Cureus
Part of SPRINGER NATURE

Open Access Case Report

First-Episode Psychosis in a Patient With Flaccid Paraplegia Following Childhood Poliomyelitis

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Review began 09/29/2026
Review ended 10/16/2026
Published 10/20/2026

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DOI: 10.7759/cureus.118229

Abstract

First-episode psychosis in patients with pre-existing neurological disability presents important diagnostic and therapeutic challenges, particularly when the relationship between the two conditions is uncertain. This case report describes a 49-year-old man with flaccid paraplegia following childhood poliomyelitis who was involuntarily admitted for a first documented acute psychotic episode. His presentation included behavioural disorganisation, self-neglect, aggression, disorganised speech, and persecutory delusions. Initial laboratory investigations, including a complete blood count, inflammatory markers, electrolytes, glucose, and renal and hepatic function tests, were unremarkable. Urine toxicology screening was negative for the substances tested, while cranial computed tomography and neuropsychological assessment did not reveal acute structural abnormalities or major cognitive impairment that could explain the psychotic symptoms. However, more extensive neurological investigations were not performed. Clinical improvement was achieved with oral aripiprazole followed by long-acting injectable aripiprazole, alongside coordinated psychiatric and social support. This case highlights the importance of avoiding unsupported causal assumptions between remote neurological disease and new-onset psychosis, while recognising the diagnostic limitations of the available investigations. It also illustrates how chronic physical disability and psychosocial vulnerability can complicate psychiatric management, making multidisciplinary care essential for treatment continuity and functional stability.

Categories: Psychiatry
Keywords: differential diagnosis, first-episode psychosis, long-acting injectable antipsychotic, neurological disability, poliomyelitis

Introduction

Poliomyelitis is an enteroviral infection with tropism for motor neurons, particularly those of the anterior horn of the spinal cord, and may result in acute flaccid paralysis and permanent motor sequelae [1,2]. Although widespread vaccination has markedly reduced its incidence, poliomyelitis remains a concern in areas with insufficient immunisation coverage. The disease mainly affects children under five years of age, although unvaccinated individuals of any age remain susceptible [3]. Paralytic poliomyelitis may result in persistent muscle weakness, atrophy, and impaired mobility. Decades after the acute infection, some survivors develop post-polio syndrome, typically 15-40 years later, characterised by new or progressive weakness, fatigue, and pain [1,2,4]. These chronic consequences can substantially affect mobility, independence, participation, and quality of life [5].

Beyond physical limitations, long-standing disability may involve dependence on others, environmental barriers, and difficulties accessing healthcare and social support, potentially contributing to psychosocial vulnerability. Psychiatric morbidity has also been described among poliomyelitis survivors. However, available epidemiological evidence does not establish a specific relationship between poliomyelitis and psychotic disorders, and a direct pathophysiological mechanism linking remote poliovirus infection to psychosis has not been demonstrated [6]. The coexistence of both conditions in an individual patient should therefore not be interpreted as evidence of causality.

Psychosis is a clinical syndrome characterised by symptoms such as delusions, hallucinations, and disorganised thinking or behaviour. However, the identification of psychotic symptoms does not necessarily establish a specific psychotic disorder, which requires consideration of the clinical presentation, symptom duration, course, and differential diagnosis according to established classification systems [7]. In particular, distinguishing primary psychotic disorders from psychosis associated with medical conditions or substance use is essential for appropriate diagnostic assessment [8].

New-onset psychosis in middle age warrants careful assessment for secondary causes, particularly when neurological disease or disability is already present. The extent of neurological investigation should be guided by the clinical presentation and by features that raise suspicion of an underlying neurological, infectious, metabolic, toxic, or immune-mediated disorder [8]. Autoimmune encephalitis and related syndromes are important considerations when psychiatric symptoms occur with warning signs such as

seizures, altered consciousness, movement abnormalities, catatonia, autonomic instability, or new focal neurological findings [9-11]. A systematic review and meta-analysis found clinically relevant abnormalities on brain magnetic resonance imaging (MRI) in a minority of patients with first-episode psychosis, underscoring the importance of specifying which imaging investigations were performed [12].

This case report describes a first documented psychotic episode in a man with long-standing flaccid paraplegia following childhood poliomyelitis. Its objective is to illustrate the diagnostic challenges of identifying and classifying new-onset psychosis in the context of chronic neurological disability, while examining how psychosocial vulnerability may influence clinical presentation and management. The case was selected to highlight the importance of avoiding unsupported causal assumptions, acknowledging diagnostic uncertainty, and integrating psychiatric treatment with individualized psychosocial and community support.

