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Articles:

Case Report

Beyond Thrombosis: Late-Onset Psychosis with Catatonic Features in Primary Antiphospholipid Syndrome

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CONFLICTS OF INTEREST STATEMENT

None of the above authors have any conflicts of interest to report regarding this case report.

ABSTRACT

This case highlights an unusual presentation of primary antiphospholipid syndrome with late-onset psychosis and catatonic features. Given the limited literature on neuropsychiatric manifestations of APS, we are hopeful this case will contribute to the existing literature on the subject.

Keywords: antiphospholipid syndrome, autoimmune, catatonia, psychosis

INTRODUCTION

Antiphospholipid syndrome (APS) is a systemic autoimmune disorder characterized by thrombosis and pregnancy morbidity in the presence of antiphospholipid antibodies, including lupus anticoagulant, anticardiolipin antibodies, and beta-2 glycoprotein I antibodies. APS may occur as a primary disorder or in association with other autoimmune diseases, most commonly systemic lupus erythematosus (SLE).[1]

Neurologic involvement is common in APS. Cerebral ischemia and stroke represent the most frequently reported manifestations, although other central nervous system complications including cognitive dysfunction, seizures, headaches, and neuropsychiatric symptoms have also been described.[2,3] Psychiatric manifestations associated with APS include psychosis, depression, bipolar disorder, and schizophrenia-spectrum presentations, though these remain incompletely understood.[2,4]

Although neuropsychiatric symptoms have been described in APS, psychosis remains an uncommon and poorly characterized manifestation. A 2018 systematic review identified only 23 published reports of APS-associated psychosis, most of which consisted of isolated case reports or small case series.[5] Delusions and hallucinations were the most frequently described psychiatric symptoms.[5]

The pathophysiology of neuropsychiatric APS remains incompletely understood. Proposed mechanisms include microvascular thrombosis, endothelial dysfunction, inflammatory injury, disruption of the blood-brain barrier, and immune-mediated neuronal damage.[2,6]

CASE PRESENTATION

A male in his 70s with a medical history significant for hypertension, hyperlipidemia, type 2 diabetes mellitus, coronary artery disease, recurrent lower extremity deep vein thromboses, and prior pulmonary embolism on chronic rivaroxaban therapy presented to the emergency department with left-sided pressure-like chest pain lasting several minutes. Initial cardiac workup showed negative troponins and first-degree atrioventricular block on electrocardiogram.

The patient had no prior psychiatric diagnoses, psychiatric hospitalizations, suicide attempts, or psychotropic medication exposure. He denied illicit substance use and consumed alcohol occasionally.

On hospital day 2, the patient developed psychiatric symptoms characterized by confusion, auditory hallucinations, and visual hallucinations. He reported hearing the voices of his son and daughter-in-law despite their absence and described seeing holes in the walls and lights behind paintings that were not present. His wife reported these symptoms had been occurring intermittently for approximately 1 to 2 weeks prior to admission.

Neurology initiated an extensive diagnostic evaluation. Laboratory studies including complete blood count, comprehensive metabolic panel, thyroid stimulating hormone, vitamin B12, erythrocyte sedimentation rate, C-reactive protein, morning cortisol, anti-thyroid peroxidase antibodies, anti-thyroglobulin antibodies, antinuclear antibodies, anti-double stranded DNA antibodies, HIV testing, and syphilis testing were unremarkable. Ammonia was mildly elevated.

Magnetic resonance imaging (MRI) of the brain demonstrated no acute intracranial abnormalities, showing only minimal chronic microvascular ischemic changes. Electroencephalography (EEG) showed no epileptiform activity. Cerebrospinal fluid analysis demonstrated mildly elevated protein with normal glucose, no pleocytosis, and negative infectious studies.

Autoimmune evaluation revealed positive lupus anticoagulant, anticardiolipin antibodies, and beta-2 glycoprotein antibodies. Psychiatry was consulted on hospital day 5, and the patient was started on trazodone 25 mg nightly for presumed multifactorial encephalopathy. His symptoms improved during hospitalization, and he was discharged home.

Three months later, the patient returned to the emergency department after his wife found him holding kitchen knives while expressing suicidal ideation. She described a rapid escalation of symptoms over several days. The patient expressed fixed persecutory beliefs that he had murdered individuals, buried them in his backyard, and molested his granddaughter. He perseverated on the need to confess these crimes despite his wife adamantly denying they had occurred. He additionally endorsed paranoia regarding neighbors and suicidal ideation with a plan to drown himself.

