Case Report

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Megaloblastic Anaemia with Ataxia in a Four-Year-Old Child: A Case Report

Paediatrics Section

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ABSTRACT

Vitamin B12 deficiency is an important yet frequently neglected cause of megaloblastic anaemia in children, most notably among populations with limited access to animal food sources. Although rare, children can still experience neurological effects from vitamin B12 deficiency, including ataxia as well as sub-acute combined degeneration of the spinal cord; however, these are usually reversible if treated quickly. We present a four-year-old male patient with an extremely low vitamin B12 level who presented to our clinic with skin pallor and hyperpigmentation, as well as difficulty in walking. Upon further digging into the patient's medical history and after initial examination of the patient, it was found that he had sensory ataxia and gross tremors on hand examination, a positive Babinski response, and loss of proprioception; complete work up revealed megaloblastic anaemia (with low vitamin B12 level, high homocysteine and methylmalonic acid), as well as MRI of the brain confirming subacute combined degeneration of the spinal cord. After receiving Intramuscular (IM) vitamin B12 injections, the patient demonstrated excellent improvement in both clinical and haematological parameters, disease remission at follow-up (3 months) and complete resolution of neurological symptoms. Our findings point towards the need to consider vitamin B12 deficiency when evaluating a child with unexplained anaemia and neurological symptoms, especially in those with high risk for developing vitamin B12 deficiency due to dietary considerations.

Keywords: Cobalamin deficiency, Hyperpigmentation, Paediatric nutrition, Sensory ataxia, Subacute combined degeneration

CASE REPORT

A four-year-old male child presented to paediatric outpatient department with progressive weakness, poor growth, and difficulty in walking for six months.

The child developed gradual onset of weakness and lethargy over the past six months. Parents noticed progressive loss of appetite, poor weight gain, and increasing difficulty in maintaining balance whilst walking, manifesting as swaying movements. The child also developed darkening of the skin over palms, soles, knuckles, and fingertips during this period. There was no history of fever, seizures, altered consciousness, or trauma.

The child had normal developmental milestones until 18 months of age. Immunisation was complete as per the National Immunisation Schedule up to 18 months of age. No significant past illnesses were reported.

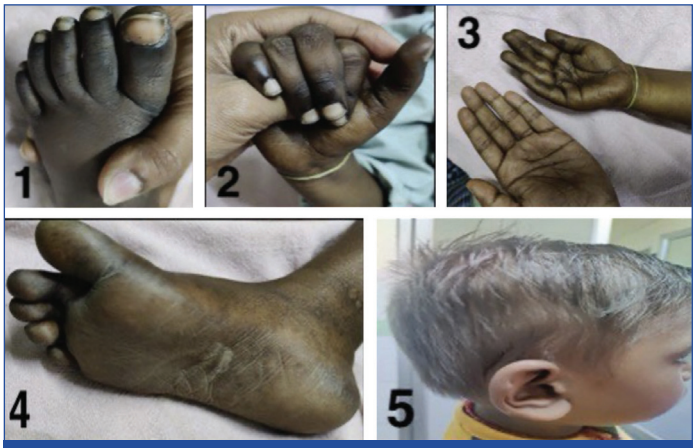
The child had been on a strict vegetarian diet since birth with no consumption of any animal-origin foods including meat, fish, eggs, or dairy products. Intake of fresh fruits and vegetables was occasional and irregular.

The child was born to non-consanguineous parents. No family history of anaemia, neurological disorders, or metabolic conditions was reported.

Clinical Examination

General examination: The child appeared pale and malnourished. Anthropometric measurements revealed Severe Acute Malnutrition (SAM) with weight and height below the third percentile for age. Marked hyperpigmentation was noted over the palms, soles, knuckles, and terminal phalanges [Table/Fig-1-4]. The child had pallor of the conjunctivae, conjunctival xerosis, glossitis and hypopigmented, fragile hair [Table/Fig-5].

