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CASE REPORT

Early-Onset Major Neurocognitive Disorder Presenting as Schizophrenia-Like Psychosis: A Case Report

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ABSTRACT

Early-onset neurocognitive disorders may initially present predominantly with psychiatric symptoms and can therefore be mistaken for primary psychotic disorders. A 42-year-old man with no previous psychiatric illness or history of substance use presented with an abrupt onset of recurrent formed visual hallucinations, consisting of seeing a snake emerging from a hole in the wall and subsequently moving along the walls of his house. He developed persecutory beliefs that someone had deliberately placed the snake to harm him, accompanied by hypervigilance, severe sleep disturbance, irritability, aggression toward family members, social withdrawal, and reduced appetite. Three days before admission, he wandered away from home and was subsequently found in a dishevelled and perplexed state, unable to recall where he had been. Schizophrenia was initially considered. However, mental status examination demonstrated impaired attention, recent and remote memory, abstract thinking, insight, and judgement. His Mini-Mental State Examination score was 12/30. Routine investigations, including hepatic, renal, thyroid and vitamin B12 assessments, were within normal limits. Magnetic resonance imaging of the brain demonstrated bilateral periventricular and deep white-matter hyperintensities consistent with chronic ischemic changes, together with cerebral volume loss and frontal-predominant atrophy. Following neurological assessment, the diagnosis was revised to early-onset major neurocognitive disorder with psychotic symptoms, with a possible vascular contribution. The case illustrates how early-onset neurocognitive disorders may present to psychiatric services with prominent symptoms of psychosis. Marked cognitive impairment, disorientation, and structural abnormalities on neuroimaging should prompt consideration of an underlying neurological disorder rather than assuming a primary schizophrenia-spectrum illness.

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KEYWORDS

• Early-onset neurocognitive disorder • Psychosis • Visual hallucinations • Frontal atrophy • Cognitive impairment • First-episode psychosis • Neuropsychiatry

INTRODUCTION

Early-onset dementia (EOD) is defined as dementia with onset before 65 years and accounts for roughly 5–10% of all dementias.[1] Unlike many late-life dementias, early-onset disorders may initially present with behavioural, psychiatric, language, or executive symptoms rather than an obvious memory-predominant syndrome. Consequently, patients may first present to psychiatric services and receive a diagnosis of schizophrenia, mood disorder, or another primary psychiatric condition. The differentiation is clinically important because the prognosis, therapeutic approach, family counselling, safety planning, and long-term care requirements differ substantially between a primary psychotic disorder and a neurocognitive disorder. Atypical age of onset, rapid deterioration, prominent cognitive impairment, neurological abnormalities, visual hallucinations, and an unusual course should increase suspicion of an underlying neurological disorder. We report the case of a 42-year-old man who presented with prominent formed visual hallucinations and persecutory delusions and was initially diagnosed with schizophrenia. Subsequent assessment revealed substantial cognitive impairment, while magnetic resonance imaging (MRI) demonstrated cerebral volume loss with frontal-predominant atrophy and chronic ischemic white-matter changes.

CASE REPORT

A 42-year-old unmarried man was brought to the psychiatry outpatient department by family members because of a relatively abrupt change in behaviour characterized by visual hallucinations, suspiciousness, fearfulness, disturbed sleep, aggression, and progressive functional deterioration. About two months before hospitalization, the patient began repeatedly reporting that he could see a snake emerging from a hole in a wall in his house. The visual experience persisted despite reassurance from family members and repeated inspection of the area. He subsequently began reporting that the snake was moving along the walls and became increasingly convinced that someone had deliberately placed it in the house to kill him. His fear progressively intensified. He became hypervigilant, particularly at night, and remained awake for prolonged periods in an attempt to monitor the perceived snake. He became increasingly irritable toward family members and on several occasions attempted to attack them, believing that they had deliberately placed the snake in the house. Over approximately one month, he developed reduced sleep and appetite, increasing social withdrawal, and deterioration in his usual behaviour.

