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Functional Recovery in Late-Onset Schizophrenia: A Case Report

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Abstract: Schizophrenia is typically characterized by an early adulthood onset and a chronic, frequently deteriorating course. However, late-onset schizophrenia (LOS), which occurs after age 40, presents distinct clinical challenges, particularly regarding differential diagnosis with neurodegenerative and cerebrovascular disease and the potential for functional recovery. We report the case of a 60-year-old woman with a continuous late-onset psychotic illness, comprising an approximately 12-month prodrome and an active phase extending beyond six months, characterized by persecutory and mystical-religious delusions, auditory and visual hallucinations, and progressive social isolation. An extensive organic work-up (including magnetic resonance imaging and 18F-FDG PET/CT) showed only mild age-related white matter microangiopathy and preserved cortical metabolism and did not indicate a neurodegenerative or space-occupying process. After optimization of low-dose risperidone, the patient achieved symptomatic remission and returned to her premorbid functional baseline, assessed clinically and by informant report, over approximately 22 months of follow-up before being lost to follow-up. This report illustrates the diagnostic challenge at the LOS/very-late-onset schizophrenia-like psychosis (VLOSLP) boundary and emphasizes that favorable functional outcomes are achievable in late-onset schizophrenia with appropriate multidisciplinary management and pharmacological adherence.

Keywords: Late-Onset Schizophrenia; Psychosis; Functional Recovery; Differential Diagnosis; Case Report; Risperidone.

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1. Introduction

Schizophrenia encompasses a heterogeneous spectrum of symptoms, including positive and negative manifestations, cognitive deficits, and various mental health comorbidities. According to the DSM-5-TR, the diagnosis requires at least two characteristic symptoms present for a significant portion of a one-month active phase, with continuous signs of the disturbance persisting for at least six months, accompanied by impairment in social or occupational functioning and the exclusion of organic etiologies [1]. The clinical trajectory of the disorder is highly variable, influenced by age of onset, symptom stability, and multidisciplinary care. Although early-onset schizophrenia (EOS) is the traditional paradigm, a substantial proportion of patients do not follow the stereotyped pattern of gradual decline.

A significant knowledge gap remains regarding functional recovery in patients with late-onset schizophrenia (LOS), defined as onset between the ages of 40 and 60. Data suggest that only approximately 14% of patients achieve sustained recovery following a first episode of schizophrenia [2]. Recovery is defined by the absence of severe psychotic symptoms, minimal negative symptoms, and adequate role performance in social, occupational, or domestic spheres [2].

LOS cases frequently exhibit a female predominance and may require lower dosages of antipsychotics compared with EOS. However, when onset occurs near or after age 60,

the clinician faces a "diagnostic crossroads" between LOS and very-late-onset schizophrenia-like psychosis (VLOSLP), the latter carrying a higher risk of emergent neurocognitive impairment [3,4]. This case illustrates a rare trajectory of functional recovery in a 60-year-old patient, contributing to the limited literature on high-functioning outcomes in late-life psychoses.

Phenomenologically, late-onset psychoses differ in several respects from early-onset schizophrenia. Patients with LOS and VLOSLP more frequently present persecutory and partition delusions and hallucinations across multiple sensory modalities, whereas formal thought disorder, affective blunting, and negative symptoms are comparatively uncommon [5, 6]. VLOSLP may additionally follow a two-stage progression of the psychotic episode [6]. However, operational diagnostic criteria for these late-onset entities remain incompletely defined, which complicates clinical assessment and sustains diagnostic uncertainty at the boundary with neurodegenerative disease [5].

