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Childhood Psychosis Linked to Vitamin B12 Deficiency: A Case Series

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Abstract

Psychosis in children is rare and can be challenging to treat. Treatment-resistant psychosis is particularly difficult to manage and may have underlying organic causes that are often overlooked. This case series presents two children with psychosis secondary to undiagnosed vitamin B12 deficiency, who were unresponsive to multiple antipsychotic medications. Both cases showed rapid resolution of symptoms following B12 supplementation. These cases emphasize the need for clinicians to consider vitamin deficiencies, particularly in patients with refractory psychiatric symptoms, and highlight the importance of early diagnosis and individualized treatment.

Keywords: Childhood Psychosis; Vitamin B12 Deficiency; Nutritional Correction; Antipsychotics

Introduction

Psychosis in childhood is uncommon and often associated with serious psychiatric conditions such as schizophrenia or mood disorders. However, when psychosis fails to respond to antipsychotic medications, clinicians should consider alternative causes, including metabolic or nutritional deficiencies. Vitamin B12 deficiency, although rare in children, can lead to a range of neuropsychiatric symptoms, including cognitive decline, mood disturbances, and, in severe cases, psychosis. Vitamin B12 deficiency-induced psychosis may present as an isolated psychiatric condition or in conjunction with other neurological symptoms. Importantly, it can be reversed with appropriate vitamin supplementation, which, when delayed, may result in irreversible neurological damage. This case series presents two pediatric patients with psychosis who were initially unresponsive to antipsychotic medications but showed significant improvement following the correction of vitamin B12 deficiency.

Case Descriptions

Case 1: A 10-Year-Old Boy with Treatment Resistant Psychosis

A 10-year-old boy presented with psychotic symptoms over a span of 18 months, characterized by auditory and visual hallucinations, paranoid delusions, and disorganized behavior. His parents reported that the psychotic symptoms appeared suddenly, with no prior history of psychiatric illness or developmental delay. He had no family history of psychiatric disorders. The boy was treated with various antipsychotics, including risperidone (up to 4 mg/day), olanzapine (up to 20 mg/day), and aripiprazole (up to 15 mg/day), prescribed sequentially over the course of treatment. Despite multiple trials, there was no significant

improvement in his symptoms. Most recently, he was on quetiapine 50 mg/ day, which also failed to alleviate his symptoms. He experienced marked cognitive decline, fatigue, and irritability, which raised concerns about an underlying organic cause.

Given his lack of response to antipsychotic therapy, further investigations were conducted, which revealed a low serum vitamin B12 level (93 pg/mL; normal range: 200–900 pg/mL) and elevated homocysteine levels (22 µmol/L; normal range: 5–15 µmol/L), confirming vitamin B12 deficiency. A diagnosis of vitamin B12 deficiency-related psychosis was made. The patient was started on intramuscular vitamin B12 injections (1,000 µg twice weekly). Within two weeks of supplementation, his psychotic symptoms began to improve, and a gradual tapering of quetiapine was initiated. Over the next three months, the boy remained stable and psychosis-free, with antipsychotics successfully discontinued. His vitamin B12 supplementation was adjusted to a maintenance dose of 1,000 µg monthly.

Case 2: A 14-Year-Old Girl with First Episode Psychosis

A 14-year-old girl presented with her first episode of psychosis, exhibiting paranoid delusions, social withdrawal, and irritability since the past two weeks. She had no prior psychiatric history and no family history of psychotic disorders. Initial treatment with risperidone (2 mg/day) was initiated; which was increased up to 6 mg/ day. However, after twelve weeks, her symptoms persisted without improvement. Upon further questioning, she reported a history of mild fatigue over the past several months, but there were no obvious neurological symptoms.

Laboratory evaluation revealed low serum vitamin B12 levels (105 pg/mL; normal range: 200-900 pg/mL), elevated homocysteine levels (19 µmol/L; normal range: 5-15 µmol/L), and mild macrocytic anemia (MCV 104 fL; normal range: 80-100 fL) with low hemoglobin (9.8 g/dL; normal range: 11.5-14.5 g/dL). She was diagnosed with vitamin B12 deficiency-related psychosis. Treatment with vitamin B12 injections (1,000 µg weekly) was started, and within one month, there was a significant reduction in psychotic symptoms. Over the following two months, her antipsychotic dose was reduced, and she was maintained on vitamin B12 therapy. The girl made a full recovery, and risperidone was tapered off entirely after three months of vitamin supplementation.