Mental status examination demonstrated speech latency, decreased speech production, psychomotor slowing, and restricted affect. Given these findings and decreased engagement, psychiatry raised concern for catatonia. A lorazepam challenge with oral lorazepam 1 mg resulted in noticeable improvement in engagement and speech latency. Lorazepam 1 mg three times daily was initiated but later reduced because of sedation. Low-dose risperidone 0.5 mg nightly was also initiated.

Repeat MRI, EEG, cerebrospinal fluid studies, and autoimmune/paraneoplastic encephalitis panels remained unrevealing. However, repeat antiphospholipid antibody testing again revealed positive lupus anticoagulant, anticardiolipin antibodies, and beta-2 glycoprotein antibodies. In conjunction with his history of recurrent venous thromboembolism, these findings confirmed primary APS.

Given concern for an immune-mediated neuropsychiatric process, the patient was treated with high-dose intravenous corticosteroids followed by an oral taper. Hematology additionally recommended transition from rivaroxaban to warfarin with an enoxaparin bridge. Following this regimen, the patient demonstrated marked clinical improvement. At two-year follow-up, he remained psychiatrically stable without recurrence of psychosis or evidence of progressive cognitive decline.

DISCUSSION

The differential diagnosis included autoimmune encephalitis, rapidly progressive neurocognitive disorder, primary psychotic illness, and APS-associated psychosis. Autoimmune encephalitis remained an important consideration given the subacute psychiatric presentation and steroid responsiveness; however, extensive repeat testing was unrevealing.

A primary neurocognitive disorder was felt to be less likely given the absence of progressive cognitive decline, minimal structural abnormalities on neuroimaging, and the patient's return to baseline functioning. Similarly, a primary psychiatric disorder was considered less likely given the patient's late age of onset, absence of prior psychiatric history, fluctuating course, catatonic features, and underlying autoimmune disease.

Psychosis associated with APS remains rare and incompletely understood. Existing literature suggests that later age of onset, abrupt symptom development, hallucinations, delusions, and coexisting thrombotic disease should raise suspicion for APS-associated psychosis.[5]

The pathophysiology underlying neuropsychiatric APS is likely multifactorial. Proposed mechanisms include microvascular ischemia, endothelial dysfunction, inflammatory injury, blood-brain barrier disruption, and direct neuronal effects of antiphospholipid antibodies.[2,6] The patient's favorable response to corticosteroids and anticoagulation further supports a possible immune-mediated process.

Overall, this presentation emphasizes the importance of considering autoimmune etiologies in patients presenting with atypical late-onset psychosis, particularly in the setting of thrombotic disease or persistently positive antiphospholipid antibodies.

CONCLUSION

This case describes an unusual presentation of primary APS manifesting as recurrent late onset psychosis with catatonic features. Clinical improvement following corticosteroids, lorazepam, antipsychotic treatment, and transition to warfarin further supports a possible immune-mediated neuropsychiatric process. Autoimmune etiologies should be considered in patients presenting with atypical late-onset psychosis and catatonia.

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Editorial

Antipsychotic-Induced Weight Gain Isn't a Side Effect — It's Iatrogenic Metabolic Syndrome

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CONFLICTS OF INTEREST STATEMENT

The above author does not have any conflicts of interest to report regarding this editorial.

ABSTRACT

Editorial on the concept of weight gain associated with antipsychotic use.

Keywords: weight gain, antipsychotics, metabolic syndrome, side effect

Introduction

Antipsychotic medications are the cornerstone of treatment for managing schizophrenia spectrum disorders and bipolar disorder and are often used in a litany of other psychiatric conditions. Yet, for too long, significant weight gain and metabolic derangements have been viewed as mere "side effects." This language undermines the severity of the issue and delays interventions. It's time we reframe antipsychotic-induced weight gain (AIWG) for what it truly is: a foreseeable, iatrogenic metabolic syndrome. As psychiatrists, we must stop tolerating preventable harm and begin treating metabolic risks with the same urgency as psychiatric symptoms.

The Scope of Harm

Antipsychotic-induced weight gain impacts approximately 20-30% of adults within the first year of treatment. These rates tend to be higher in children and adolescents. The severity and risk of weight gain vary, with clozapine and olanzapine carrying the highest risk (4-10 kg over 6-12 months). Even so-called "lower-risk" of "weight neutral" antipsychotics such as aripiprazole are not metabolically neutral in real-world use.