2. Case Report

A 60-year-old married woman presented to the Psychiatry Outpatient Clinic of a tertiary referral hospital in August 2022, accompanied by her husband. Her husband described an approximately 12-month prodrome of progressive social withdrawal, persecutory ideation, and behavioral disorganization: she had begun locking all doors and windows and controlling the movements of household members, disconnected the home's electrical system to keep out "bad energy," was frequently seen with a fixed, perplexed gaze, spoke and gestured as if conversing with unseen interlocutors, and had begun sleeping with the lights on. Over the three months preceding presentation these changes intensified, with prominent persecutory and mystical delusions, frequent soliloquies, and near-complete social isolation. Her husband reported no decreased need for sleep, impulsivity, irritability, or expansive mood. The patient had no prior history of anxiety or depressive episodes and no family history of psychiatric disorders.

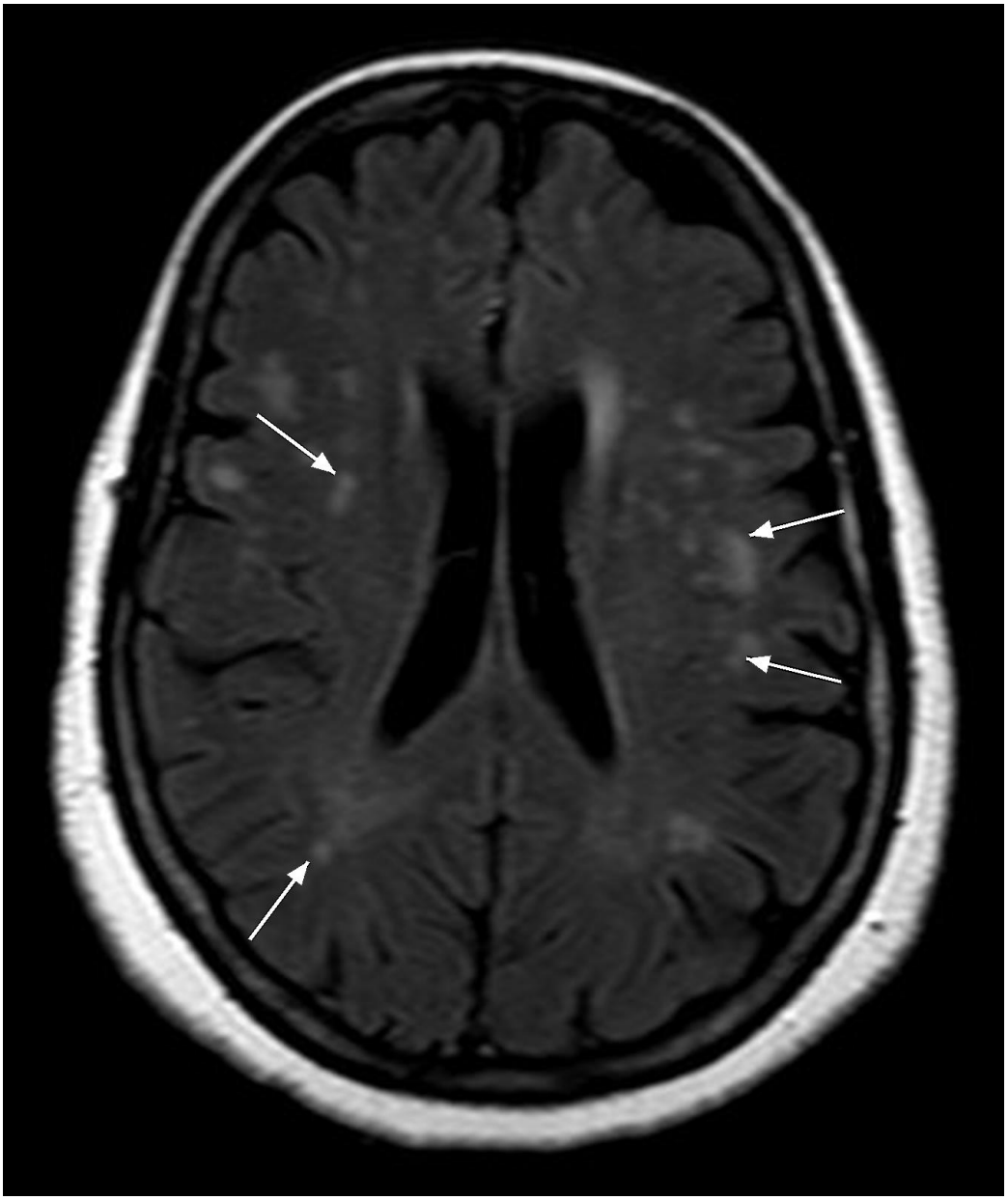
On direct assessment, the patient gave a retrospective account of a continuous psychotic process. From 2021 she described derealization and depersonalization ("I went to sleep and woke in a completely different reality"); from 2022, depreciative auditory hallucinations in familiar voices together with a conviction of inhabiting a parallel, "spiritual" world; and during 2023, complex and vivid visual hallucinations ("as if inside an image projector," seeing people and different eras throughout the day), mystical-religious delusions, and a belief in a lifelong spiritual gift affording simultaneous visions of past and future. She reported frequent tearfulness and a passive wish to stop living while explicitly denying suicidal ideation and denying sadness; no full manic or major depressive episode was identified at any point.