Cardiologic, respiratory and per abdominal examination were normal. [Table/Fig-6] shows laboratory findings suggestive of severe vitamin B12 deficiency with megaloblastic anaemia and pancytopenia.



[Table/Fig-1-5]: (1-3) Shows hyperpigmentation of dorsum of legs, knuckles and palmsl (4) Shows hyperpigmentation of soles; (5)Shows hypopigmented fragile hair.

Investigation	Results	Normal range
Haemoglobin (g/dL)	6.2	11-14
Mean corpuscular volume (fL)	118	70-86
Total leucocyte count (/µL)	3,200	6,000-17,000
Platelet count (/µL)	82,000	150,000-450,000
Serum vitamin B12 (pg/mL)	98	200-900
Serum folate (ng/mL)	8.2	3-17
Serum homocysteine (µmol/L)	52	5-15
Methylmalonic acid (µmol/L)	3.8	<0.4
Serum LDH (U/L)	1,850	125-220

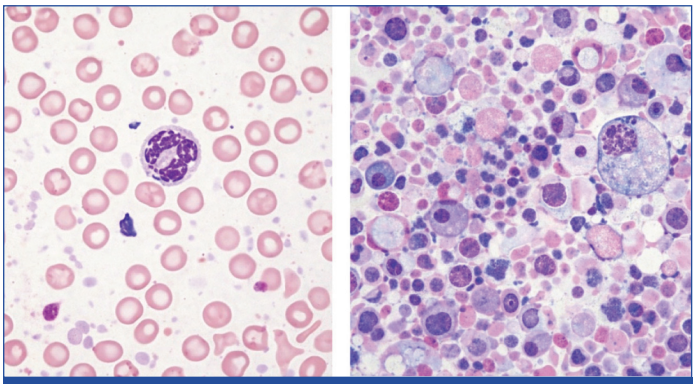
[Table/Fig-6]: Findings suggestive of severe vitamin B12 deficiency with megaloblastic anaemia and pancytopenia.

Neurological examination: The child demonstrated sensory ataxia with a wide-based gait and swaying whilst walking. Coarse tremors were present in the upper limbs. Muscle tone was normal. Power

was 4/5 in all four limbs. Deep tendon reflexes were diminished. Plantar response showed bilateral extensor (Babinski positive). Sensory examination revealed loss of proprioception and vibration sense in the lower limbs. Higher mental functions and cranial nerve examination were normal.

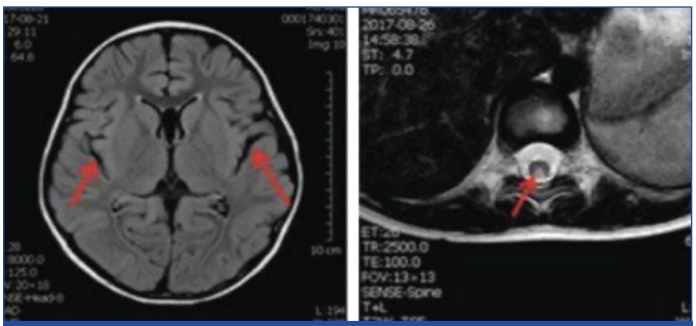
Peripheral blood smear: [Table/Fig-7] Macrocytic red blood cells with hypersegmented neutrophils (>5 lobes), leukopenia, and thrombocytopenia.

Bone marrow examination: [Table/Fig-8] Hypercellular marrow with megaloblastic changes. Howell-Jolly bodies, band cells, and metamyelocytes were observed. Megakaryocytes were normal in number and morphology.



