A case report on Subacute Combined Degeneration of spinal cord in children – A rare neurological manifestation in Vitamin B12 deficiencies

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Received: 2017-11-24; Revised: 2017-11-27; Accepted: 2017-11-29
Available online: 1st December 2017

Abstract: Megaloblastic anemia is a common feature between 6 months – 2 years and rarely occurs after 5 years of age, especially in a child consuming non-vegetarian diet. B12 deficiency may occur after 5 years of age because of chronic diarrhea, malabsorption syndrome, or intestinal surgical causes. Pernicious anemia causes B12 deficiency, but nutritional B12 deficiency with subacute combined degeneration causing ataxia is rare.

Keywords: vitamin B12, degeneration, spinal cord

Introduction
This case highlights Subacute combined degeneration of spinal cord, refers to degeneration of the posterior and lateral columns of the spinal cord as a result of vitamin B12 deficiency.

Case Summary
An 8-year-old male child born of consanguineous marriage, strict vegetarian, complains of high grade fever dry cough and very poor appetite for 1 month. Repeated bouts of intense abdominal pain, colicky in nature repeated episodes of nausea and vomiting. BMI (<3rd percentile) and unable to walk in a straight line.

Laboratory Findings:
CBC showed:
- Hb–8.8 g/dl,
- hematocrit (Ht)–27.5,
- WBC– 4000/cumm,
- MCV–115.1 fl,
- MCH-36.8 pg,
- MCHC–32.0%
- RDW–56.8%.
PBS showed macrocytic hypochromic.
Vitamin B12 levels – 148pg/.

MRI findings
MRI spine findings with T2 W sagittal image revealed intra-parenchymal abnormal signal intensity in the spinal cord predominantly in the posterior columns at lower levels, lesions are present dorsally.

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DOI: http://dx.doi.org/10.21746/ijbio.2017.6.12.2
Discussion

Vitamin B12 deficiency is uncommon between 6 months – 2 years and rarely occurs after 5 years of age. Causes of this deficiency could be because of chronic diarrhea, malabsorption syndrome, or intestinal surgical causes. Nutritional B12 deficiency presenting with megaloblastic anemia can lead to neurological symptoms such as subacute combined degeneration(SCD) causing ataxia. The pathological findings in SCD consist of patchy losses of myelin in the dorsal and lateral columns. Patients present with weakness of legs, arms, trunk, tingling and numbness that progressively worsens. Vision and mental state may also be altered. Bilateral spastic paresis may develop and pressure, vibration and touch sensations are diminished. This case has megaloblastic anemia presenting with SCD which is rare. Early response to treatment is satisfying, the long-term consequences of nutritional vitamin B12 deficiency in children emphasize the need for prevention or early recognition of this syndrome. Recommended daily requirements range from 0.4 mcg (infants) to 2.4 mcg (>14 years age).

Conclusion

SCD in vitamin B12 Deficient children is a rare finding but can be reversible with treatment.

References


Cite this article as:


DOI: http://dx.doi.org/10.21746/ijbio.2017.6.12.2

Source of support: Nil.

Conflict of interest: None Declared