Case Presentation

A 49-year-old man with a medical history of paralytic poliomyelitis in childhood and chronic flaccid paraplegia of the lower limbs was involuntarily admitted to a psychiatric unit in 2022 following reports of progressively worsening behavioral disturbances, characterized by marked self-neglect, accumulation of objects, disorganized speech, recurrent interpersonal conflicts, and episodes of aggression directed toward neighbors. The historical diagnosis of poliomyelitis was documented in his medical history; however, records from the original childhood illness were not available for review. He had permanent wheelchair dependence and received social support for daily living. His educational attainment was equivalent to the sixth grade, and he had not been engaged in regular employment for several years. No relevant family history was known, and he did not identify family members or friends as sources of social support. Previous psychiatric follow-up between 2013 and 2015 had been related to alcohol and other psychoactive substance use. At the time of the index admission, he was not receiving regular psychiatric care or maintenance psychotropic medication. The available clinical information did not allow his current substance use status to be reliably established.

During the weeks preceding admission, the community mental health team and people in his residential environment reported progressive self-neglect, characterized by poor personal hygiene and deterioration in daily self-care, alongside disorganized behavior, including the repetitive collection and accumulation of bottles and papers. Recurrent interpersonal conflicts and episodes of aggression directed toward neighbors were also reported. His speech became increasingly disorganized, with derailment of thought. Regarding thought content, he expressed persecutory beliefs involving his neighbors, whom he perceived as hostile and discriminatory toward him because of his immigrant background. Grandiose and religiously themed ideas were also present, including statements in which he claimed to be God. No hallucinations were documented during clinical assessment.

On admission, he was poorly groomed but oriented to person, place, and time. During the interview, he displayed an excessively familiar interpersonal manner, with reduced social boundaries toward the examiner. Speech was spontaneous, while the form of thought was characterized by tangentiality and derailment, reflecting impaired coherence and continuity of thought. Thought content included the persecutory and grandiose-religious beliefs described above. Mood was assessed as anxious, with affect congruent with the expressed emotional content. Mood was anxious, with broadly congruent affect. He did not express active suicidal or homicidal intent during examination. There was no fluctuating level of consciousness. Neurological examination was notable for the known chronic flaccid paraplegia and areflexia of the lower limbs, without newly identified focal neurological deficits.

Initial laboratory investigations included a complete blood count, inflammatory markers, renal and hepatic function tests, electrolytes, and glucose. No clinically relevant abnormalities were identified. Urine toxicology screening was negative for cannabinoids, cocaine, amphetamines, opioids, and benzodiazepines. Non-contrast cranial computed tomography showed no acute intracranial lesion, hemorrhage, mass effect, or other structural abnormality that could account for the presentation. Formal neuropsychological assessment demonstrated preserved global cognitive performance without evidence of major cognitive impairment or a dementia syndrome.

No seizures, new abnormal involuntary movements, autonomic instability, catatonia, fever, delirium, or rapidly progressive cognitive decline were documented during admission. In the absence of additional clinical warning signs suggestive of an acute neurological or autoimmune process, further investigations, including brain magnetic resonance imaging, electroencephalography, cerebrospinal fluid analysis, and neuronal autoantibody testing, were not pursued at that stage. The investigations performed did not identify an evident metabolic, substance-related, acute structural, or major cognitive explanation for the psychotic presentation. Nevertheless, the neurological and autoimmune work-up was not exhaustive, and other potential causes could not be definitively excluded.

Aripiprazole was initiated orally and titrated to 20 mg/day for the treatment of psychotic symptoms, with progressive improvement in behavioral organization and attenuation of delusional intensity. Given limited insight and concerns regarding adherence after discharge, treatment was transitioned to long-acting

injectable aripiprazole 400 mg intramuscularly every month after initial stabilization. Quetiapine 200 mg nightly was prescribed for sleep disturbance, while lorazepam 2.5 mg was administered from the beginning of hospitalization to manage anxiety and agitation. The pharmacological regimen was well tolerated, with no adverse effects reported or observed during admission.