[Table/Fig-7]: Peripheral blood smear showing macrocytic red blood cells with anisocytosis and presence of hypersegmented neutrophils (>5 lobes). The smear also demonstrates reduced white blood cells and platelets, consistent with leukopenia and thrombocytopenia in megaloblastic anaemia.
[Table/Fig-8]: Bone marrow aspirate smear demonstrating hypercellular marrow with megaloblastic erythroid precursors characterised by nuclear–cytoplasmic asynchrony. Howell-Jolly bodies are noted within erythrocytes, along with band forms and metamyelocytes. Megakaryocytes are preserved in number and morphology. (Images from left to right)

Magnetic Resonance Imaging (MRI) of Spine: T2-weighted images demonstrated hyperintense signal changes in the dorsal columns of the cervical and thoracic spinal cord, characteristic of subacute combined degeneration. The classical 'inverted V sign' was noted on axial sections [Table/Fig-9,10].



[Table/Fig-9]: MRI demonstrating central nervous system involvement. Axial brain MRI (FLAIR sequence) showing bilateral, symmetrical hyperintense signals in the deep gray matter regions (indicated by red arrows), suggestive of metabolic or nutritional aetiology.
[Table/Fig-10]: Axial spinal MRI showing abnormal intramedullary signal intensity in the posterior aspect of the spinal cord (red arrow), consistent with dorsal column involvement. (Images from left to right)
Note: Parental consent was obtained for clinical photography and publication of this case report.

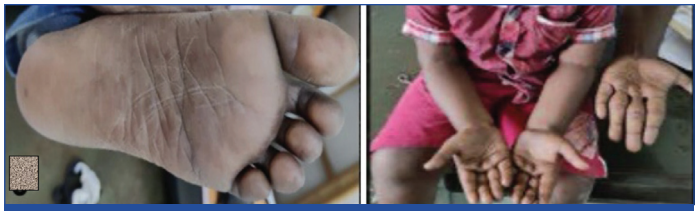
Differential Diagnosis

Based on the combination of pallor, hyperpigmentation, and ataxia, several differentials were considered for this condition: iron deficiency anaemia with neurologic pathology, vitamin B12 deficiency, copper deficiency, chronic lead poisoning, posterior fossa tumours, and inherited metabolic disorders; this is due to the finding of macrocytic anaemia, hypersegmented neutrophils, and pancytopenia with subacute combined degeneration of the spinal cord consistent with MRI findings, along with low serum B12 levels and elevated homocysteine and methylmalonic acids confirming vitamin B12 deficiency.

Final diagnosis: Severe vitamin B12 deficiency with megaloblastic anaemia, pancytopenia, and subacute combined degeneration of the spinal cord secondary to dietary insufficiency.

Treatment and follow-up: The initial therapy was using vitamin B12 injection (cyanocobalamin) at a dose of 1000 µg administered Daily Intramuscular (IM) injection for the first 7 days, followed by one injection weekly for four weeks, then subsequently once a month for six months. After two weeks of receiving vitamin B12, the patient was prescribed oral iron supplementation (6 mg/kg/day) and oral folic acid (5 mg/day) to treat the patient's anaemia and to prevent any further deficiencies during the recovery of his haematological parameters. The parents received detailed instructions about including animal-source foods (such as dairy, and eggs) as well as fortified with vitamin B12 supplements into child's diet.

As a result of the above therapies, the patient showed an marked clinical improvement. Child demonstrated increased appetite and overall well-being within several days after initiation of therapy. Neurologically, there was significant improvement after two weeks of therapy, with resolution of gait ataxia, normalisation of the plantar reflex from extensor to flexor; considerable improvement in hyperpigmentation [Table/Fig-11,12]; and resolution of tremors.



[Table/Fig-11]: Post-treatment clinical photograph showing improvement in palmar hyperpigmentation following intramuscular vitamin B12 therapy.
[Table/Fig-12]: Post-treatment image demonstrating resolution of hyperpigmentation of the palms and improved overall skin tone after vitamin B12 supplementation at follow-up. (Images from left to right)

At his three-month follow-up, the patient has gained weight from 10.5 kg at the time of initial visit to 13.7 kg (gain 3.2 kg). Laboratory testing continued to confirm complete recovery of all haematology parameters, including normal haemoglobin levels (12.8 g/dL), white blood cell count and platelet count which were all within normal limits. Neurologically, the ataxia no longer existed and he was able to ambulate normally and have full proprioceptive function and normal sensation of vibration as well as normal deep tendon reflexes. The patient continued monthly injections of vitamin B12 and continued to adhere to a diet with animal-source protein.

