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A Case of Early-Onset Alzheimer's Disease With Comorbid Schizophrenia: Diagnostic and Therapeutic Challenges

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Abstract

Early-onset Alzheimer's disease (EOAD) is defined as symptom onset before the age of 65. The disease frequently presents with prominent psychiatric features such as psychosis, behavioral disturbances, and personality changes, which can overlap with schizophrenia, creating a significant diagnostic challenge. The coexistence of EOAD and schizophrenia is rare, which can cause a delay in the recognition of the underlying neurocognitive disorder.

We present a 58-year-old female patient with EOAD and comorbid schizophrenia who had progressive cognitive decline, behavioral disturbances, hallucinations, and functional and memory impairment. Neuroimaging demonstrated significant cerebral atrophy, supporting the diagnosis of a neurodegenerative process in conjunction with the patient's clinical presentation. The patient is managed on maintenance therapy with paliperidone palmitate 156 mg monthly injection, donepezil 10 mg at night, benztropine 2 mg at night, and memantine 10 mg twice daily. Following the long-acting injectable antipsychotic therapy, medication adherence improved, and psychiatric symptoms remained relatively stable despite ongoing cognitive decline. The patient was seen at an outpatient clinic for four years, and longitudinal outpatient follow-up over this period required minor adjustments to the treatment regimen.

This case highlights the diagnostic complexity of EOAD presenting alongside schizophrenia, where the psychiatric manifestations may obscure the recognition of the underlying neurodegenerative disorder. It highlights the importance of maintaining EOAD in the differential diagnosis of patients with progressive cognitive decline, behavioral changes, and psychotic symptoms, particularly when functional impairment worsens over time. Furthermore, this case demonstrates the potential role of long-acting injectable antipsychotics combined with cognitive-enhancing therapies in promoting treatment adherence and maintaining psychiatric stability. This case contributes to the limited literature on EOAD coexisting with schizophrenia and the multidisciplinary longitudinal management and approach to optimize patient outcomes and preserve quality of life in complex neuropsychiatric conditions.

Categories: Neurology, Psychiatry
Keywords: adult neurology, clinical psychiatry, early-onset alzheimer's disease, neuroimaging differential diagnosis, schizophrenia

Introduction

Alzheimer's disease (AD) is the most common cause of neurocognitive disorders and is clinically characterized by impairments in memory, thought processes, and behavior. The severity of cognitive impairment generally correlates with disease progression. This chronic neurodegenerative disorder primarily affects individuals over the age of 65 years [1].

Histopathologically, AD is characterized by the extracellular deposition of neurotoxic β-amyloid and the accumulation of hyperphosphorylated tau protein, which form senile plaques and neurofibrillary tangles, respectively [1]. Definitive histopathological diagnosis traditionally relies on brain tissue examination through biopsy or autopsy. In clinical practice, diagnosis is based on clinical features and neuroimaging; however, these approaches have limited specificity in identifying the underlying cause of the disease [1,2].

Schizophrenia is a psychiatric disorder characterized by a broad spectrum of symptoms, including delusions, hallucinations, disorganized behavior, and negative symptoms such as flat affect, social withdrawal, and impairments in memory and cognition [3]. Its prevalence is relatively similar in males and females. However, the age of onset differs between sexes: males typically develop symptoms in their early 20s, whereas females generally experience symptom onset in their late 20s to early 30s [3].

There is considerable overlap between neurocognitive disorders and psychiatric illnesses, creating

diagnostic challenges in clinical practice. Early neurodegenerative disorders, including AD, may present with prominent psychiatric symptoms such as delusions, hallucinations, behavioral disturbances, apathy, and mood changes, which can resemble primary psychiatric illnesses [4]. AD is conventionally associated with non-bizarre delusions and visual hallucinations, whereas schizophrenia is more commonly associated with bizarre delusions and auditory hallucinations [4]. Schizophrenia is also associated with cognitive impairment and functional decline, potentially mimicking a neurocognitive disorder. However, cognitive changes in schizophrenia typically involve attention, executive function, and learning, and may remain relatively stable following the initial decline [4]. The substantial overlap in clinical manifestations makes it challenging to differentiate the two conditions, particularly when prominent cognitive impairment, functional decline, and psychotic symptoms are present [4]. Preliminary research has identified shared genetic signatures between AD and schizophrenia. Deficiencies in myocyte enhancer factor 2C (MEF2C), reported in both disorders, may represent a potential link between their overlapping clinical features, as reduced MEF2C activity can impair neurogenesis and synaptic development [4]. These shared clinical and genetic features further highlight the complexity of distinguishing between the two disorders and the importance of accurate differential diagnosis. Diagnostic ambiguity may delay appropriate treatment and management, potentially compromising clinical outcomes.