No structured psychotherapeutic intervention was documented during admission. Management also involved social services and the community mental health team. Home-based support for meals and cleaning was organized, together with structured psychiatric follow-up after discharge. As his behavior stabilized and he accepted treatment, the admission status was changed from involuntary to voluntary. The discharge diagnosis was psychosis not otherwise specified [psychosis not otherwise specified (NOS); International Classification of Diseases (ICD)-10 code F29], reflecting the presence of a clinically significant psychotic syndrome without sufficient evidence to establish a more specific psychotic disorder. At discharge, he was cooperative and behaviorally settled, with improved thought organization and no active psychotic symptoms associated with immediate risk. During the available outpatient follow-up, he remained clinically stable on monthly long-acting injectable aripiprazole without a documented further psychotic decompensation. He also reported feelings of loneliness associated with his mobility limitations and was awaiting a replacement wheelchair.

Discussion

The principal diagnostic challenge in this case was determining the nature of a first documented psychotic episode in a middle-aged patient with long-standing neurological disability. The clinical presentation was characterized by persecutory beliefs, grandiose-religious ideation, formal thought disorder with tangentiality and derailment, behavioral disorganization, limited insight into the need for psychiatric treatment, and marked functional deterioration. These findings supported the identification of a clinically significant psychotic syndrome. The diagnosis established at discharge was psychosis not otherwise specified (psychosis NOS; ICD-10 code F29), reflecting the presence of psychotic symptoms without sufficient evidence to establish a more specific disorder. According to ICD-11, the classification of primary psychotic disorders requires consideration of symptom characteristics, duration, longitudinal course, and exclusion of alternative explanations [7]. In this patient, the available clinical information did not permit a more specific diagnostic classification, emphasizing the importance of longitudinal reassessment.

Schizophrenia was an important diagnostic consideration because of the coexistence of delusional beliefs, formal thought disorder, and significant functional impairment. However, the available documentation did not establish whether characteristic psychotic symptoms had persisted for the minimum duration required by ICD-11 [7]. Acute and transient psychotic disorder was another possibility, given the relatively recent clinical deterioration and subsequent improvement with treatment. Nevertheless, the precise onset, duration, and pattern of symptom fluctuation were insufficiently documented to confirm its diagnostic requirements. Delusional disorder was considered less compatible with the clinical presentation because of the prominent formal thought disorganization and behavioral deterioration, which extended beyond a relatively circumscribed delusional system [7]. These considerations supported maintaining a nonspecific diagnosis rather than assigning a more narrowly defined primary psychotic disorder.

Psychotic symptoms occurring in the context of a mood disorder also warranted consideration. Although anxiety was documented during the psychiatric examination, the available clinical information did not establish a concurrent manic, mixed, or depressive episode sufficient to support a mood disorder with psychotic symptoms or schizoaffective disorder [7]. Substance-induced psychosis was another relevant differential diagnosis, particularly given the patient's previous psychiatric follow-up for alcohol and other psychoactive substance use. Urine toxicology screening at admission was negative for the substances tested, but this result could not establish sustained abstinence or definitively exclude recent substance exposure. Furthermore, the available history did not demonstrate a clear temporal relationship between substance use or withdrawal and the onset of psychotic symptoms. Consequently, a substance-induced etiology was not supported by the documented findings, although it could not be conclusively excluded.

Secondary psychosis related to a medical or neurological condition was particularly relevant because the psychiatric episode occurred in middle age in a patient with pre-existing neurological disability [8]. Initial laboratory investigations showed no clinically relevant metabolic abnormalities, cranial computed tomography revealed no acute intracranial abnormalities, and neuropsychological assessment did not demonstrate major cognitive impairment. The absence of documented fluctuations in consciousness, seizures, new focal neurological deficits, new abnormal involuntary movements, autonomic instability, or rapidly progressive cognitive deterioration reduced clinical suspicion of delirium or an acute neurological process. Nevertheless, these findings cannot be equated with comprehensive exclusion of secondary causes of psychosis. Autoimmune encephalitis and related immune-mediated conditions may initially present with prominent psychiatric manifestations, particularly when accompanied by neurological warning signs [9-11]. Brain magnetic resonance imaging may identify clinically relevant abnormalities in a minority of patients with first-episode psychosis [12].

The relationship between childhood poliomyelitis and the subsequent psychotic episode requires equally cautious interpretation. Poliomyelitis primarily affects motor neurons, and the patient's childhood infection

occurred several decades before the psychiatric presentation. Although an epidemiological cohort study reported increased overall psychiatric hospitalization among polio survivors, the available evidence does not establish a specific causal association between poliomyelitis and psychotic disorders [6]. Accordingly, the available evidence does not support a direct causal relationship between remote poliovirus infection and psychosis, although the potential contribution of psychosocial adversity associated with chronic disability should also be considered.