Approximately three days before admission, he wandered away from home and was subsequently found in a dishevelled and perplexed state. When questioned, he was unable to recall where he had been or provide a coherent account of his whereabouts. His family subsequently brought him to the psychiatry outpatient department, from where he was admitted for further evaluation. There was no previous history of psychiatric illness, substance use, head injury, or similar episodes. There was no family history of psychiatric illness. Before the onset of the current illness, he was an introverted but responsible and caring individual who maintained cordial relationships with family members and peers. He was educated up to the 12th standard in a Hindi-medium school, with average academic performance, and subsequently worked as a carpenter. He was religious and enjoyed watching cricket and movies. General and systemic examination were within normal limits. On mental status examination, the patient was conscious but appeared perplexed. He had persecutory delusions concerning the alleged snake and reported recurrent formed visual hallucinations of snakes. Attention was poorly sustained, with a forward digit span of three and a backward digit span of two; serial subtraction was impaired. Recent and remote memory were impaired, and abstract thinking was poor. Insight was severely impaired, with a score of 1/5, and judgement was impaired in both personal and social domains. In view of the prominent persecutory delusions and hallucinations, a provisional diagnosis of schizophrenia was made at admission. A Mini-

Mental State Examination (MMSE) yielded a score of 12/30, indicating substantial global cognitive impairment. Routine laboratory investigations, including complete blood count, liver function, blood sugar (Fasting & postprandial), HIV & syphilis serology, renal function, thyroid function, and vitamin B12 levels, were within normal limits. MRI of the brain demonstrated multiple T2/FLAIR hyperintense areas involving the bilateral periventricular white matter, corona radiata, and centrum semiovale. These lesions were isointense on T1-weighted images and showed no diffusion restriction, consistent with chronic ischemic white-matter changes. The ventricular system and cortical sulci were dilated, with prominent frontal-predominant cerebral volume loss. The imaging findings were interpreted as demonstrating cerebral volume loss with frontal-predominant atrophy and chronic ischemic white-matter changes. The imaging findings were not considered diagnostic of a specific neurodegenerative disorder in isolation.

The combination of abrupt-onset psychotic symptoms, recurrent formed visual hallucinations, marked cognitive impairment, impaired attention and memory, impaired judgement, wandering with disorientation, and structural abnormalities on MRI raised significant concern for an underlying neurological or neurocognitive disorder. Following neurological consultation, the working diagnosis was revised from a primary psychotic disorder to early-onset major neurocognitive disorder with psychotic symptoms, with a possible vascular contribution based on the presence of chronic ischemic white-matter changes.

The patient was jointly managed by the psychiatry and neurology teams. Olanzapine was initiated at 2.5 mg at bedtime and subsequently increased to 5 mg at bedtime and Donepazil 5 mg/day. The family received counselling regarding the nature of the illness, the need for supervision and safety measures, possible progression of cognitive and behavioural symptoms, prognosis, and caregiver support. However, after discharge the patient was lost to follow up.

DISCUSSION

The present case illustrates the diagnostic difficulty posed by new-onset psychosis in a relatively young adult when psychiatric symptoms are accompanied by prominent cognitive impairment and structural brain abnormalities. The patient initially appeared to have a primary psychotic disorder because of persecutory delusions, hallucinations, behavioural disturbance, aggression, and sleep disruption. However, several features were atypical for a straightforward diagnosis of schizophrenia, including the patient's age at first presentation, abrupt symptom onset, prominent formed visual hallucinations, marked cognitive impairment, perplexity, impaired recent and remote memory, impaired judgement, and wandering with subsequent inability to recall his whereabouts.

Early-onset neurocognitive disorders are particularly prone to initial psychiatric misdiagnosis because behavioural and psychiatric symptoms may dominate the clinical picture. A recent review specifically emphasized that young patients with neurodegenerative disorders may initially be diagnosed with schizophrenia or another psychiatric disorder and recommended careful attention to atypical symptom patterns, cognitive decline, neurological findings, collateral history, neuropsychological assessment, and neuroimaging.

