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LATE ONSET PSYCHOSIS – A CASE REPORT

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CASE REPORT **OPEN ACCESS**

ABSTRACT

Introduction – Late-onset psychosis is a disorder that is well-known but poorly understood, and it has emerged as an increasingly significant issue in geriatric patients. There is no clear information about whether this psychosis occurs for the first time at this age. Despite being underdiagnosed, this late-onset psychosis responds well to treatment. A bizarre, persecutory delusion is a hallmark symptom of late-onset schizophrenia.
Methods – Presented the case of a 67-year-old woman who visited the Emergency Psychiatry Department complaining of persecutory thoughts, auditory hallucinations, and mild cognitive impairment.
Results – This case report explores the diagnostic process and treatment options for very late-onset schizophrenia-like psychosis, including non-pharmacological and pharmacological approaches.
Discuss – Psychotic symptoms can be caused by a variety of general medical conditions in elderly patients. It is necessary to rule out delirium, dementia, substance-related disorders, delusional disorder, and dissociative disorder before diagnosing Late-onset psychosis. A late-onset psychosis is not only challenging to diagnose but also to treat. Non-pharmacological treatments are the first option in managing late-life psychosis. It includes psycho-education for patients and their families, as well as cognitive behavior therapy (CBT). It is important to monitor for adverse reactions while administering medication, and the lowest dosage should be used to achieve short-term efficacy. In addition to antipsychotic medication, anticholinesterase inhibitors may also be effective in treating these patients.
Conclusion: A careful observation and approach are required to make an etiological diagnosis of late-onset psychosis since there are no pathognomonic signs or symptoms. To achieve the best results, their efficacy and side effects should be monitored with regularity.

Keywords: late-onset psychosis, dementia, schizophrenia.

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INTRODUCTION

Psychiatric disorders encountered at any age, and especially in later life, require careful evaluation to rule out organic pathologies. Late-onset psychosis is commonly known as "very late-onset schizophrenia-like psychosis," according to the International Late-Onset Schizophrenia Group. It occurs after the age of 60 years. Persecutory delusions are common and often elaborate, thus called "partition" delusions. In addition, hallucinations are also prevalent and can be experienced in multiple modes, such as auditory, visual, and olfactory.[1,2] Delusions and hallucinations can often be linked, and other symptoms like disorganization, negative symptoms, and thought disorder, are rarely observed. There is a frequent occurrence of cognitive impairment. A diagnosis of late-onset psychosis is not included in the current psychiatric diagnostic system such as the Diagnostic and Statistical Manual of Mental Disorders-V, International Classification of Diseases-10. However, despite exclusion from the diagnostic criteria, a consistent clinical picture has been observed. In addition, it is unclear whether late-onset psychosis differs from schizophrenia and whether it might be a sign of dementia.
There are a variety of biological, psychological, social, and environmental factors that play a role in the development of psychotic disorders in late life. There are higher rates of morbidity and mortality associated with psychosis among older adults than among younger adults.[3] There are various terms used to describe primary late-onset psychosis. Among these are paranoia, pre-senile delusional insanity, late-onset schizophrenia, late paraphrenia, delusional disorder, paranoid psychosis, and very late-onset schizophrenia-like psychoses.[4,5] There is an estimated 1 percent prevalence of late paraphrenia in general population studies.[6] An organic factor may play a significant role in late-onset psychosis, so it is important to rule out cognitive impairments or dementias before concluding that such a diagnosis is necessary. Researchers have suggested that sensory impairments, especially hearing loss, maybe a contributing factor to late-onset psychosis. In older adults, cognitive decline is a risk factor for psychotic disorders. There is also evidence that brain structural abnormalities contributed

to this progressive cognitive decline. There are other risk factors associated with late-onset psychosis, including poor health status, sensory deficits, premorbid personality disorder, neuropsychological abnormalities, female gender, and visual impairment, and negative life events which seem to be more common in females with sensory deficits, social isolation, and with a family history of schizophrenia.[7,8] In addition, older onset age was associated with lower rates of substance abuse, better premorbid psychosocial functioning, and higher school achievement. Psychological burdens such as unemployment were associated with late-life schizophrenia.[9]