DISCUSSION

Vitamin B12 (cobalamin) is a vital water soluble vitamin that is involved in DNA synthesis, haematopoiesis, central nervous system myelination and neurotransmitter metabolism [1]. All dietary sources of vitamin B12 are of animal origin (e.g., meat, fish, eggs, and dairy products). Deficiency of this essential nutrient can result from a variety of causes, including inadequate dietary intake, malabsorption, genetic disorders affecting vitamin B12 metabolism, and increased requirements during periods of rapid growth [2].

The clinical signs resulting from vitamin B12 deficiency are multifactorial and can impact multiple organ systems. The haematological manifestations of B12 deficiency typically include megaloblastic anaemia, which is characterised by macrocytic red cells (mean corpuscular volume greater than 100 fL), hypersegmented neutrophils (i.e., neutrophils with six or more lobed nuclei), leukopenia, and thrombocytopenia (resulting in pancytopenia) [3]. The pathophysiology of megaloblastic anaemia due to vitamin B12 deficiency involves impaired DNA synthesis caused by ineffective production of thymidine, resulting in ineffective erythropoiesis and destruction of haematopoietic cells in the bone marrow. This explains the very elevated lactate dehydrogenase levels that are frequently seen in these patients [4].

Cutaneous changes due to vitamin B12 deficiency are less frequently reported in the literature than the haematological manifestations, but can have clinical implications. Hyperpigmentation occurs on the palms, soles, and in flexural areas due to increased production of melanin secondary to defective methylation reactions [5]. The glossitis seen in our patient represents atrophy of the lingual papillae and is consistent with megaloblastic anaemia. Importantly, skin abnormalities secondary to vitamin B12 deficiency often occur prior to or concurrently with blood abnormalities. This should alert clinicians to assess for vitamin B12 deficiency in an individual with skin changes [6].

The neurological complications of vitamin B12 deficiency can have serious consequences for children since these complications can lead to lasting effects on development if they are not identified and treated in a timely manner. Pathophysiology consists of demyelination of the posterior and lateral columns of the spinal cord, peripheral nerves and sometimes the optic nerve and cerebral white matter [7]. This condition is termed subacute combined degeneration and manifests clinically as a combination of sensory ataxia, spastic paraparesis, loss of proprioception and vibration sense, and peripheral neuropathy. The use of the word 'subacute' denotes that the onset of symptoms is typically gradual over a period of weeks to months, although the rate of progression can vary considerably from one person to another [8].

The mechanism responsible for neurological damage in vitamin B12 deficiency relates to its role as a cofactor for two key enzymes: methionine synthase and methylmalonyl-CoA mutase. When vitamin B12 is deficient, there is an increase in the levels of two compounds, homocysteine and methymalonate, both of which are necessary for the proper synthesis of myelin, as well as for proper incorporation of fatty acids into neuronal cell membranes. Accumulation of both homocysteine and methymalonic acid causes a progressive demyelination, particularly involving the dorsal and lateral columns of the spinal cord, where the largest number of myelinated nerve fibres are found [9]. The presence of bilateral symmetric changes in the dorsal column is represented on MRI with the inverted "V" sign [10].

Those first neurological signs of B12 deficiency are loss of proprioception and vibratory sensation, which is the result of damage to the posterior column; as neurologic demyelination progresses, damage to the lateral column produces evidence of upper motor neuron damage (e.g., spasticity, hyperreflexia and extensor plantar responses), whereas injury to peripheral nerves produces diminished or absent reflexes and distal paraesthesia [11]. The presence of both upper and lower motor signs is consistent with a finding that together represent a mixed picture of either the presence or absence of B12 deficiency in the differential diagnosis. Subacute combined degeneration is well recognised in the adult population, but is very uncommon in the paediatric population due to shorter duration of deficiency and superior compensatory mechanisms in the developing nervous system [12]. However, cases can occur, particularly in children who have been exclusively breastfed for an extended period of time by B12 deficient mothers, are on strict vegetarian diets, or have malabsorption syndromes. In a case similar to ours, Keskin EY and Keskin M (2015) reported a 15-year-old male with severe vitamin B12 deficiency presenting with pancytopenia and haemolysis, demonstrating how variable presentation can be among children [13].

