



Case Report

# Psychosis associated with Vitamin B12 deficiency - A case report with review of literature

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## Abstract

Vitamin B12 is one of the essential vitamins affecting various systems of the body. Cases of neuropsychiatric disorders due to its deficiency are more common in elderly patients with a prevalence rate of 10-20%; however there have been few cases reported in children and adolescents as well. The most common psychiatric symptoms reported in the literature associated with vitamin B12 deficiency was depression, mania, psychotic symptoms, cognitive impairment, dementia, delirium, acute confusional states and obsessive compulsive disorder. Subacute combined degeneration (SCD) is a neurological complication of vitamin B12 deficiency, characterized by demyelination of the dorsal and lateral spinal cord. With an early diagnosis and treatment, further development of symptoms can be prevented, before psychosis; dementia and severe depression can develop. The treatment is simple and effective and often gives very good results in these symptoms. Here we have reported a case of vitamin B12 deficiency in 19 years old, male who presented with 6 months history of paraparesis and 3 months history of psychosis. The patient was non vegetarian. Past medical history, psychiatric and family history was insignificant. Premorbid personality was unremarkable with no substance use/ exposure or infections. No stressors were present. He was diagnosed with sub acute combined degeneration with psychosis due to vitamin B12 deficiency. He was treated with antipsychotics and parenteral vitamin B12. Patient improved but some residual weakness persisted in lower limbs after 10 days of parental treatment with Vitamin B12.

## Key words

Vitamin B12 deficiency, Psychosis, Subacute combined degeneration.



## Introduction

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Vitamin B12, also known as Cobalamin is an essential nutrient. Its deficiency is a common finding in developing countries, around the world and strikes all ages and sexes. Macrocytic anemia, neuropsychiatric symptoms and glossitis are the typical symptoms reported, but this triad is lacking in many cases. The serum vitamin B12 is the best first line test; however, normal level does not exclude deficiency, therefore when the level of vitamin B12 is in the low normal range, high serum levels of methylmalonic acid and homocysteine should be used for diagnosis. There are various causes of B12 deficiency reported in the literature but most common cause is pernicious anemia. Other causes include strict vegetarians, atrophic gastritis, stomach ulcers, surgically removed stomach or intestine, crohn's disease, celiac disease, bacterial growth particularly helicobacter pylori, or parasite and medications including proton pump inhibitors (PPIs) for indigestion. We have reported a case of 19 years old boy with vitamin B12 deficiency with neuropsychiatric symptoms.

## Case report

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Patient was apparently well prior to 6 months, when he started with difficulty in walking as his right foot started getting inverted. This was followed by abnormal behavior for 3 month, consisted of running out of the house, talking irrelevantly, persecutory ideas against the family and neighbors, severe agitation, decreased eating and disturbed sleep. No history of psychoactive substance use, encephalitis, use of antipsychotics/ antiemetic, exposure to carbon-monoxide or organophosphate compounds or stressors was present. The patient was non vegetarian. Past medical history and family history was unremarkable for both

psychopathology and chronic medical disorders. Patient was seen by Psychiatrist in hospital setting and he was treated with antipsychotics, Olanzapine 15 mg/day and trifluoperazine 15 mg/day and was given a course of electroconvulsive therapy (ECTs). He improved in his psychiatric condition but his neurological condition started deteriorating hence was referred to neurology and later to psychiatry.

On physical examination, power in both upper limbs was grade 5 and lower limb was grade 4. Right toe dorsiflexion was weak and bilateral toe grips were weak. His deep tendon reflexes were depressed at both ankles. Joint position sense and vibration was impaired bilaterally up to ankle joint. Right Babinski reflex was present. His cranial nerves were intact. His gait was abnormal as there was dragging of right foot. On mental status examination, he was conscious, co-operative, relevant, and coherent. He denied any psychotic experiences and depressive cognition. He was oriented, his memory was intact. His mini mental status examination (MMSE) score was 30/30. His hemoglobin (Hb) 14.3 gm/dl, total white blood cell (WBC) 7600 cu/mm, differential counts, platelets, renal function tests, fasting blood sugar, lipid profile, liver function tests were within normal range. Thyroid function test was normal. Serum calcium, phosphorous and uric acid level were also within normal range. HIV - Elisa test was negative. His Serum Vitamin B12 level was 143 pg/ml (211-911 pg/ml is normal range).

His cerebrospinal fluid (CSF) was also normal. Fundus examination did not reveal any abnormality. No Kayser-Fleischer ring (K.F.) noted on slit lamp examination of eye. Plain CT of brain was normal. MRI scan of the whole spine was performed using sagittal T2W and STIR sequences revealed no significant abnormality.



Based on the clinical history, neuropsychiatric examination and low levels of vitamin B12, a diagnosis of Subacute combined degeneration (SCD) with psychosis with vitamin B12 deficiency was made and he was started on injectable vitamin B12 supplements (1000 mcg) for 10 days and later on shifted to oral preparation of vitamin B12. His neurological deficit improved partially within 10 days and then he was discharged from hospital with oral maintenance therapy of vitamin B12 and low doses of antipsychotics, Olanzapine 10 mg/day and trifluoperazine 10 mg/day with further plan to reduce and stop antipsychotics at follow up. However patient failed to follow up.

