted to wholesome complementary feeding, which he tolerated well. Weight gain of the child at discharge was 10.7 grams per kilogramme per day.

***Case 5***

A 9-month-old female child was hospitalised due to a 2-day history of fever, cough, cold, and increased work of breathing. On examination, the child had tachypnea, distress, and falling saturation levels; hence, oxygen was administered by nasal prongs. She also had delayed developmental milestones. Blood investigations were suggestive of an acute bacterial infection with severe megaloblastic anaemia which was confirmed on peripheral smear and B12 levels. Chest X-ray showed right upper lobe consolidation. Injectable antibiotics and starter diet were initiated. Vitamin B12 injectable therapy was started after a haematology opinion. Oxygen was weaned off after 48 hours, once the child was stable. For the first 3 days, the child did not tolerate feedings well due to dysphagia. On the fourth day, gavage feeds were simultaneously introduced and tolerated by the child. After 72 hours, oxygen support was weaned off. A significant improvement in milestones was noticed after rehabilitation phase and introduction of complementary feeds. Throughout the course of hospitalization, oro-motor stimulation as well as sensory stimulation therapy were carried out. Weight gain of the child at discharge was 6.4 grams per kilogramme per day.

## DISCUSSION

The most frequent cause of severe vitamin B12 deficiency in India is nutritional in origin; however pernicious anaemia still stands out to be the most severe cause of vitamin B12 deficiency in western countries.[4] The infant of a mother with vitamin B12 deficiency may be born with the deficiency or it may occur if he or she is exclusively breast fed, usually between four and six months of age.[4-6] Typical manifestations of Vitamin B12 deficiency in children include failure of brain development, overall growth and development, developmental regression, hypotonia, feeding difficulties, lethargy, tremors, hyperirritability and coma.[4-6] Brain imaging may reveal atrophy and delayed myelination. Anaemia may be present. Vitamin B12 replacement results in rapid improvement in responsiveness, and many infants recover fully; however, the longer the period of deficiency, the more likely that there will be permanent disabilities.

In children with megaloblastic anaemia, the process of asynchrony affects haematopoiesis as well as rapidly renewing tissues such as gastrointestinal and neural cells. Dysphagia, therefore, may be associated in children with megaloblastic anaemia, further worsening the nutritional outcome. Neurogenic dysphagia is defined as swallowing disorder caused by diseases of the central and peripheral nervous systems, neuromuscular transmission, or muscles.[7] It can result from a disorder in the oral, pharyngeal, or oesophageal phases of swallowing. Its most important sequelae include aspiration pneumonia, malnutrition, and dehydration. Neurogenic dysphagia may arise from involvement of the cortical areas concerned with swallowing, their efferent pathway, the brain stem motor or sensory nuclei, the lower cranial nerves in their distal course, their neuromuscular junctions, or the striated muscle components of the swallow pathway.[8] Vitamin B12 deficiency is associated with demyelination of the cervical and thoracic dorsal and lateral columns of the spinal cord.

Prompt introduction of appropriate nutrition and parenteral B12 therapy, therefore, is a critical step in dealing with severe malnutrition, anaemia, and swallowing difficulties. To begin with, feeds must be quantified and introduced in small amounts as a “starter diet” (75 kcal energy and 0.9 g protein per 100 ml of feed, i.e., low in protein and sodium and high in glucose to prevent hypoglycemia) and gradually upgraded to a substantial amount.[9] After 2–7 days and once the child is hemodynamically and clinically stable, transitioning to a “catch-up diet” (100 kcal and 2.9 g protein per 100 ml) is advisable. In cases with poor weight gain, gavage feeding according to the SAM protocol with F-75 and F-100 should be initiated. The management of megaloblastic anaemia has been revolutionised by the use of intramuscular parenteral B12 therapy. It should be administered daily for one week after two test doses, followed by a weekly therapy for four weeks and then switched to oral daily therapy for three months.

## CONCLUSION

Vitamin B12 replacement therapy results in rapid improvements in dysphagia and delayed development, and many infants recover fully. However, longer deficiency periods are more likely to result in permanent disabilities. Mothers of infants with vitamin B12 deficiency often themselves have unrecognised macrocytic anaemia, but alternatively, they may have a history of a long-term vegetarian or vegan diet as is common in India. Measurement of methylmalonic acid, homocysteine, or

both are used to confirm vitamin B12 deficiency in untreated patients; an elevated level of methylmalonic acid is more sensitive and specific for the diagnosis. For patients with pernicious anaemia or malnutrition, lifelong vitamin B12 therapy is indicated.

Dysphagia is an uncommon manifestation of vitamin B12 deficiency that is potentially reversible if diagnosed and treated within the first six months. The presumptive cause of dysphagia is neurogenic. Few such cases have been reported in adults; however, literature on the same for paediatric age group is scarce. This case series in paediatrics is reported to bring about greater awareness of the need for prompt attention towards early recognition of a compatible clinical condition and the need for collaborative efforts from a spectrum of medical professionals. Instantaneous medical treatment and therapeutic nutrition, oro-motor stimulation, sensory stimulation, play therapy, occupational therapy, and frequent counselling of parents, are hence of utmost necessity. These contribute to a more comprehensive management and better prognosis.

*Funding: No funding sources*
*Conflict of interest: None declared*
*Ethical approval: Not required*

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**Cite this article as:** Parikh R, Mudaliar S, Prabhu S, Bodhanwala M. Role of nutrition in curing dysphagia: a case series of five rare cases of severe acute malnutrition with severe megaloblastic anaemia. Int J Contemp Pediatr 2023;10:376-9.