Nevertheless, the patient's chronic motor disability and psychosocial circumstances were important for understanding his clinical presentation and management. Wheelchair dependence, limited social support, prolonged occupational inactivity, and dependence on community services created practical vulnerabilities that were amplified by psychotic disorganization, impaired self-care, and reduced insight. His reported loneliness and difficulties associated with mobility further illustrate the psychosocial burden of long-standing disability. Qualitative research involving polio survivors has emphasized the importance of accessibility, participation, and independence in determining quality of life [13]. These circumstances may be understood within the stress-vulnerability model, which recognizes that psychosocial stressors interact with individual biological and psychological vulnerabilities in the development and expression of psychotic symptoms [14]. However, the extent of any pre-existing individual or genetic vulnerability, as well as the contribution of specific psychosocial stressors to the onset of psychosis in this patient, could not be established. Therefore, a multifactorial interpretation is appropriate, without attributing a direct causal role to chronic disability or any individual psychosocial factor.

Treatment was directed toward reducing psychotic symptoms, restoring behavioral organization, and ensuring continuity of care after discharge. Following clinical improvement and good tolerability with oral aripiprazole, long-acting injectable aripiprazole was selected for maintenance treatment, taking into account the patient's psychotic presentation, response to oral treatment, and anticipated difficulties with adherence due to limited insight into the need for psychiatric treatment. Evidence from studies involving patients with schizophrenia supports the use of long-acting injectable antipsychotics for maintenance treatment, particularly when adherence and continuity of care are priorities [15]. The pharmacological regimen was well tolerated, and clinical improvement was observed during admission. Treatment was combined with community psychiatric follow-up and social intervention, including home-based support for daily living. Such measures are consistent with evidence supporting community-based social interventions for people with severe mental illness [16]. Although clinical stabilization was achieved, the observational nature of this report prevents attributing the outcome to any single component of treatment.

This report has several limitations. First, although childhood paralytic poliomyelitis was part of the established medical history and the chronic neurological phenotype was compatible with previous lower motor neuron injury, the original diagnostic records were unavailable. Second, the neurological investigation of the psychotic episode was clinically guided rather than exhaustive. Brain magnetic resonance imaging, electroencephalography, cerebrospinal fluid studies, and neuronal autoantibody testing were not performed, preventing the definitive exclusion of all potential neurological or immune-mediated causes of psychosis. Third, the available psychiatric history and follow-up were insufficient to establish the long-term diagnostic stability of the psychotic disorder. In a longitudinal study of 470 patients admitted for a first episode of psychosis, Bromet et al. found that approximately half experienced a diagnostic change at some point during the 10-year follow-up, particularly among those initially assigned less specific diagnoses [17]. These findings are relevant to the present case, in which a diagnosis of psychosis NOS was established at discharge, and reinforce the importance of longitudinal reassessment as additional information regarding symptom course and clinical evolution becomes available. Finally, a single case cannot establish a causal association between poliomyelitis and psychosis or determine the frequency of psychotic disorders among polio survivors.

The main clinical contribution of this report lies in illustrating the diagnostic and therapeutic challenges of new-onset psychosis in a patient with chronic neurological disability. A pre-existing neurological diagnosis should inform the differential diagnosis without being assumed to explain newly emerging psychiatric symptoms. Diagnostic conclusions should remain proportional to the available clinical evidence and investigations performed. At the same time, chronic disability and psychosocial vulnerability may substantially influence adherence, discharge planning, and functional outcomes, making coordinated psychiatric, medical, and community care particularly important.

Conclusions

This case highlights the importance of distinguishing the coexistence of neurological and psychiatric conditions from an established causal relationship. In patients with chronic neurological disability who develop psychosis, diagnostic assessment should remain grounded in the clinical phenomenology, relevant differential diagnoses, and longitudinal evolution, rather than attributing psychiatric symptoms to a pre-existing neurological condition. Furthermore, the interaction between individual vulnerability, psychosocial adversity, and functional limitations may influence both the expression of psychiatric symptoms and the challenges of clinical management, without necessarily establishing a causal relationship.

The particular relevance of this case lies in the convergence of diagnostic uncertainty, long-standing

physical disability, and limited social support. It illustrates how psychiatric care in such circumstances requires an individualized approach that extends beyond symptom control to include treatment continuity, functional needs, and community integration. These considerations reinforce the value of a comprehensive biopsychosocial assessment and coordinated multidisciplinary care, while acknowledging the limits of causal and diagnostic conclusions that can be drawn from an individual case.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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