Due to the risk of metabolic syndrome—elevations in weight, dyslipidemia, and diabetes—both the American Psychiatric Association and the American Diabetes Association recommend routine monitoring for all patients prescribed antipsychotics. Despite these clear guidelines from the APA and ADA, adherence in clinic practice remains inconsistent. Too often, weight gain is ignored or deferred to primary care to manage.

Reframing Language and Responsibility

Much like monitoring agranulocystosis with Clozapine, psychiatrist must be equipped and expected to manage metabolic health proactively. This begins with transparent, pre-treatment discussions about weight gain risk and includes thoughtful antipsychotic selection. But it must go further.

Proactive Management Strategies

Lifestyle modifications—education on nutrition, physical activity, and behavioral changes—are considered first line although evidence suggests the impact is modest without structured support. Many psychiatrists feel unprepared to guide patients in nutrition or exercise due to inadequate training. However, we are well-positioned to use motivational interviewing techniques to support behavior change.

Pharmacologic treatment should no longer be considered a last resort, it should be discussed and implemented early. Off label Metformin is supported by strong evidence for the treatment of AIWG with typical losses of 3-4kg. GLP-1 receptor agonists—such as liraglutide and semaglutide—are emerging as highly effective options. Tirzepatide (a GLP-1 and GIP agonist) could also be considered although current data is limited. While cost and access remain barriers, the risk-benefit ratio increasingly favors early pharmacologic intervention. Switching to a lower-risk antipsychotic may be appropriate in select cases but should never compromise psychiatric stability.

An Ethical Imperative

If a patient were to develop metabolic syndrome because of psychiatric treatment, would we consider this an inevitability or a preventable complication? If we accept AIWG as iatrogenic harm, we are obligated to prevent and treat it. Reframing also impacts patients by allowing open dialogue and shared decision making.

Conclusion

Antipsychotic-induced weight gain is not just a side effect. It is a foreseeable and preventable condition. Prevention, including open discussion of risks and optimizing medications based on risk profile, is paramount when prescribing antipsychotics. Psychiatry must move beyond passive monitoring to active prevention and intervention. Just as we would never prescribe clozapine without monitoring white blood cell counts, we should never prescribe antipsychotics without a plan to address metabolic health. This is not just good medicine—it's our responsibility.

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Vitamin D Deficiency/Insufficiency and Mental Health in a Midwestern Child and Adolescent Inpatient Psychiatric Population

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CONFLICTS OF INTEREST STATEMENT
None of the above authors have any conflicts of interest to report.

ABSTRACT
Background: Vitamin D deficiency is a growing concern in the United States, particularly among children and adolescents. Vitamin D deficiency is often linked to disorders of cognition and mental health and is common in psychiatric patients.
Aims: This aim of this study was to determine the prevalence of vitamin D deficiency in the Midwestern child and adolescent psychiatric population. We examine the potential influence of vitamin D deficiency on mental health outcomes, considering the findings of relevant nutritional studies.
Methods: In this retrospective descriptive study, we reviewed charts of 1,363 child and adolescent psychiatric inpatients in a Midwestern community teaching hospital between January 1, 2025 and June 30, 2025. Of those patients, 585 had blood work including a Vitamin D level checked. We defined vitamin D deficiency as a level less than 20 ng/ml and insufficiency as less than 30 ng/ml. Data analysis was completed using a one-sample t test.
Results: 29.2% (171 out of 585) patients were classified as deficient in Vitamin D and 41.6% (243 out of 585) were classified as insufficient. Among children aged 4-11, the rate for deficiency was 35.7% (25 out of 70) and the rate for insufficiency was 27.1% (19 out of 70.) For adolescents aged 12-17, the rate for deficiency was 28.3% (146 out of 515) and the rate for insufficiency was 43.3% (223 out of 515.) Therefore overall, 70.8% (414 out of 585) of our population was either vitamin D deficient or insufficient.
Conclusions: Vitamin D deficiency remains prevalent in the child and adolescent psychiatric population. In particular, the rate of deficiency among the child and adolescent psychiatrically hospitalized patients was significantly higher than the general population. There is a need for further research highlighting the relationship between vitamin D deficiency and mental illness in the child and adolescent psychiatric population. In addition, this population is actively growing and developing, potentially placing them at risk for long-term mental illness; and future issues such as osteoporosis or poor bone development.

