Case Report

Dietary Deficiency of Cobalamin Presented Solely as Schizoaffective Disorder in a Lacto-Vegetarian Adolescent

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ABSTRACT

Cobalamin is an important nutrient. It is not synthesized in human body and supplied only in nonvegetarian diet. Its deficiency reported with range of psychiatric disorders. Only four pediatric cases have been reported as psychiatric disorders. Authors report a case of dietary deficiency of cobalamin presenting solely as schizoaffective disorder without hematological/neurological manifestations. Early diagnosis and treatment of cobalamin deficiency is an opportunity to reverse pathophysiology. This case highlights the importance of diet history and serum cobalamin level in atypical psychiatric presentations.

Key words: *Children, dietary deficiency, psychiatry, vitamin B12*

INTRODUCTION

Cobalamin (Vitamin B12) is an important nutrient for nervous and hematological system. It is not synthesized in human body and supplied exclusively in non-vegetarian diet.[1] Some Indians are strict vegetarians/lacto-vegetarians leading to cobalamin deficiency.[2] Dietary deficiency of cobalamin is one of various reasons for cobalamin deficiency.

Neuropsychiatric disorders precede hematologic signs and often the presenting manifestation of B_{12} deficiency.[3] Psychiatric disturbances include apathy, depression, dementia, delirium, delusions, hallucinations, irritability, incoherent speech, and catatonia are commonly reported in adults/elderly.[4-6] Till date, the psychiatric presentations of cobalamin deficiency are reported in four children. First as psychotic mood disorder with neurological manifestations due to gastric helicobacter pylori colonization.[7] Second, oral vitamin-B12 monotherapy showed dramatic improvement in psychosis, but relapsed a year later after stopping; however, author did not mention serum vit-B12 level or other manifestations.[8] Third, inadequate dietary intake of vitamin B12 and folate presented as neuropsychiatric changes and megaloblastic anaemia.[9] Last, a case of psychotic disorder and thrombotic manifestations of vitamin B12 and folate deficiency.[10] However, there is no report of vegetarian pediatric case presenting solely as psychosis due to dietary deficiency. Authors report a first case of dietary deficiency of cobalamin in lacto-vegetarian adolescent presenting solely as schizoaffective disorder without hematological/neurological manifestations.

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CASE REPORT

A 13-year-old lacto-vegetarian class VIII boy was presented from lower socioeconomic status with nil significant family and personal history. No past history of gastrointestinal pathology, surgeries, or use of alcohol or proton-pump blockers. Patient admitted with 1 week history of mutism, rigidity, immobility, staring look, disturbed sleep, ideas of worthlessness and hopelessness, aimless wandering, guilt, and suicidal ideas. Physical examination (PE) found no abnormality. On mental status examination (MSE), patient had partial mutism, manneristic posture, depressed mood, paranoid delusions, thought echo/broadcast, running commentary, and command hallucinations. Patient was diagnosed as acute schizophrenia-like psychotic disorder (ICD-10:F23.2). Complete blood count with peripheral blood smear, blood sugar, renal/liver function test, serum iron, total iron binding capacity, urinary iron binding capacity, transferring, and transferrin saturation were normal. He responded quickly with lorazepam-6 mg/day (stopped third day), olanzapine-15 mg/day and sertraline-50 mg/day, and discharged in 5 days. Subsequently patient was asymptomatic, but had functional disability for which aripiprazole 15 mg/day was added and followed up for the next 2 months.

Then patient relapsed despite good adherence with 1 week history of suspiciousness, hearing voices, over-talkativeness, over-cheerfulness, inflated self-esteem, decreased need for sleep, increased appetite, increased pleasurable activities, and disruptive socio-education. PE found no abnormality. MSE revealed inflated self-esteem, over-familiarity, spontaneous over-productive speech, increased psycho-motor activity, elated affect, flight of ideas, thought echo and broadcasting, delusions of persecution and reference, command and commentary second person hallucinations, impaired judgment, and absent insight. Diagnosis revised to schizoaffective disorder, manic type (ICD-10:F25.0). Sertraline stopped and divalproate sodium-500 mg/day added along with olanzapine-15 mg/day and aripiprazole-15 mg/day. Patient came with worsening of symptoms after 4 days. Divalproate increased to 1 gm and lithium carbonate 600 mg and haloperidol-10 mg were added.

Two days later, patient was re-admitted for worsening of symptoms for evaluation of organicity. Repeat routine investigations normal. No abnormality in magnetic resonance imaging of brain, thyroid function test, and gastric mucosal biopsy by upper gastrointestinal endoscopy was reported. Serum cobalamin level was 112 ng/mL (normal range: 180 to 914 ng/mL) while serum folate was normal. Serum intrinsic factor and parietal cell antibodies were negative. Diagnosis revised to schizoaffective disorder secondary to Cobalamin deficiency. Patient started on alternate days 1 ml intramuscular injection vitcofol (Vitamin B12 = 500 mcg, folate = 15 mg andniacinamide = 200 mg per ml) for six doses, then 2 ml monthly. Patient became asymptomatic soon after second dose of cobalamin and discharged in 1 week. During followups, normal serum cobalamin level was maintained and psychiatric medications tapered and stopped on the 52nd day of vitcofol. Premorbid level was maintained on regular followups for next 6 months.