The most common reason for global vitamin B12 deficiency among children is through nutritional means, specifically through a lack of dietary intake of animal source foods [14]. This occurs with children from family backgrounds that adhere to a strict vegetarian or vegan diet, in geographic areas with limited access to animal sources of food, and among children of mothers that are themselves B12 deficient at the time of pregnancy or lactation. In our case, the complete lack of any consumption of food from animals since birth, along with your child's poor nutritional intake of fruits and vegetables, provides the ideal conditions for developing severe vitamin B12 deficiency. Other potential causes of vitamin B12 deficiency in children include: pernicious anaemia (which is uncommon in paediatrics), gastric or ileal disease affecting the production of intrinsic factor and/or the absorption of B12, parasitic infection (in particular Diphyllobothrium latum), extended use of either proton pump inhibitors or metformin, and inborn errors of B12 metabolism such as deficiency of transcobalamin II [15].

Once there is clinical suspicion of a deficiency of vitamin B12, laboratory confirmation will provide diagnosis. A serum B12 level of less than 200 pmol/L is considered diagnostic, while B12 levels between 200 and 300 pmol/L may reflect subclinical vitamin B12 deficiency [16]. B12 deficiency, resulting from impaired enzyme processes, causes high levels of methylmalonic acid and homocysteine in the serum, thus making diagnosis easier. Methylmalonic acid is exclusively related to a B12 deficiency, while both methylmalonic acid and homocysteine will be high for a B12 and folic acid deficiency respectively [17]. A peripheral blood smear will usually show macrocytosis, hypersegmented neutrophils, and (if severe), pancytopenia. A bone marrow biopsy will typically show megaloblastic changes with nuclear-to-cytoplasmic asynchrony, however this is not always necessary [18].

The MRI is extremely useful in identifying neurological consequences. T2-weighted/FLAIR sequences will show bilateral symmetric hyperintense signal changes present bilaterally in the posterior columns of the cervical and thoracic spinal cord. The 'inverted V sign', seen on axial imaging, indicates involvement of the dorsal columns and is pathognomonic for subacute combined degeneration. MRI changes may be present prior to the onset of symptomatology and may remain even after biochemical correction but usually improve with treatment [19].

The treatment of a B12 deficiency must occur parenterally (by injection) with cyanocobalamin, particularly if neurological symptoms are involved or the deficiency is severe. The use of parenteral therapy is justified in that it will provide rapid replenishment of body stores as well as precluding the possibility of an absorption defect [20]. Daily intramuscular injections of 1,000 µg are used to saturate tissue stores in the first week or two of therapy. After that, weekly injections over a period of one month are given to ensure that there are adequate levels in preparation for maximal haematopoietic recovery. After maximal haematopoietic recovery has been completed, a monthly maintenance regimen will continue [21]. For patients with a dietary deficiency who cannot absorb B12 normally, oral high-dose B12 (1,000-2,000 µg a day) may be considered for long-term therapy after initial repletion has been accomplished. However, attention must be paid to patient compliance, as this is critical to achieving treatment success.

In addition to supplementation of iron and folic acid (i.e., 100 µg per day) in the second week of B12 treatment to help meet the increased demands of rapid erythropoiesis, these two nutrients will prevent the development of functional deficiencies that may impede the recovery process of the haematological system as a result of these increased requirements [22]. Therefore, folate must not be given alone in the presence of suspected B12 deficiency, as although this may correct the haematological abnormalities, it will allow the neurological deterioration associated with this condition to continue to progress unopposed, a phenomenon known as subacute combined degeneration of the cord [23].

