Case Report

Misdiagnosed schizophrenia turned out to be Megaloblastic anemia

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Abstract

Medical disorders may be mistaken for a primary psychiatric disturbance because of prominent and commonly associated psychiatric or behavioural manifestations. As Vitamin B12 is essential for normal blood synthesis and neurological function; its deficiency association with many psychiatric disorders like mood disorders. In this case report, we have studied the case that presented with treatment for Schizophrenia since 7 years and investigated and treated for megaloblastic anemia.

Key words

Megaloblastic anemia, Schizophrenia, Misdiagnosis.

Introduction

Medical disorders may be mistaken for a primary psychiatric disturbance because of prominent and commonly associated psychiatric or behavioural manifestations [1]. Though the psychiatric treatment may appropriate for the symptoms but the lack of recognition of underlying medical condition often leads to misdiagnosis and relapse of the symptoms after withdrawal of treatment. Many of the times underlying medical condition is missed by the treating physician and leads to misdiagnosis. As Vitamin B12 is essential for normal blood synthesis and neurological function; its deficiency association with many psychiatric disorders like mood disorders (depression, mania, mixed episodes), confusion, delirium, panic attacks with and without phobia, hallucinations, delusion, psychosis (acute and chronic), catatonia and insomnia have been described [2, 3, 4]. Psychiatric symptoms seldom precede anaemia and present as the principal manifestation of B12 deficiency [5]. Hence cases with psychiatric manifestation are treated with only antipsychotics. However, very few case studies have been reported psychosis associated with vitamin B 12 deficiencies. In this case report, we studied the case that presented with treatment for Schizophrenia since 7 years and

investigated and treated for megaloblastic anemia.

Case report

A young 23 years old male came with parents in the psychiatric OPD of a tertiary care hospital. He presented with abnormal behaviour in form of fearfulness towards people as they are harassing him, occasionally hearing voices of unknown people. He was suspicious towards people and neighbors that they will harm him. He would cry inappropriately and shout occasionally, he would become irritable on small things and would not sleep in night. All these symptoms were present since one month, which were gradually increasing.

On detail history, he had developed psychiatric symptoms since 7 yrs which were on and off. This started with suspiciousness towards people that they will harm him, irritable behavior, disturb sleep, scholastic backwardness. He had consulted to psychiatrist who based on symptoms diagnosed as schizophrenia and started him on antipsychotics. Symptoms improved with treatment with continued long. In between he had developed increased symptoms and was stable sometimes for few months but never had full recovery. In last one year he had depressive symptoms twice lasted for 2-3 months for which antidepressant were added.

Four months back he consumed 30-35 tabs of some antipsychotic and antidepressant and was found to be a suicidal consumption and not a deliberate self harm for which he was admitted to government hospital, after discharge from hospital he was better for 3 months and again started with above presenting symptoms. As the symptoms were worsening and no complete relief, he was brought to this hospital for the first time after 7 years of onset of symptoms.

There was no history of fever, headache, vomiting, head trauma, drug intake or any addiction. There was no family history of psychiatric illness, no history of substance abuse.

On present mental status examination, he was poorly groomed with thin built, fearful, downward gaze, not maintaining eye to eye contact, occasionally shouting, sometimes muttering to self, crying inappropriate, holding mother's hand constantly, speech was incoherent and irrelevant saying people will harm him, delusion of persecution, uncooperative for attention, perceptual abnormalities, orientation, memory, concepts, insight. His judgment was impaired.

An independent diagnosis of schizophrenia was also considered but despite of the almost continuous treatment for schizophrenia, symptoms were worsening as well as relapsing possibility of Vitamin B 12 deficiency was also considered.

On further investigations, Hemogram Hb 7.6 gm/dl, WBC- 2500 /cmm with hypersegmented polymorphs, RBCs- hypochromia, anisopylokilocytosis and macrocytes ++, MCV- 115 microcmm MCH- 43 pg, MCHC- 37.5 g/dl, RDW- 21.2% , all on higher side suggestive of megaloblastic anemia. Lever function test were normal except for Serum Bilirubin was slightly raised. HbsAg for Hepatitis B was Normal. Renal Function Test and BSL were Normal. Serum gastrin was towards higher side (123.00 pg/dl), Intrinsic factor IgG: 2.3 U/ml (Negative), Anti Parietal Cell Antibodies was not detected, reduced Vitamin B12 level was detected of 164 pg/ml. Electroencephalograph shown low voltage alpha activity.