Keywords: Vitamin D deficiency, mental health, hospitalization

Introduction

Vitamin D plays a crucial role in bone health, immune function and has been linked to mental health concerns. Studies have found an association between vitamin D deficiency and various mental health conditions, including depression, anxiety, and schizophrenia [1, 2]. The Midwest, characterized by limited sun exposure during winter months due to temperature and sunlight hours; and potential dietary limitations, presents risk factors for vitamin D deficiency in the child and adolescent population. The level utilized for determining whether a person is deficient varies based on the study and by different governing bodies. For the purpose of this study, we classified deficient as less than 20 ng/ml and insufficient as less than 30 ng/ml as done in other studies[3].

Several studies have documented a high prevalence of vitamin D deficiency among psychiatric inpatients [4, 5, 6]. Notably, Zheng et al. (2016) found that over 67% of adult psychiatric inpatients in Kansas City exhibited vitamin D deficiency or insufficiency [6]. While research specifically focused on a small region of the Midwest is limited, these findings suggest a similar trend might be present in the region's child and adolescent psychiatric population.

Method

In this retrospective descriptive study, we reviewed charts of 1, 363 child and adolescent psychiatric inpatients in a midwestern community teaching hospital between January 1, 2025 and June 30, 2025. Of those patients, 585 had blood work including a vitamin D level checked. We defined vitamin D deficiency as a level less than 20 ng/ml and insufficiency as less than 30 ng/ml.

A one-sample t test was used to compare our sample of child and adolescent patients with deficient or insufficient vitamin D levels, to a hypothesized population mean. A one-sample t test assumes that the dependent variable is normally distributed within the population, and the data are independent, meaning the vitamin D levels of one patient are not dependent on the vitamin D levels of the other patients. We compared adolescent and pre-adolescent vitamin D levels at insufficient, and deficient percentages to the national percentages of insufficient, and deficient vitamin D levels.

Setting

This review examined charts from a 60-bed inpatient child and adolescent psychiatric hospital which treats psychiatrically ill patients in the Midwest. Patients treated at this hospital range in age from 4 to 17 years. The hospital serves a wide variety of patients encompassing all sociodemographic characteristics found throughout the region. Data for this study was collected between January 1st 2025 and June 30th 2025.

Sample

The sample included patients that were selected for the review based on having bloodwork completed while in the hospital, including a vitamin D Level. The majority of patients who receive bloodwork during an admission will have vitamin D checked, if no record of it is already on file within the last 6 months. Psychiatric evaluation was performed by attending psychiatrists as part

of the routine assessment using criteria from the DSM-V TR. All patients received a complete medical assessment from the onsite pediatric service during their hospitalization.

Laboratory Analyses

Serum 25-(OH) D was analyzed through in house laboratory services.

Data Analysis

Study data were analyzed for statistical significance when compared to national levels of vitamin D at insufficient, and deficient levels. A sample of 585 (n=585) patients was included in data analysis. Sample groups included pre-adolescent (n=70) and adolescent patients (n=515). Data from the national population included a sample of 826 individuals (n=826). Vitamin D deficiency was defined as a level less than 20 ng/ml and vitamin D insufficiency was defined as less than 30 ng/ml.

Results

Data analysis showed the mean rate of vitamin D deficiency in the adolescent patient population was 14.12, while the mean rate of vitamin D insufficiency was 23.95. In the pre-adolescent patient population, the mean rate of vitamin D deficiency was 16.61, and the mean rate of vitamin D insufficiency was 24.25.

Among patients included in the study sample (n=585), 171 (29.2%) were classified as being deficient in vitamin D, while 243 (41.6%) were classified as being insufficient in vitamin D. In children aged 4-11, (n=70), 25 (35.7%) had deficient vitamin D levels, and 19 (27.1%) had insufficient vitamin D levels. For adolescents, aged 12-17, (n= 515), 146 (28.3%) were classified as vitamin D deficient, while 223 (43.3%) were classified as vitamin D insufficient. Therefore, 414 (70.8%) of patients included in the study had either deficient or insufficient levels of vitamin D.

According to a previous study, which included a general population sample (n=826), 195 (23%) of the participants were vitamin D deficient, while 218 (42%) were Vitamin D insufficient[3]. For our data analysis, we compared the percentages of pre-adolescents and adolescents who were vitamin D deficient and insufficient, to the national percentages of the population who were also vitamin D deficient and insufficient.

Results indicated a statistically significant difference (p=0.05, CI=95%) among vitamin D deficient adolescents compared with vitamin D deficient individuals of the national population. There was also a statistically significant difference (p=0.05, CI=95%) among pre-adolescents who were vitamin D deficient compared to the national population, as well as among pre-adolescents who were vitamin D insufficient compared to the national population.