At the initial evaluation, the clinical hypothesis included a late-onset primary psychotic disorder versus an emergent neurocognitive syndrome. Investigations included cranial magnetic resonance imaging (MRI) (Figure 1) and comprehensive laboratory screening (metabolic and infectious) to exclude organic causes. Risperidone 0.5 mg/day was initiated, with only partial improvement; the patient subsequently discontinued the medication on her own after approximately ten days. MRI demonstrated only mild diffuse cortical volume reduction and mild periventricular and subcortical white matter microangiopathy, with symmetric hippocampi of normal volume and signal and no focal, expansive, or diffusion-restricting lesions, findings that did not indicate a neurodegenerative or structural etiology; laboratory results were unremarkable. Because the presentation was at that point of limited documented duration and had appeared to improve, a provisional impression of brief psychotic disorder (F23.0) was initially entertained, pending longitudinal characterization.

Off adequate pharmacological treatment, the psychotic symptoms persisted rather than remitting. Approximately fifteen months after the initial presentation, she re-presented with florid symptoms, mental confusion, visual and auditory hallucinations, and

complex mystical-religious and persecutory delusions, that had been continuously present for at least three months and persisted until pharmacological optimization, so that active psychotic symptoms spanned well beyond six months over the illness course. She exhibited significant loss of global functioning and somatic complaints related to visual acuity and tinnitus (both excluded by ophthalmological and otorhinolaryngological evaluation).

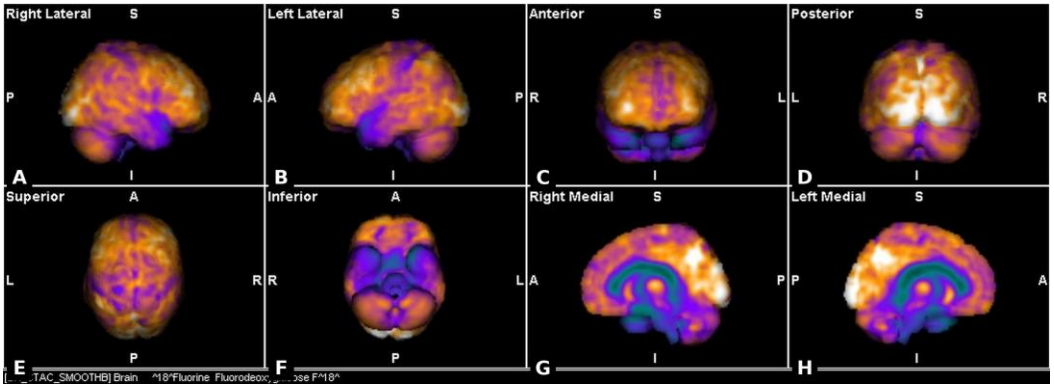
Figure 1. Representative axial fluid-attenuated inversion recovery (FLAIR) image from the cranial MRI performed at the initial evaluation. The study showed mild diffuse brain volume reduction, with accentuation of the cortical sulci and sylvian fissures, and scattered periventricular and subcortical white matter hyperintensities (arrows) consistent with mild microangiopathy. The hippocampi were symmetric, with normal volume and signal; the ventricular system and corpus callosum were preserved; and there were no focal parenchymal lesions, expansive processes, or areas of diffusion restriction.