The coexistence of early-onset AD (EOAD) and schizophrenia presents additional challenges in diagnostic attribution and long-term management. Clinicians must consider whether psychotic symptoms arise from the neurocognitive disorder, a coexisting primary psychiatric illness, or overlapping disease processes, while balancing symptom control against potential medication-related adverse effects.

Here, we report a case of severe early-onset neurocognitive disorder in a patient with a clinical diagnosis of schizophrenia who was evaluated in an outpatient setting. This case highlights the atypical co-presentation of neurological decline and the overlapping clinical manifestations of these two disease processes.

Case Presentation

Clinical presentation

A 58-year-old woman with no significant past medical history was referred to the outpatient clinic for maintenance therapy for EOAD with comorbid schizophrenia. According to her husband, she had previously been fully functional but began gradually exhibiting psychiatric symptoms in February 2020, including delusions, hallucinations, unusual behaviors, and apathy. These symptoms persisted and progressively worsened over approximately eight months, eventually leading to her first psychiatric hospitalization for 11 days in November 2020. During the eight months preceding her hospitalization, her husband received several calls from the patient's workplace reporting unusual behavior. Her coworkers observed her standing in the workplace lobby, staring at the ceiling, and talking to herself. When approached, she stated that she was speaking with her brother and deceased mother. During this period, she became increasingly withdrawn and began forgetting important personal details and milestones. For example, she sometimes failed to acknowledge that she had two children, both in their 30s, and expressed the belief that another woman was "sabotaging" her and that she needed to return home to her three-month-old child. She had no history of infection, medication use, toxic exposure, or substance or alcohol abuse. In July 2020, the patient visited her primary care provider, who referred her to a psychiatrist. Initial investigations reportedly included CT and MRI scans, which were described in the patient's chart as showing normal cerebral volume. However, despite attempts by the authors to obtain the outside hospital records, the original imaging studies and complete radiology reports could not be retrieved for independent review. The patient's electroencephalogram (EEG) was also unremarkable. She was started on quetiapine 12.5 mg twice daily and was given a clinical diagnosis of schizophrenia. According to her husband, although her psychotic symptoms improved, her cognitive function continued to deteriorate rapidly.

At admission in November 2020, the patient was alert and oriented to person, place, and time but was unable to identify current events. During this period and throughout her hospitalization, her husband reported that she was increasingly forgetting her daily tasks at work and experiencing difficulty performing activities of daily living. She would leave the stove on, leave the water running in the hot tub, and leave the house doors open. An informal bedside cognitive assessment indicated significant cognitive impairment. She was able to follow simple commands but became increasingly irritable as the encounter progressed. A standardized Mini-Mental State Examination (MMSE) was not administered; therefore, a valid MMSE score could not be established. Neurological examination revealed no involuntary movements of the body or limbs and no significant cranial nerve abnormalities. The differential diagnoses included paraneoplastic or autoimmune encephalitis, subacute sclerosing panencephalitis, EOAD, and a primary psychiatric disorder.

The primary physician ordered laboratory investigations and diagnostic tests to help exclude paraneoplastic or autoimmune encephalitis and subacute sclerosing panencephalitis. Laboratory testing was negative for several infections, including human immunodeficiency virus (HIV), human T-lymphotropic virus types 1 and 2 (HTLV-I/II), syphilis, and John Cunningham virus (JC virus). Vitamin B1 and B12 levels were within normal limits. The patient had no history of alcohol use or abuse. Urine drug screening was negative for cocaine, cannabis, and benzodiazepines.