Nutritional Considerations

Vitamin D is obtained through sun exposure and dietary intake. Fatty fish, egg yolks, and fortified milk are primary dietary sources. However, achieving adequate vitamin D levels solely

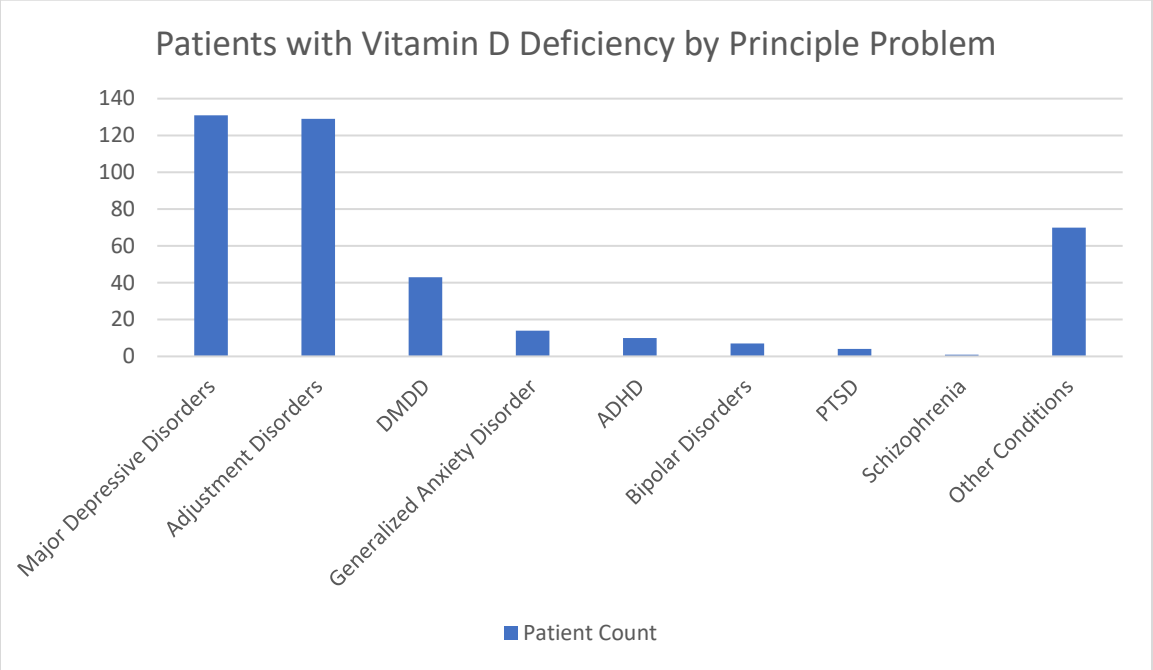
through diet can be challenging, particularly for children with limited dietary diversity or those with lactose intolerance.

The population included in our study varies widely in terms of socioeconomic status, access to a variety of healthy foods, and dietary limitations. Each of these factors may have contributed to the vitamin D deficiencies and insufficiencies frequently found in the study population. Thus, it is vital for these individuals to have additional vitamin D supplementation.

Impact of Vitamin D on Mental Health

The mechanisms underlying the potential link between vitamin D deficiency and mental health remain unclear. Vitamin D receptors are present in brain regions associated with mood regulation and neurotransmitter function [7]. Studies suggest vitamin D may influence neurotrophic factors, essential for neuronal growth and survival [8].

Observational studies have shown a correlation between low vitamin D levels and increased risk of depression [1]. Supplementation trials have yielded mixed results, with some demonstrating improvement in depressive symptoms, while others showed limited effects [8, 9]. Further research is needed to determine the efficacy of vitamin D supplementation for treating mental illness.



Conclusions:

Limited sun exposure and potential dietary limitations raise concerns about the prevalence of vitamin D deficiency in the Midwestern child and adolescent psychiatric population. Studies suggest a possible link between vitamin D deficiency and mental health; however, a cause-and-effect relationship has yet to be established. Our study indicated a statistically significant

difference in the percentage of pre-adolescents and adolescents who are vitamin D deficient or insufficient, when compared to the national population, which is an important factor to consider when treating mental illness in this population. In our study, patients who were deficient in Vitamin D presented with principle problems most commonly related to psychosocial stressors and depressive symptoms. They presented at a higher rate than patients who presented for behavioral disturbances. Further research investigating the interaction between vitamin D status and mental health outcomes in this specific population is warranted.

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