Comorbidity of Vitamin B$_{12}$ Deficiency And Depression

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ABSTRACT

Physical illness can have neuropsychiatric manifestations which mimic psychiatric syndromes. Comorbidity of psychiatric syndrome and such physical illness can mislead the treating physician, resulting in delayed recognition and management of associated physical disease. A case of psychotic depression who did not respond to antidepressants and antipsychotics but recovered significantly following replacement of vitamin B12 is reported here.

1. Introduction

Vitamins are a group of substances required for effective human metabolism. Under normal circumstances it is essential that they are present in a person’s diet because, with few exceptions, the human body is unable to manufacture them. Vitamin B12 (cobalamin) is necessary for the development of red blood cells, growth, and maintenance of nervous system. The deficiency of Vitamin B 12 left untreated can cause serious neurological and neuropsychiatric complications which mimic psychiatric syndromes. A case of vitamin B12 deficiency presenting as depression, who had one depressive episode earlier, which resulted in diagnostic and therapeutic dilemma, is being reported.

2. CASE REPORT

A 42 years old male married, vegetarian, hindu patient from middle socio-economic status with no family history of psychiatric illness, no history of substance abuse, premorbidly well adjusted, presented to out-patient department of psychiatry in April 2013 with symptoms of keeping to self, sadness, decreased interest in work and excessive sleep of 15 days duration. He had a previous history of an episode in 2008 when he felt sad, kept to himself, had decreased interest in work and felt suspicious. With pharmacological treatment patient recovered considerably within 1½-2 months and stopped treatment on his own. Patient was asymptomatic till now.

Initial physical examination was unremarkable. Mental state examination revealed decreased psychomotor activity, subjectively sad and objectively depressed mood. Patient was diagnosed as Recurrent depressive disorder. Excessive sleep was ascribed to depression. He was started on Cap. Fluoxetine 20mg/day. Over the next 10-12 days, he perceived minimal improvement but developed nocturnal urinary incontinence and suspiciousness that people are talking about him & stealing his things. Due to the presence of psychotic symptoms, Tab. Aripiprazole 5mg was added; dose of Fluoxetine was increased to 40mg/day and later to 60mg/day. Over the period of next two months patient’s affective and psychotic symptoms improved partially but he continued to have urinary incontinence, increased sedation, tremors of hand and gait ataxia. Urinary incontinence was ascribed to sedation secondary to medicines. Tremors and difficulty in walking were thought to be part of drug induced extra pyramidal syndrome (EPS) and Tab. Trihexyphenidyl 2mg was added. After one month patient was found to have residual depression with psychotic symptoms, confused behaviour during night and continued urinary incontinence. Mental state examination revealed higher mental function disturbances in the form of poor attention span, disorientation to time and recent memory deficits. Neurological examination revealed grip weakness in right side (power 3/5), brisk deep tendon reflexes on right side, extensor planter response on right side, positive Romberg’s sign (swaying to right) and impaired tactile localization. In view of these findings, Organic Brain Syndrome (OBS) was considered and CT scan of brain was done which revealed early neurodegenerative changes. At this stage pigmentation of knuckles was noted. An estimation of vitamin B12 levels revealed low value of 99 pg/ml (normal range 211–911 pg/ml). Thyroid profile was normal.

He was started on intramuscular injection of mecobalamin 1000 mcg, pyridoxine HCL 100mg and nicotinamide 100mg per day. Antipsychotics were stopped and antidepressants were continued. Vitamin replacement therapy resulted in dramatic
improvement in mood, psychotic symptoms, complete reversal of higher mental function and neurological deficits. He became almost asymptomatic 10 days after vitamin replacement therapy. Parental treatment was gradually tapered off over a period of 2 months and oral supplements were started. Patient continues to maintain improvement 3 months post treatment.

3. DISCUSSION

Vitamin B₁₂ deficiency can present with wide array of psychiatric and neurologic manifestations. Most common presentation is in the form of depression[1,2,5,6,8]. Mania[8,9], catatonia[5], paranoid psychosis[3,4,7,8] and organic brain syndromes[5] have also been reported. Neurological manifestations take the form of fine & gross abnormal motor movements and pyramidal signs. Radiological abnormalities in the form of cortical atrophy have been reported[5]. Psychiatric manifestations can precede hematological changes and neurological manifestations[6]. Vitamin supplementation usually results in full remission of symptoms[3-6, 8, 9].

The present case is interesting due to several reasons. Patient had a previous major depressive episode, which was treated and resulted in complete remission of symptoms. The initial clinical picture in the present episode was depression with psychotic symptoms and it was diagnosed & treated as Recurrent depressive disorder. Presence of previous depressive episode and misattribution of initial neurological manifestations to side effects of medicines resulted in diagnostic delay of Vitamin B₁₂ deficiency. Mood disturbances preceding neurological manifestations as occurring in this case can further complicate the clinical presentation. Depression in the present episode could be a new episode of depression or as a result of Vitamin B₁₂ deficiency or both. This issue is difficult to resolve. Presence of similar psychiatric symptoms in both the episodes suggests that this episode could be another depressive episode. However in this case, treatment with psychotropic agents resulted only in partial remission. Patient remitted completely only after vitamin replacement therapy. This indicates the vitamin B₁₂ deficiency played a significant role in the occurrence of depressive symptoms. Another point of interest is, appearance of initial neurological manifestations coinciding with starting of Aripiprazole which resulted in misattribution of them to Aripiprazole. Most patients receiving Aripiprazole report insignificant incidence of extra pyramidal symptoms [10]. Appearance of neurological manifestation after Aripiprazole should alert the physician about other possible causes of EPS and should to be evaluated.

This case highlights the need of according due importance to higher mental function disturbances and neurological deficits in psychiatric syndromes.

4. CONCLUSION

Physical illness can present initially with psychiatric symptoms. Thorough evaluation of physical status of patient at initial contact and during subsequent follow up is essential to detect and manage physical illness masquerading as psychiatric syndromes.

5. References