A CT scan of the head and brain revealed diffuse cerebral volume loss and old lacunar infarcts within the left lentiform nucleus. A CT scan of the abdomen showed gallbladder dilatation, which was considered unrelated to the patient's psychiatric presentation. A chest X-ray revealed no abnormalities. The patient was discharged on quetiapine (Seroquel) 50 mg.

The patient was hospitalized again in December 2020 because of worsening hallucinations and psychosis. This hospitalization lasted three weeks. During this admission, she exhibited a more negative and constricted affect and was significantly more withdrawn. A repeat CT scan revealed prominent global cerebral atrophy, as shown in Figure *1* and Figure *2*. Her cognitive decline was also marked: she was unable to state the date and repeatedly provided her date of birth instead. Based on the CT findings, clinical symptoms, and overall presentation, a clinical diagnosis of early-onset AD with comorbid schizophrenia was made. The patient was discharged on quetiapine 12.5 mg, aripiprazole 2 mg, memantine 5 mg, and haloperidol and lorazepam 2 mg as needed (PRN). This was her last hospitalization, after which she was referred for outpatient follow-up.

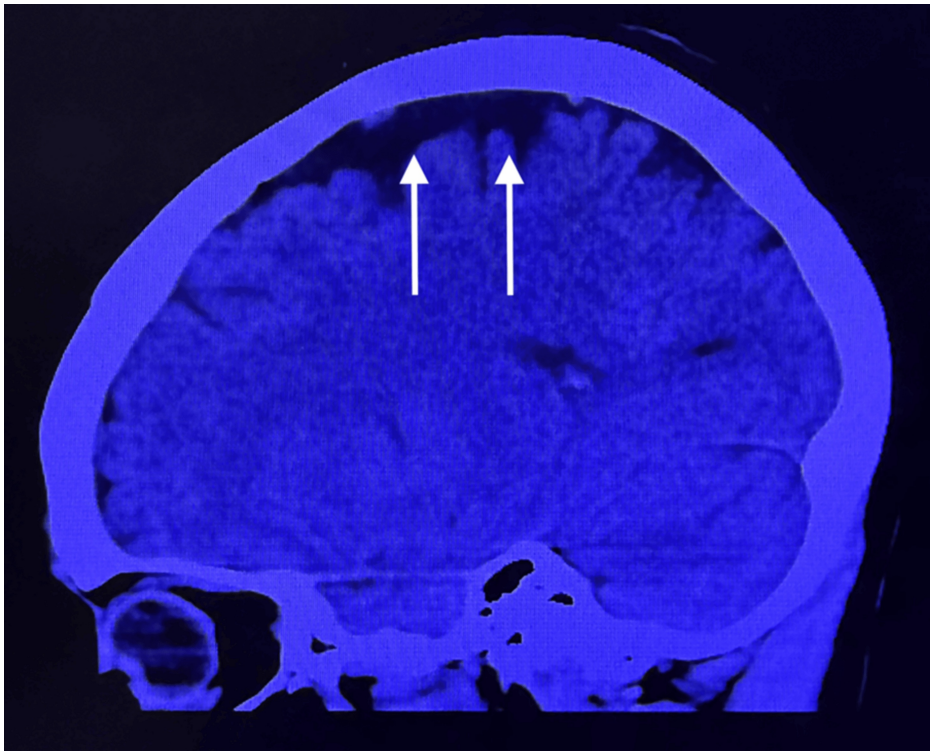


FIGURE 1: CT image showing a sagittal view of the patient's brain during her second hospitalization in December 2020, demonstrating cerebral atrophy.

The lateral ventricles and cortical sulci are prominent beyond what would be expected for the patient's age, consistent with global cerebral atrophy.