The presence of neuroimaging findings associated with schizophrenia has been consistently observed in patients with early-onset, late-onset, and very late-onset schizophrenia. As a result of these findings, there is volumetric atrophy, white matter hyper-intensity, and poor functional connectivity between the frontal and temporoparietal lobes. As well, the hippocampal and amygdala volumes were smaller. Furthermore, a decrease in the hippocampal/amygdala ratio was correlated with the age of schizophrenia onset, indicating a neurodevelopmental cause. Late-onset schizophrenia is associated with larger thalamic volumes, more atrophy in the cerebellum, and a decrease in corpus callosum volume, according to some researchers.[10,11]

A thorough history is taken from the patient suffering from psychotic symptoms to diagnose the condition. To gather information about overall physical health and possible neurodegenerative changes, a physical examination, including neurological examinations and cognitive assessments, is necessary. Laboratory tests and brain imaging, such as magnetic resonance imaging (MRI) and computerized tomography (CT), can detect focal structural abnormalities in cerebral lesions. To differentiate diagnosis, complete blood counts, liver function, renal function, electrolyte imbalances, metabolic panels, thyroid function tests, vitamin B12, folic acid concentrations, urine toxicology, and infectious disease markers such as syphilis, HIV panels, and autoimmune panels are helpful. Additionally, psychotic symptoms can be diagnosed based on neuropsychological tests that determine cognitive changes in individuals.

Non-pharmacological treatments are the first option in managing late-life psychosis. It includes psycho-education for patients and their families, as well as cognitive behavior therapy (CBT). It is known that CBT improves both positive and negative symptoms. It also has a positive impact on treatment compliance, but there is little evidence that it improves psychotic symptoms in older people.[12] There are other approaches to achieve clinical efficacy, such as functional adaptation skills training, cognitive remediation therapy, family interventional therapy, self-management training, lifestyle interventions, and family psycho-education.[13] Adherence can be improved by providing patients and their families with thorough psycho-education. It has been suggested that antipsychotics should be prescribed cautiously for a short time. This is because adverse effects such as extrapyramidal and anticholinergic effects and drug interactions can result in significant morbidity in older adults. Also, possible side effects should be observed closely. Clinically, anticholinesterase agents, such as donepezil and rivastigmine, offer a second pharmacological option to clinicians managing a patient with late-onset psychosis because cognitive decline is often associated with psychotic symptoms.

METHOD

Presented the case of a 67-year-old woman who visited the Emergency Psychiatry Department complaining of persecutory thoughts, auditory hallucinations and mild cognitive impairment. This case report was reported with the approval and permitted by the Head of Clinical Care Department, Erada & Mental Health Complex - Taif, 21944, Saudi Arabia, the patient, and her family.

CASE REPORT

The patient was a 65-year-old Saudi woman who was educated, but unemployed, and had no history of mental illness until she was 64 years old. She presented to the emergency department with symptoms of suspiciousness, explained that she was being monitored by various electronic devices, including televisions and cameras, and heard voices talking about her. Her husband passed away due to illness 10 years ago, so she had to live alone. She complained about loneliness and said that her sons had stopped supporting her. She has persecutory ideas about neighbors who said they might be stealing her house. During the mental status examination, she is fearful with a flat effect. She also reported ideas of reference, persecutory delusions, auditory hallucinations, and mild cognitive impairment. She denied suicidal and homicidal ideation. No history suggested of psychiatric illness in her family or substance abuse. She has not heard properly for three years and has impaired vision due to her old age. She is isolated from other people, prefers to be alone, and has mild cognitive impairment. In recent months, her neighbor said she has been having paranoid traits like being suspicious of us, but now she says we will steal her house.

She has a history of hypertension and dyslipidemia, taking bisoprolol 5 mg once daily with atorvastatin 10 mg once daily. Currently, she is stable on her medications. She was referred in the past to neurologists due to her cognitive decline. She started on donepezil 10 mg once daily, but she has not been taking the medication. She had no history of cigarette smoking or other illicit substance abuse. A general physical examination was reported to be within normal limits. On neurological examination, she had postural and intention tremors, and mild cognitive impairment. She had mild visual or hearing impairment due to senility. Routine laboratory tests, such as liver and kidney function tests, complete blood cell count, vitamin B12 level, thyroid function test, and serology were within normal limits. However, her cholesterol level is slightly higher as she suffers from dyslipidemia and is on irregular medication. A magnetic resonance imaging study revealed mild diffuse brain atrophy at the frontal lobes in the patient, are shown in Figure 1. In concern of her non-cooperation and inattention, she did not undergo neuropsychological testing, but a mini mental status examination revealed mild cognitive impairment.