Typically, patients will experience dramatic responses to B12 therapy, which occur mainly in phases. For example, patients will frequently report subjective improvements in well-being and appetite within days after beginning treatment, even before any significant or measurable improvements have occurred in their haematological parameters [24]. The reticulocyte count generally peaks between 5-7 days after treatment is initiated. Therefore, many patients will experience an immediate and brisk erythropoietic response. After

7-10 days, patients' haemoglobin levels will begin to rise and will typically be normalised within 4-8 weeks following inception of therapy. Most patients will have normalised their leucocyte and platelet counts (i.e., leucopenia and thrombocytopenia) after 1-2 weeks of treatment. Neurological recovery after a deficiency can have a non-linear timeline; however, improvement can occur within weeks in contrast to the fact that full resolution may not occur for 3-12 months, depending on how severe and how long the deficiency was [25]. In some situations, particularly when having a prolonged severe deficiency, there can be irreversible neurological deficits even with adequate treatment has occurred, demonstrating the necessity for early detection of deficiency and early intervention if appropriate.

Managing patients with anaemia long-term requires fixing the underlying source of anaemia. For dietary deficiencies, education on comprehensive nutrition and dietary modifications would be the most important things to address [26]. Families should receive counselling on the numerous sources of dietary B12 that have been shown to be reliable sources. Dairy products, eggs, fortified cereal products, or alternative plant-based milk (if the patient is not vegetarian/vegan), or if they are, B12 should be supplemented with oral supplements on a permanent basis, or periodically given through parenteral routes. Parenteral therapy also would occur in all patients diagnosed with malabsorptive disorders or pernicious anaemia [27].

The presence of cases presenting similarly has been documented in recent literature in children diagnosed with vitamin B12 deficiency. For example, a two-year-old child was discovered to have a lack of peripheral blood because of a vitamin B12 deficiency. This highlights the variability of age in patients who present with a lack of peripheral blood and lack of normal cellular characteristics due to vitamin B12 deficiency [28]. Additionally, neurological manifestations occurred in approximately 30% of children with anaemia due to vitamin B12 deficiency per a review of a series conducted at one facility by Oltean A et al., [29]. Additionally, Philip R et al., documented an infant with megaloblastic anaemia and a lack of peripheral blood; although this type of anaemia is not common, it should still be included in the differential diagnosis when considering suspected unexplained anaemia in a young child [30].

We will reiterate the following clinical lessons learned from our case. First, any child with unexplained macrocytic anaemia should always be evaluated for the presence of vitamin B12 deficiency, especially if the child participates in restrictive diets or has a malabsorption disorder. Second, neurological symptoms must be evaluated and treated emergently because waiting can lead to irreversible neurological damage. Third, when conducting an evaluation for anaemia in children, it is essential to obtain a detailed dietary history because nutritional deficiencies are still a preventable source of morbidity. Finally, we have documented the effectiveness of administering appropriate treatment in a timely manner to reverse the severity of vitamin B12 deficiency.

CONCLUSION(S)

This case illustrates that severe vitamin B12 deficiency in children may present with megaloblastic anaemia, pancytopenia, skin hyperpigmentation, and subacute combined degeneration of the spinal cord. Careful clinical assessment, particularly a detailed dietary history, together with appropriate laboratory and radiological investigations, plays a pivotal role in establishing an early diagnosis. Timely initiation of parenteral vitamin B12 therapy can lead to complete haematological and neurological recovery. Early identification is vital to prevent potentially irreversible neurological sequelae, especially in children adhering to strict vegetarian diets. However, clinicians should be aware that haematological improvement following vitamin B12 therapy does not invariably exclude an underlying inborn error of metabolism or immunodefiency. Persistent lymphopenia, recurrent infections or incomplete immune recovery should prompt further immunological evaluation.

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