DISCUSSION

Atypical presentation of case consists of acute onset, fluctuating course of psychotic spectrum initially with catatonia, depressive symptoms, first rank symptoms, (FRS), and later as mania, FRS, and relapsed soon on medications. These lead for the search of organicity. Dietary deficiency of Cobalamin was found. Psychiatric medication was quickly stopped. Patient reached quickly and maintained the premorbid level in the short-term follow-up with only cobalamin supplementation.

Dietary deficiency of cobalamin associated with psychosis is significant in developing country like India for two reasons. First, the high prevalence of dietary deficiency of Cobalaminin Indian children,[11] and second, course and prognosis with supplementation is better. This emphasizes the importance of diet history and performing serum cobalamin level as a routine investigation in first episode, atypical presentation, and treatment resistant cases, especially in vegetarian.

Early diagnosis and treatment of cobalamin deficiency in neuropsychiatry is recommended for following reasons: first, better prognosis is expected if treated in window period[12] to avoid irreversible neuronal injury.[13] Second, to avoid treatment resistance[14] and last, finding that high vitamin B12 level is associated with better treatment outcomes.[15]

Authors put forth following hypothesis for psychiatric manifestations. Methylene-tetrahydrofolate reductase, an enzyme of cobalamin metabolism, is involved in metabolism of tetrahydrobiopterin and latter is required for synthesis of dopamine and serotonin.[16]

Short-term followup is a limitation, but, rapid disappearance of florid psychopathology and maintaining pre-morbid level solely on cobalamin supplementation is vital.

CONCLUSION

Dietary deficiency of cobalamin is an important reversible cause of psychosis. Early diagnosis and treatment of cobalamin deficiency during

narrow window period is an opportunity to revert pathophysiology. This highlights the importance of diet history and serum cobalamin level in atypical psychiatric presentations.

REFERENCES

1. Babior BM, Bunn HF. Megaloblasticanemias. In: Kasper DL, Braunwald E, Hauser SL, Fauci AS, Longo DL, Jameson JL, editors. Harrison's Principles of Internal Medicine. New York: McGraw-Hill Companies Inc; 2005. p. 601-6.
2. Mehta BM, Rege DV, Satoskar RS. Serum vitamin B12 and folic acid activity in lactovegetarian and nonvegetarian healthy adult Indians. Am J Clin Nutr 1964;15:77-84.
3. Lindenbaum J, Healton EB, Savage DG, Brust JC, Garrett TJ, Podell ER, *et al*. Neuropsychiatric disorders caused by cobalamin deficiency in the absence of anemia or macrocytosis. N Engl J Med 1988;318:1720-8.
4. Hutto BR. Folate and cobalamin in psychiatric illness. ComprPsychiatry 1997;38:305-14.
5. Masalha R, Chudakov B, Muhamad M, Rudoy I, Volkov I, Wirguin I. Cobalamin-responsive psychosis as the sole manifestation of vitamin B12 deficiency. Isr Med Assoc J 2001;3:701-3.
6. Catalano G, Catalano MC, Rosenberg EI, Embi PJ, Embi CS. Catatonia. Another neuropsychiatric presentation of vitamin B12 deficiency? Psychosomatics 1998;39:456-60.
7. Tufan AE, Bilici R, Usta G, Erdoğan A. Mood disorder with mixed, psychotic features due to vitamin B12 deficiency in an adolescent: Case report. Child Adolesc Psychiatry Ment Health 2012;6:25.
8. Denson R. Letter: Vitamin B12 in late-onset psychosis of childhood. Can Med Assoc J 1976;114:113.
9. Middleman AB, Emans SJ, Cox J. Nutritional vitamin B12 deficiency and folate deficiency in an adolescent patient presenting with anemia, weight loss, and poor school performance. J Adolesc Health 1996;19:76-9.
10. Dogan M, Ozdemir O, Sal EA, Dogan SZ, Ozdemir P, Cesur Y, *et al*. Psychotic disorder and extrapyramidal symptoms associated with vitamin B12 and folate deficiency. J Trop Pediatr 2009;55:205-7.
11. Taneja S, Bhandari N, Strand TA, Sommerfelt H, Refsum H, Ueland PM, *et al*. Cobalamin and folate status in infants and young children in a low-to-middle income community in India. Am J Clin Nutr 2007;86:1302-9.
12. Martin DC, Francis J, Protetch J, Huff FJ. Time dependency of cognitive recovery with cobalamin replacement: Report of a pilot study. J Am Geriatr Soc 1992;40:168-72.
13. Stenier I, Kidron D, Soffer D, Wirguin I, Abramsky O. Sensory peripheral neuropathy of vitamin B12 deficiency: A primarily demyelinating disease? J Neurol 1988;235:163-4.
14. Kate N, Grover S, Agarwal M. Does B12 deficiency lead to lack of treatment response to conventional antidepressants? Psychiatry (Edgmont) 2010;7:42-4.
15. Hintikka J, Tolmunen T, Tanskanen A, Viinamäki H. High vitamin B12 level and good treatment outcome may be associated in major depressive disorder. BMC Psychiatry 2003;3:17.
16. Bolander-Gouaille C, Bottiglieri T. Homocysteine: Related Vitamins and Neuropsychiatric Disorders. Paris: Springer; 2007.

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