Psychotic symptoms may occur in several neurodegenerative disorders. Their phenomenology and prevalence vary according to the underlying pathology. In FTD, psychosis has traditionally been regarded as relatively uncommon compared with behavioural disinhibition, apathy, loss of empathy, compulsive behaviour, and executive dysfunction. Previous studies suggest that up to half of FTD patients are initially misdiagnosed with psychiatric disorders. Psychosis occurs in about 10–20% of FTD cases, especially with right-frontal or temporal involvement. However, more recent studies indicate that psychosis may be substantially more frequent in particular genetic and pathological subgroups. A systematic review of genetically or pathologically confirmed FTD found psychosis in approximately one-quarter of cases, with the highest frequency among C9orf72 mutation carriers. Delusions were particularly common among C9orf72 carriers, whereas visual hallucinations were relatively prominent among patients with GRN mutations.[2,3]

The present patient had several features that raised the possibility of a frontal-predominant neurodegenerative process, particularly his impaired judgement, behavioural dysregulation, aggression, poor insight, and frontal-predominant cerebral atrophy. However, he did not have a sufficiently documented syndrome to establish probable bvFTD with confidence. The revised bvFTD criteria require a characteristic combination of behavioural and cognitive features, while probable bvFTD additionally requires functional decline and characteristic neuroimaging.[4,5,6] Therefore, it would be inappropriate to label the present case definitively as FTD solely on the basis of frontal atrophy.

One of the most striking features of the case was the presence of recurrent, formed visual hallucinations involving snakes. The hallucinations were sufficiently convincing to the patient that he repeatedly searched for the snake, remained vigilant throughout the night, and developed a persecutory explanation for its presence. Formed visual hallucinations are an important diagnostic clue in neuropsychiatry. They may occur in several neurological and psychiatric conditions and are particularly characteristic of DLB. The revised DLB diagnostic criteria recognize recurrent visual hallucinations as one of four core clinical features, alongside fluctuating cognition, spontaneous parkinsonism, and REM sleep behaviour disorder, which were not evident in our patient.[7,8] Thus, the visual hallucinations alone do not establish DLB.

The initial diagnosis of schizophrenia was understandable given the presence of delusions, hallucinations, aggression, social withdrawal, and behavioural disturbance. However, several clinical features argued against an uncomplicated primary schizophrenia-spectrum disorder. First, the onset occurred at 42 years of age without a previous psychiatric history. Second, the hallucinations were predominantly formed visual phenomena rather than the auditory hallucinations more commonly encountered in schizophrenia. Third, the patient demonstrated substantial impairment across multiple cognitive domains, including attention, memory, abstraction, and judgement. Fourth, he developed perplexity and wandering with inability to recall his whereabouts. Finally, MRI demonstrated significant structural abnormalities. These findings collectively increased the probability of an underlying neurological disorder. The distinction is clinically important because misdiagnosis of neurodegenerative disease as a primary psychiatric disorder can delay appropriate neurological assessment, family counselling, safety planning, and anticipatory care.

The immediate clinical priority in the management of this case was reduction of psychotic symptoms, aggression, and behavioural risk. Olanzapine was therefore initiated at a low dose and gradually increased to 5 mg/day. The decision to prescribe donepezil was made because of the significant cognitive impairment and the possibility of a mixed neurodegenerative/vascular process. Antipsychotic treatment in patients with neurocognitive disorders requires caution because of the potential for sedation, extrapyramidal effects, metabolic complications, cerebrovascular adverse events, and worsening functional impairment. Treatment should therefore generally use the lowest effective dose with regular reassessment of benefit and adverse effects.[9]

CONCLUSION

The case underscores that new-onset psychosis in midlife, particularly when accompanied by visual hallucinations and objective cognitive impairment, should prompt consideration of an underlying neurological or neurocognitive disorder.

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