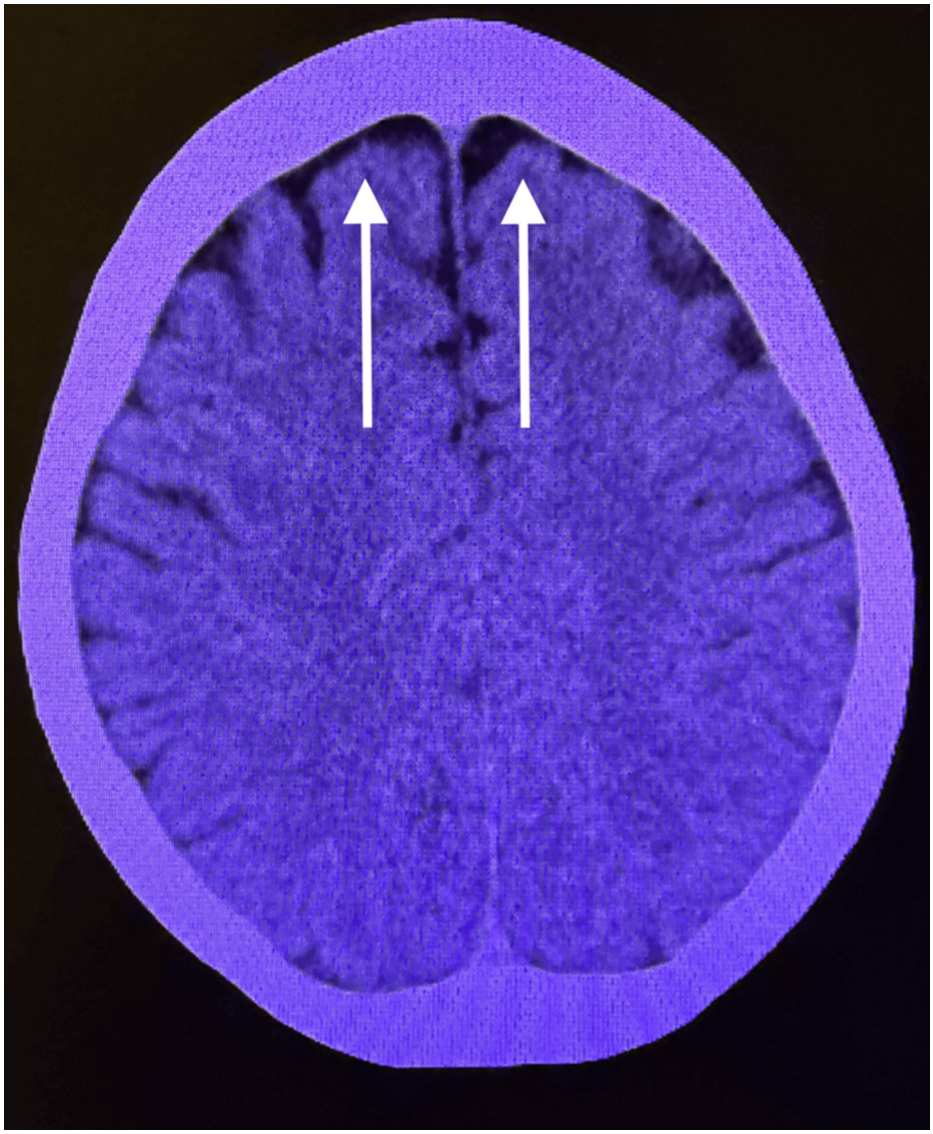


FIGURE 2: CT image showing an axial (transverse) view of the patient's brain during her second hospitalization in December 2020, demonstrating cerebral atrophy.

Since her last hospitalization, the patient has attended regular outpatient follow-up appointments with a psychiatrist. Her current medication regimen includes monthly paliperidone palmitate injections (156 mg), donepezil 10 mg, benztropine 2 mg, and memantine 10 mg. Her cognitive function remains unchanged from the time of her December 2020 hospitalization, and her affect remains restricted, with persistent social withdrawal. The current medication regimen, together with strict adherence to treatment, has reduced her hallucinations. She has remained clinically stable and has not required further hospitalization over the past four years.