A second, more extensive organic screening, including 18F-fluorodeoxyglucose positron emission tomography–computed tomography (PET/CT) (Figure 2) and repeat laboratory tests, yielded results within normal limits. Differential diagnoses considered were brief psychotic disorder (F23.0), late-onset schizophrenia (F20), schizoaffective disorder, psychotic disorder due to another medical condition (F06.2), and vascular (VLOSLP-spectrum) psychosis.

Considering the continuous course exceeding six months, characteristic active-phase symptoms, functional impairment, and the exclusion of a neurodegenerative or space-occupying process on the available investigation, the diagnosis was consolidated as late-onset schizophrenia. Risperidone was optimized to 2 mg/day, with remission of residual auditory hallucinations that had been recurring approximately every 15 days. With maintenance treatment and psychotherapeutic support, the patient progressively returned to her premorbid basic and instrumental activities of daily living, with no cognitive impairment observed or reported by the patient or her husband and no adverse effects. She was followed until June 2024, approximately 22 months after her first presentation, after which she was lost to follow-up despite documented attempts to re-establish contact.

Figure 2. Brain 18F-fluorodeoxyglucose (18F-FDG) PET/CT obtained at the relapse work-up, displayed as eight standardized three-dimensional surface projections of cortical glucose metabolism. Overall, the study shows preserved and symmetric cortical uptake, without the regional hypometabolic patterns typically associated with neurodegenerative disease. Panels: (A) right lateral view; (B) left lateral view; (C) anterior view; (D) posterior view; (E) superior view; (F) inferior view; (G) right medial view; and (H) left medial view. In each panel, the orientation labels indicate S = superior, I = inferior, A = anterior, P = posterior, R = right, and L = left.



3. Discussion

This case details a first psychotic episode at age 60, a period representing a critical diagnostic threshold. In late-life psychosis, the exclusion of primary organic etiologies (specifically neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease, and Lewy Body Dementia) is mandatory, as these conditions frequently present with hallucinations and delusions [3]. The absence of neurodegenerative or space-occupying abnormalities on advanced neuroimaging, such as PET-CT and MRI, is crucial for validating a primary psychiatric diagnosis in the sixth decade of life. In this patient, imaging revealed only mild diffuse volume reduction and mild white matter microangiopathy, nonspecific, largely age-related changes rather than a defined neurodegenerative pattern, while preserved hippocampal volume and signal, together with symmetric, normal cortical FDG metabolism, further argued against an underlying neurodegenerative process.

The observed clinical profile, female sex, onset at the upper limit of the LOS range, and high sensitivity to low doses of antipsychotics, is highly consistent with established literature on the late-onset schizophrenia subtype [4]. Patients with LOS frequently achieve remission with lower-than-standard doses of second-generation antipsychotics,

minimizing the risks of side effects. However, the age of onset (60 years) also places this patient at the transition to VLOSLP. The literature suggests that although these patients respond well to low doses of medication, there is a documented association with subsequent memory impairment over time [7]. Therefore, continuous long-term follow-up is essential, not only for symptomatic maintenance but also to monitor potential conversion to a neurodegenerative disorder.