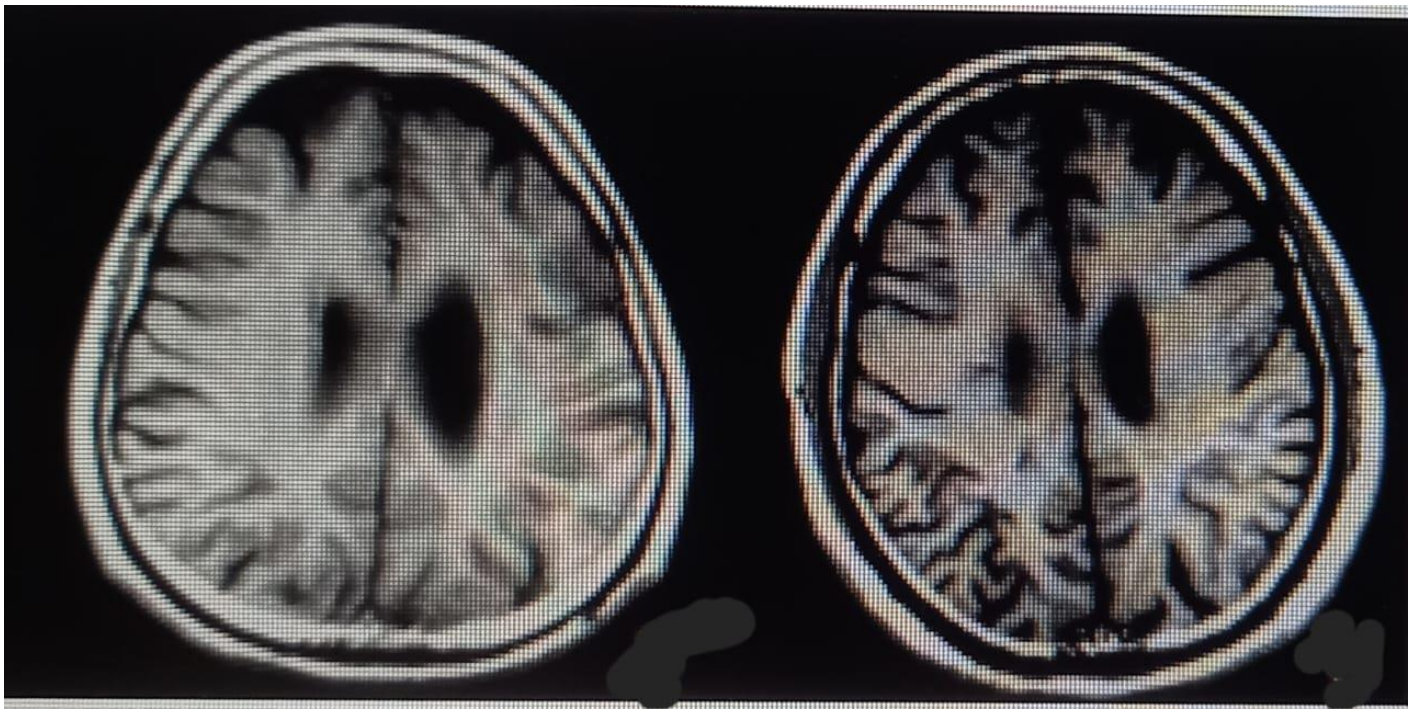

Figure 1. A magnetic resonance imaging study revealed mild diffuse brain atrophy at the frontal lobes in a patient with late-onset psychosis.

RESULT

Based on her psychotic symptoms and test results, she was diagnosed with late-onset schizophrenia. The patient and her family were referred to a psychologist and social worker for cognitive behavior therapy. We found that it was effective at improving positive symptoms, and it had a positive impact on treatment compliance. She was also given functional adaptive skills training, cognitive remediation therapy, family interventional therapy, self-management training, lifestyle interventions, and family psycho-education. Along with psychotherapies, we treated her with haloperidol 1.5 mg once daily. She tolerated it well, and the dose was titrated to 3 mg per day. On her first follow-up after two weeks, her perceptual disturbance was reduced, and later, after 3 months her other symptoms almost disappeared.