Management

During the initial hospitalization in November 2020, the patient was treated with quetiapine 12.5 mg twice daily, in addition to lorazepam 1 mg three times daily as needed (PRN). Her condition stabilized following the administration of the antipsychotic medication and lorazepam. Upon discharge, she was prescribed quetiapine 25 mg and risperidone 3 mg, with instructions to continue the medications. However, she took them for only a few days before becoming noncompliant, resulting in a worsening of her symptoms, as reported by her husband. He stated that she refused to swallow or take the pills as instructed because she disliked how the medications made her feel. No medications for dementia or AD, such as acetylcholinesterase inhibitors, were prescribed during this initial hospitalization because the treating clinicians did not suspect an underlying neurocognitive disorder.

During the second hospitalization, the patient's pharmacological treatment included titration of quetiapine

to 200 mg at bedtime, aripiprazole 2 mg daily, and memantine 5 mg twice daily. Haloperidol and lorazepam 2 mg were also prescribed as needed (PRN). The hospitalization lasted three weeks and required frequent medication adjustments. Quetiapine was discontinued toward the end of the hospitalization because the patient was not taking it. Before discharge, the psychiatrist counseled the patient and her husband about the importance of medication adherence. She was discharged on memantine 5 mg twice daily and monthly intramuscular injections of paliperidone palmitate (Invega Sustenna) 156 mg. The inpatient psychiatrist referred her to an outpatient clinic for continued care.

The patient has received regular outpatient care over the past four years, with minor adjustments to her medication regimen. Her current medications include monthly paliperidone palmitate injections (156 mg), donepezil 10 mg at night, benztropine 2 mg at night, and memantine 10 mg twice daily. Benztropine was added to manage extrapyramidal symptoms associated with paliperidone palmitate.

Although the patient's overall cognitive function has not improved, she has remained clinically stable, with no reported hallucinations or further hospitalizations since December 2020. Her affect remains restricted, and she continues to exhibit social withdrawal. The overall benefit of the current medication regimen has been the maintenance of her clinical baseline, without significant further cognitive or functional deterioration.

Clinical outcome

In this patient, despite persistent and progressive cognitive impairment, long-term stabilization of psychotic and behavioral symptoms was achieved following the initiation of maintenance therapy with long-acting injectable paliperidone in combination with donepezil and memantine. This outcome was assessed based on clinical presentation and collateral information provided by the patient's husband. Following her second psychiatric hospitalization in December 2020, she experienced no further hospitalizations or episodes of acute psychiatric decompensation throughout the outpatient follow-up period. Hallucinations, disorganized behaviors, and agitation decreased substantially in frequency and severity, while medication adherence improved considerably with the use of long-acting injectable antipsychotic therapy. Both the patient and her husband preferred this treatment approach over oral medications.

Although the patient continued to exhibit severe cognitive impairment, restricted affect, psychomotor slowing, and limited verbal communication, consistent with the reported progression of EOAD, her overall clinical course remained stable relative to her established baseline. The primary therapeutic benefit was not the reversal of cognitive decline but rather sustained psychiatric stabilization, preservation of outpatient functioning, and prevention of recurrent hospitalizations. This case illustrates the diagnostic complexity of EOAD presenting with prominent psychotic symptoms that initially mimicked a primary psychiatric disorder. It emphasizes the importance of considering neurodegenerative disease in patients presenting with psychosis and progressive cognitive decline. In particular, this case highlights the potential role of long-term maintenance therapy with a long-acting injectable antipsychotic combined with cognitive-enhancing medications in controlling behavioral symptoms, reducing recurrent hospitalizations, and maintaining baseline functioning despite ongoing cognitive impairment.

Discussion

Background

AD, the most common neurocognitive disorder, affects more than 30 million individuals worldwide and is projected to triple in prevalence over the next 25 years [4]. Its underlying pathological features include the accumulation of neurotoxic β-amyloid and hyperphosphorylated tau, which damage synaptic pathways, contribute to neuronal cell death, and lead to progressive atrophy of brain structures [4]. AD primarily affects individuals over the age of 65 years, with approximately 95% of cases having a sporadic onset [4]. Overlapping clinical manifestations, including cognitive impairment, delusions, and hallucinations, may occur in both AD and schizophrenia [4].