## Discussion

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This patient visited a Psychiatrist in a private setting due to florid psychotic symptoms which overshadowed neurological symptoms like paraparesis and numbness of the lower limbs; hence neurological symptoms were probably overlooked during the course of treatment. He was treated for psychosis with antipsychotics and electroconvulsive treatment (ECT). He showed partial improvement in psychotic features; however his neurological condition started to deteriorate hence he reported to a neurologist in a tertiary care hospital.

Vitamin B12 deficiency typically appears as lower-extremity paresthesia or ataxia, most often with concurrent folate deficiency and megaloblastic anemia [1]. Psychiatric symptoms due to a B12 deficiency commonly occur in the elderly, however these symptoms are rare in children and adolescents; hence they tend to be overlooked, like in our patient where organic nature of the Psychosis was probably not entertained due to young age.

Murat Dogan, et al. (2009) reported a case of 12 years old boy developing psychosis and extra pyramidal symptoms due to B12 and folate deficiency, improved with injectable B12 daily and folic acid twice a week. His B12 level was < 111 pmol/L, folate 5.8 nmol/L, Hb 8.4 mmol/L, MCV 98 [2]. Ali Evren (2012) reported 16 years old boy with memory problems, irritability, sleeplessness, apathy, hallucinations, delusions, concentration problems, crying, ataxia, shoulder and elbow rigidity, coordination problems, diminished thinking capability, glossitis with Vitamin B12 level 122 pmol/L, Hb 6.2 mmol/L, and MCV 98. He was treated with low dose Risperidone 0.5 mg/day + B12 injections 500 mcg a day. Risperidone was stopped in the second week; B12 injections were maintained monthly. Symptoms did not come back in the following six months [3]. These reported cases presented with various neuropsychiatric symptoms associated with low levels of vitamin B12 and hemoglobin.

Vitamin B12 deficiency usually presents with pernicious anemia and various neuropsychiatric manifestations, whereas in our patient hemoglobin level was normal (14.3 gm/dl). Numerous cases reported in the literature state that neuropsychiatric symptoms can antedate anemia for years together. Greenfield and O'Flynn (1933) stated that 14% of patients with combined degeneration of the spinal cord have normal blood values [4]. In 1960, The British Medical Journal published A.D.M Smith's 'Megaloblastic Madness'. He wrote: "The occurrence of subacute combined degeneration of the cord prior to the onset of anemia is well recognized and clinicians are now fully alive to this possibility." "The time-lag may be considerable and may give rise to diagnostic difficulty unless this situation is constantly borne in mind. Owing to the many tragedies that have resulted from unawareness, with subsequent



irreversible cord damage, it is now unusual to come across cases of this nature” [5].

Estimation of thyroid function test is also very important in B12 deficiency as there is high prevalence of B12 deficiency in patient with hypothyroidism. Jabbar A, et al. (2008) evaluated 116 hypothyroid patients from his endocrine clinic for signs and symptoms of vitamin B12 deficiency and found that there is high (approximately 40%) prevalence of B12 deficiency in hypothyroid patients [6]. Our patient did not show signs of hypothyroidism.

There are rare cases reported with association of optic neuritis with B12 deficiency. Zehetner C and Bechrakis NE reported a case of 40 years old patient with white central retinal hemorrhages in vitamin B12 deficiency [7]. Our patient had normal fundus and there was no evidence of Kayser Fleischer ring on slit lamp examination. His HIV status was also normal. A retrospective review conducted by Hepbur MJ, et al. (2004), it was seen that, low serum B12 levels occur commonly among HIV-infected patients, even at early stages without overt symptoms of B12 deficiency. Antiretroviral therapy may increase serum B12 levels [8].

MRI in Vitamin B12 deficiency may produce an increased T2-weighted signal, decreased T1-weighted signal, and contrast enhancement of the posterior and lateral columns of the spinal cord, mainly of the cervical and upper thoracic segments; however spinal cord MRI may not be a highly sensitive early test for subacute combined degeneration [9] as symptoms may precede any imaging abnormality. There was no significant abnormality seen in MRI of the whole spine in our case. In this patient several factors were pointing to an organic cause for psychosis as initial symptoms were neurological abnormality, in a well adjusted person, no previous psychiatric history with no history

suggestive of Psychosis in the family. We feel that had his psychosis been recognized and treated as a symptom of vitamin B12 deficiency much earlier, a complete reversal of his neuropsychiatric symptoms might have been expected. Although patient showed improvement in his psychosis with antipsychotics and ECTs, his neurological symptoms improved with injectable vitamin B12.

### Conclusion

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Psychiatric symptoms of a B12 deficiency are common and can be severe. With an early diagnosis and treatment, further worsening of symptoms can be prevented. All patients old and young presenting with neuropsychiatric symptoms with or without anemia should be investigated for possible Vitamin B12 deficiency and to determine its cause and whether it might be reversible. In all cases replacement therapy should be administered.

### Limitation

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Lack of evaluation for probable cause of vitamin B12 deficiency and administration of an antipsychotic may be counted among the limitations of the case.

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