Beyond phenomenology, converging evidence implicates age-specific neurobiological processes in late-life psychosis. Neuroimaging studies have associated VLOSLP with a cerebrovascular burden, including basal ganglia lacunar infarction and chronic white-matter small-vessel ischemic disease, capable of disrupting frontal–subcortical circuitry, while neuroinflammatory and neurodegenerative mechanisms have also been proposed [6]. Sensory impairment, comorbid physical illness, and adverse psychosocial circumstances such as social isolation are recognized predisposing factors [5,6]. Therapeutically, antipsychotic use in older adults demands caution owing to their heightened susceptibility to adverse effects, favoring the lowest effective dose combined with psychosocial and caregiver-oriented interventions [5]. In the present case, at age 60 the patient lies precisely at the LOS/VLOSLP boundary defined by the International Late-Onset Schizophrenia Group. The mild periventricular and subcortical white matter microangiopathy identified on MRI is acknowledged as a possible contributing risk factor rather than dismissed; however, its limited burden, the absence of lacunar infarction or confluent leukoaraiosis, the florid and systematized phenomenology, and the sustained response to a low dose of risperidone favor a primary late-onset schizophrenia over a predominantly vascular or prodromal-neurodegenerative psychosis.

Two further differentials warrant explicit consideration. First, affective and schizoaffective disorders were weighed, given the episodes of tearfulness and the passive death wish; however, no full manic or major depressive episode was identified, and psychotic symptoms were present in the absence of a concurrent prominent mood syndrome, so schizoaffective disorder was considered but not met. Second, given the acute "mental confusion" and sensory complaints at relapse, autoimmune encephalitis, including anti-NMDA-receptor encephalitis, which can present atypically in older adults, and a toxic-metabolic or encephalopathic process were considered. The protracted, approximately two-year course, the absence of seizures, movement disorder, dysautonomia, or depressed consciousness, and the sustained response to a low dose of antipsychotic without immunotherapy argue against a fulminant autoimmune or delirious process; nonetheless, cerebrospinal fluid analysis and electroencephalography were not performed, so these entities were excluded on clinical and imaging grounds rather than definitively.

A notable feature of this case is the functional recovery to the patient's premorbid baseline. Although schizophrenia is often viewed through the lens of chronic disability, this patient reflects the small subgroup, in the order of 14% of cases [2], that achieves substantial restoration of social and domestic role performance. This recovery was assessed clinically and through informant report rather than with a standardized functional instrument, and the observation window, although spanning roughly 22 months, ended with loss to follow-up; the outcome should therefore be interpreted with appropriate caution. It nonetheless suggests that late-onset cases, despite their diagnostic complexity, can carry a more favorable functional prognosis when metabolic, cerebrovascular, and neurodegenerative causes are reasonably excluded.

This report is subject to several limitations. As a single-case design, its findings are not generalizable, and no causal inference can be drawn. No standardized cognitive (e.g., MMSE, MoCA, or formal neuropsychological) or functional (e.g., GAF, PSP, WHODAS 2.0) instruments were administered: valid cognitive testing was precluded during the floridly psychotic initial presentations, and the assessment of recovery consequently rests on clinical and informant observation, so subtle executive dysfunction cannot be excluded. Electroencephalography and cerebrospinal fluid analysis were not performed, and autoimmune and other organic etiologies were therefore excluded on clinical and imaging

grounds rather than definitively. Part of the longitudinal history derives from the patient's retrospective account and spousal collateral. Finally, the subsequent loss of the patient to follow-up in June 2024 precludes firm conclusions regarding very-long-term functional outcome and the eventual risk of conversion to a neurodegenerative disorder.

4. Conclusion

Late-onset schizophrenia can, in selected cases, follow a favorable course culminating in symptomatic remission and functional recovery to the premorbid baseline. This case underscores the importance of a thorough organic, cerebrovascular, and neuroimaging work-up before consolidating a primary psychiatric diagnosis in later life, and of careful longitudinal characterization to satisfy diagnostic criteria at the LOS/VLOSLP boundary. It illustrates that meaningful functional outcomes are attainable with low-dose antipsychotic treatment and multidisciplinary follow-up, while the proximity to the very-late-onset spectrum and the mild microvascular findings make continued longitudinal monitoring for cognitive decline warranted.

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