Clinically, schizophrenia is more commonly associated with bizarre delusions and auditory hallucinations, whereas AD is more commonly associated with non-bizarre delusions and visual hallucinations [4]. Schizophrenia is the most common primary psychotic disorder and is associated with dysfunction in dopaminergic, serotonergic, and glutamatergic neurotransmission. Existing literature also indicates that prodromal features, including social withdrawal and unusual behaviors, may precede the onset of the first psychotic episode [3].

Physiology and pathophysiology

Schizophrenia is not yet fully understood; however, current literature suggests that its etiology is multifactorial and involves dysregulation of serotonergic, dopaminergic, and glutamatergic neurotransmission, resulting in alterations in neurotransmitter activity. Other theories implicate abnormalities in N-methyl-D-aspartate (NMDA) receptor function in the pathophysiology of schizophrenia [3]. Altered dopaminergic activity in the mesolimbic pathway is thought to contribute to the positive

symptoms of schizophrenia, including hallucinations, disorganized speech, and delusions. Conversely, reduced mesocortical dopaminergic activity has been associated with negative symptoms and cognitive deficits, including flat affect, social withdrawal, and impaired attention and memory [3]. Reduced glutamatergic neurotransmission, potentially related to NMDA receptor hypofunction, is also thought to contribute to the negative and cognitive symptoms of schizophrenia. Structural brain changes have also been observed, including enlargement of the third and lateral ventricles and reduced medial temporal lobe volumes [3].

AD is pathologically characterized by the extracellular deposition of neurotoxic β-amyloid and the intracellular accumulation of hyperphosphorylated tau, which aggregate to form senile plaques and neurofibrillary tangles, respectively. Their deposition in the medial temporal lobe and hippocampus is associated with reduced activation of these regions during memory-related tasks [2]. These degenerative processes contribute to synaptic dysfunction, progressive neuronal loss, and brain atrophy. β-amyloid-related pathological changes may begin before the formation of plaques and tangles and are associated with neuroinflammation and altered glial cell activation [2]. Apolipoprotein E (APOE) facilitates lipid transport and utilization, supporting neuronal structure and function, repair, and maintenance. Following neuronal axonal injury, residual nerve fibers undergo degenerative changes, while APOE-mediated lipid transport contributes to the processes involved in axonal and myelin repair [1]. The APOE ε4 allele is a major genetic risk factor for AD and is associated with increased β-amyloid accumulation, impaired neuronal repair, and accelerated neurodegeneration [1].

Comparative analysis

AD is classically characterized as a neurodegenerative disorder that typically manifests after the age of 65 years. Its clinical course is generally gradual and progressive, with prominent impairments in cognition and memory [4]. EOAD, generally defined as disease onset before the age of 65 years, differs in some respects from its late-onset counterpart. It may present with greater impairment in executive function, language, and visuospatial abilities. This clinical phenotype is associated with more pronounced disruption of psychosocial functioning [5].

In the context of this case, psychosocial decline may contribute to diagnostic ambiguity because it overlaps with, and may be masked by, the social and functional impairments commonly observed in schizophrenia. The age at symptom onset is also atypical relative to the usual epidemiological distributions of both AD and schizophrenia. In this case, the initial manifestations included memory decline, repetitive speech, and psychomotor agitation characterized by continuous pacing. Consistent with the presentation of EOAD, the patient experienced a marked decline in cognition and executive function. Potential links between schizophrenia and AD have been suggested by studies reporting an increased occurrence of AD among individuals with schizophrenia [6]. Furthermore, approximately 30% of individuals diagnosed with AD may develop psychotic symptoms, including delusions and hallucinations, as the disease progresses [6]. The patient exhibited visual hallucinations involving deceased relatives and a somatic delusion of pregnancy. These psychotic manifestations further complicated the diagnostic assessment in the context of suspected EOAD [6].