Discussion

The two cases presented illustrate an often-overlooked cause of pediatric psychosis: vitamin B12 deficiency. Both children demonstrated resistance to conventional antipsychotic treatment but experienced significant improvements following the correction of their vitamin B12 deficiency. This highlights the need for a broader diagnostic perspective in pediatric psychosis, particularly when patients fail to respond to standard pharmacological interventions. The importance of recognizing nutritional deficiencies as potential contributors to psychiatric symptoms is emphasized, particularly in cases where traditional treatments are ineffective.

Vitamin B12 plays a crucial role in neurological function, as it is involved in DNA synthesis, red blood cell production, and the maintenance of the myelin sheath that insulates nerve fibers. When vitamin B12 levels are low, the myelin sheath can be compromised, leading to neurological and psychiatric manifestations, including cognitive dysfunction, mood disturbances, and psychosis. Elevated homocysteine levels, often found in vitamin B12 deficiency, contribute to neurotoxicity and neurotransmitter imbalances, which can further exacerbate the psychiatric symptoms. While B12 deficiency is more commonly associated with physical symptoms such as fatigue and anemia, its psychiatric effects are significant but can be easily missed if not specifically evaluated. In both cases, the patients were initially treated with antipsychotics, including risperidone, aripiprazole, olanzapine, and quetiapine, which target dopaminergic and serotonergic systems typically implicated in psychosis. However, when psychosis is secondary to a metabolic disturbance, such as vitamin B12 deficiency, these medications do not address the underlying cause, explaining the lack of symptom resolution. The failure of antipsychotics in these cases aligns with existing literature, where the correction of the vitamin B12 deficiency, rather than the use of psychiatric medications, leads to symptom improvement [1,2].

Treatment with B12 supplementation in both patients produced rapid and marked improvements in psychotic symptoms. The 10-year-old boy, with a chronic course, required more frequent and aggressive dosing compared to the 14-year-old girl, who presented with a recent onset psychosis. Both patients had their antipsychotics tapered carefully after beginning B12 supplementation, ensuring that their psychiatric stability was maintained throughout the process. This tapering strategy minimized the risk of withdrawal symptoms or relapse, further supporting the role of vitamin B12 in their psychosis. The eventual discontinuation of antipsychotics in both cases demonstrates the effectiveness of treating the underlying deficiency and the reversible nature of B12-related psychosis. Similar cases of B12 deficiency-related psychosis have been reported in the literature, where psychiatric symptoms, including psychosis, resolved following vitamin B12 supplementation. Korde R,

et al. [3] described a 14-year-old boy with acute psychosis who experienced rapid recovery with B12 therapy. Lerner V, et al. [4] reported a 16-year-old girl with schizophrenia-like symptoms who showed complete remission of psychosis after vitamin B12 correction. In Reynolds E [5] review of pediatric cases, neuropsychiatric symptoms, including psychosis, were consistently linked to severe vitamin B12 deficiency and were resolved through supplementation.

The presence of psychiatric symptoms that are unresponsive to antipsychotics should prompt consideration of metabolic or nutritional etiologies, such as vitamin B12 deficiency. While vitamin B12 deficiency is commonly linked to physical signs like macrocytic anemia, as seen in the second case, it can manifest without significant hematological abnormalities, as in the first case. Thus, clinicians must remain vigilant, particularly when faced with non-resolving psychiatric symptoms in pediatric patients. Testing for vitamin B12 and homocysteine levels is relatively straightforward and can provide valuable diagnostic insights that may be otherwise overlooked. The rapid symptom resolution observed in both cases following B12 supplementation underscores the importance of early recognition and intervention in cases of deficiency-related psychosis. Timely diagnosis not only leads to the remission of psychiatric symptoms but also prevents potential long-term neurological damage associated with prolonged deficiency. These cases further highlight the importance of addressing the root cause of psychosis, rather than solely managing symptoms with antipsychotic medications, especially when traditional treatments fail to yield results.

Conclusion

The unique aspect of these cases lies in the identification of vitamin B12 deficiency as the underlying cause of psychosis in pediatric patients who were unresponsive to standard antipsychotic treatment. The significant clinical improvement after vitamin B12 supplementation, along with the successful tapering and discontinuation of antipsychotics, reinforces the need for routine consideration of nutritional deficiencies in the differential diagnosis of pediatric psychosis. Early detection and correction of such deficiencies can lead to full recovery and prevent unnecessary long-term pharmacological intervention.

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