Within the available information from history and based on investigation, Pychosis with Megaloblastic Anaemia was considered.

He was hospitalized and started treatment with intramuscular injection Methylcobalmin (2500 microgram), injection Multivitamin IV, orally antibiotics, Folic acid, Iron tablets with tablet Olanzapine 2.5 mg gradually increased to 10 mg in 8 days and tablet Lorazepam 1 mg in night. After 10 days of treatment his recovery was more than 90% as reported by parents and MSE was

clear. This patient followed up regularly in 15 days interval. Injection Methylcobalamin gradually reduced as once a week for 1 month then bimonthly and monthly over 6 months. Tablet Methylcobalmin (1500 micrograms), tablet Folic acid continued once a day till 6 months. Tablet Olanzapine 5 mg continued for 3 months then reduced to 2.5 mg for next 3 months.

Complete hemogram was done every 15 days with improvement in hemoglobin and WBCs. Hematocrit Values MCV, MCH, MCHC and RDW gradually reduced to normal. After 6 months of complete course hemogram was Normal and all medications were stopped.

On follow up of four years, without any antipsychotics, he had shown further improvement in his behavior. He didn't have psychotic or affective symptom and family members addressed that he is completely fine and now interacting well with the family members and others. Till today he is visiting hospital formally because of good will. He is regular in his private job and coping well with family and colleagues.

Discussion

The present case came for seeking the treatment in the psychiatric outpatient department of a tertiary care hospital. The case reported with psychiatric manifestation primarily suggestive of Schizophrenia/ Chronic psychosis. On history, it reveals that the patient was taking consultation from psychiatrist since last 7 years for schizophrenia. He had not seen any improvement except symptomatic relief with treatment and relapse after withdrawal.

On presentation with the psychiatric manifestation for first time to our psychiatric OPD, Schizophrenia was considered as a primary diagnosis. But as the case was not responding to the treatment, we planned for the investigation to rule out underlying medical condition. Various rare psychiatric manifestations can occur in cobalamine deficiency [3]. After going through the literature which described Vitamin B12 is essential for normal blood synthesis and neurological function; its deficiency association with many psychiatric disorders like mood disorders (depression, mania, mixed episodes), confusion, delirium, panic attacks with and without phobia, hallucinations, delusion, psychosis (acute and chronic), catatonia and insomnia [3, 4]. Psychotic depression, paranoid schizophrenia [4] and other mood changes have been commonly reported.

Psychiatric manifestations can occur in the presence of low serum B12 levels but in the absence of the other well recognized neurological and haematological abnormalities of pernicious anemia [6]. So, Megaloblastic madness could be the reason that the previously treating physician may not able to recognize the Vitamin B12 deficiency as a cause of psychosis. But this cannot be confirmed as the previous reports were not available with the case. Some authors also discussed the case of a patient with vitamin B12 deficiency in whom psychiatric manifestation had been successively presented during a period of 5 years before anemia have been developed. In our study, as case had reported after 7 years of symptoms it has shown changes in hematocrit. Vitamin B12 acts as a coenzyme for L-methylmalonyl-coenzyme A mutase and methionine synthetase. Accordingly, enzymatic defects resulting from vitamin B12 deficiency lead to accumulation of methylmalonic acid and homocysteine, which appear to be proportionally related to the severity of the associated neurological and psychiatric abnormalities [7].

In present case study, after investigations vitamin B 12 deficiency was observed and for that we initiated treatment for Vitamin B12 deficiency. And the cases responded very well to the treatment. After 6 months of treatment patient was completely recovered. Patient was on regular follow up for four years without any antipsychotics drugs, also no relapse was reported and he is doing well in his personal life.

Non relapse of psychotic symptoms after four years follow up with drug free period confirms treated chronic psychosis due to Vitamin B12 deficiency.

Conclusion

Rarely, Megalobastic madness can be misdiagnosed as Schizophrenia/ chronic psychosis. So we recommend consideration of serum B12 level determination in all patients with psychiatric manifestation of schizophrenia. Also the preference should be given to the cases those are on long treatment for schizophrenia and not responding or frequent relapses are seen.

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