What we learned from this case

The absence of definitive laboratory or neuroimaging markers for confirmation limits the diagnosis of schizophrenia. However, hallucinations, delusions, social withdrawal, and apathy were prominent clinical features that supported the diagnosis. These symptoms were also controlled with quetiapine during the initial periods of hospitalization, followed by risperidone and paliperidone palmitate. In this patient, the diagnosis of EOAD was supported by clinical criteria, including memory impairment and executive function decline. It was further supported by neuroimaging findings demonstrating marked global cerebral atrophy [7]. The use of imaging in EOAD is important for excluding alternative diagnoses, including other neurocognitive disorders, subdural hematomas, brain tumors, and normal pressure hydrocephalus. Cortical atrophy due to neuronal loss is a common neuroimaging finding in AD and may increase the certainty of a clinical diagnosis when supplemented by additional diagnostic criteria [7].

Additional tests commonly utilized in EOAD include cerebrospinal fluid assays and genetic testing. The collateral information provided by the patient's partner offered greater insight into the onset and progression of the patient's cognitive decline and psychiatric presentation. The pharmacological management of EOAD is similar to that of classic AD, utilizing acetylcholinesterase inhibitors and memantine to improve memory-related symptoms. For non-memory-related symptoms, the use of speech and occupational therapies can improve cognitive deficits associated with language impairment and ideomotor apraxia [8]. In this patient, a regimen of memantine and donepezil has improved her cognitive function. Improvements in speech and language comprehension have been observed, with the patient increasingly able to respond appropriately to prompts during appointments. In combination with treatment for cognitive symptoms, long-acting intramuscular paliperidone palmitate (Invega Sustenna) 156 mg/mL, administered at 28-day intervals, has been effective in reducing her schizophrenia symptoms, including hallucinations and delusions. Continuing therapies in conjunction with medication is essential to the management of EOAD, as the decline in cognitive function influences morbidity and mortality [9].

As the patient continues to show improvement in the management of both EOAD and schizophrenia, the outlook for her physical and psychosocial functioning is progressively favorable [10]. Long-term, multidisciplinary management and therapies are indicated throughout the patient's lifetime to manage symptoms and delay disease progression.

Conclusions

This case describes a patient with clinical diagnoses of EOAD and schizophrenia who received long-term maintenance treatment with paliperidone palmitate injection, donepezil, benztropine, and memantine. The patient's clinical presentation, characterized by hallucinations, delusions, memory impairment, progressive cognitive decline, and functional deterioration, together with CT findings of prominent global cerebral atrophy, supported the clinical diagnoses of EOAD and a coexisting psychiatric illness. This case highlights the diagnostic challenges associated with EOAD presenting with prominent psychiatric manifestations and emphasizes the importance of considering an underlying neurodegenerative disorder in patients with psychiatric symptoms accompanied by progressive cognitive and functional decline. Neuroimaging and clinical assessment contributed to the diagnostic evaluation, although the absence of standardized cognitive testing and access to the initial imaging studies limited further characterization of the disease course.

Over four years of outpatient follow-up, the patient experienced no further psychiatric hospitalizations, and hallucinations and behavioral disturbances decreased substantially during maintenance treatment with long-acting injectable antipsychotic therapy and cognitive-enhancing medications, despite persistent cognitive impairment. Improved medication adherence following the transition to long-acting injectable paliperidone palmitate was also reported. Although the contribution of each individual medication cannot be determined, this clinical course illustrates the potential value of sustained pharmacological management and medication adherence in reducing psychiatric relapse and recurrent hospitalization in patients with complex neuropsychiatric presentations. Overall, this report emphasizes the importance of longitudinal, multidisciplinary management, including psychiatric treatment, ongoing cognitive and functional assessment, caregiver involvement, and regular outpatient follow-up, to address the complex needs of patients with EOAD and schizophrenia.

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.

Concept and design: Atharv Joshi, Jenna Meadows, Adem Ahsan, Jonathan Hanna, Elizabeth Mirkin

Acquisition, analysis, or interpretation of data: Atharv Joshi, Jenna Meadows, Adem Ahsan, Jonathan Hanna, Elizabeth Mirkin

Drafting of the manuscript: Atharv Joshi, Jenna Meadows, Adem Ahsan, Jonathan Hanna, Elizabeth Mirkin

Critical review of the manuscript for important intellectual content: Atharv Joshi, Jenna Meadows, Adem Ahsan, Jonathan Hanna, Elizabeth Mirkin

Supervision: Atharv Joshi

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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