Follow-up in late-onset psychosis requires careful intervention due to the higher risk of secondary psychosis, higher morbidity and mortality rates, and complicated treatment considerations due to the higher incidence of adverse effects. To detect clinical phenotypes such as hallucinations (visual, olfactory, or tactile) and delusions (persecutory or partitioned), it is crucial to closely observe for possible side effects. It is also necessary to continue psychoeducation of both the patient and the family members during follow-up to increase adherence. Her psychotic symptoms almost disappeared after 3 months of follow-up period.

DISCUSS

Psychotic symptoms can be caused by a variety of general medical conditions in elderly patients. In elderly patients, it is necessary to rule out delirium, dementia, substance-related disorders, delusional disorders, and dissociative disorders before diagnosing schizophrenia. Despite many similarities with early-onset schizophrenia, late-onset schizophrenia also exhibits some differences.[14] Risk factors for late-onset psychosis include sensory deficits, premorbid personality disorder, social isolation, neuropsychological abnormalities, and female gender. Our patient is a female with paranoid traits such as being suspicious of her neighbors. She stays aloof, detached, and socially isolated, as well as having mild cognitive impairments due to aging. She has also suffered a hearing defect and slight vision loss.

Several evaluations, including history, physical examination, and laboratory tests, were conducted to rule out specific diagnoses. The differential diagnosis of Late-Onset Psychosis is a psychotic disorder due to a general medical condition, dementia-related syndromes with psychosis, delirium, psychosis secondary to substance abuse/dependence, delusional disorder, and psychosis not otherwise specified. Based on her psychotic symptoms and test results, she was diagnosed with late-onset schizophrenia. Late age at onset and no past psychiatric history would point in favor of this diagnosis. We excluded Psychotic disorder due to a general medical condition, however, she is hypertensive and there are no other medical conditions known to cause such symptoms. The few neurological abnormalities identified were generally consistent with age. Delirium is excluded because she had no fever and her blood pressure under control so this is not associated with delirium. There was no history of substance abuse and laboratory results showed no evidence of acute drug or alcohol intoxication or liver disease. So ruled out Psychosis secondary to substance abuse/dependence. Also excluded was dementia because several points against her diagnosis included a lack of marked memory disturbances, but our patient had mild cognitive impairment, no dysphasia, and no significant vasoconstriction deficits on neurological examination. There was no decline in cognitive performance over the follow-up period, and overall neuropsychological performance was within normal limits. However, the possibility of a very early dementing process could not be ruled out. Late-onset psychosis with paranoid delusions may seem to favor the diagnosis of

delusional disorder. Bizarre delusions and prominent auditory hallucinations would, however, rule out this condition. At last, we ruled out Psychosis not otherwise specified because she showed persistent symptoms of schizophrenia.

A common feature of late-onset psychosis is partition delusion, which is involved. As with our patient, she has the same findings, such as suspicion about her neighbors, claiming they will steal her house and that cameras and televisions are watching her. Additionally, she hears hearing voice of someone talking to her. In late-onset psychosis negative features are rarely seen, but in our patients most of the complaints are positive. Another significant factor is that our patient does not have any family history of psychosis nor is she taking any previously prescribed medications. Cognitive impairment patterns in patients with very late-onset schizophrenia are similar to those in our patient, and she improved after taking anticholinesterase agents, like donepezil. One of the factors in late-onset psychosis is structural brain abnormalities. In our case, the patient showed mild diffuse brain atrophy at the frontal lobes.

It is challenging not only just to diagnose late-onset psychosis, but also to treat it. There has not been an adequate study of non-pharmacological treatment strategies, but the age of patients should not constitute a barrier to accessing these therapies. However, it is the first option for treating late-life psychosis. Psychological management may reduce the distress associated with psychotic symptoms. Family therapy, CBT is known for its effectiveness in improving positive and negative symptoms. Although she has mild diffuse brain atrophy, CBT and other therapies can be utilized, despite their pros and cons. As part of our treatment, we gave functional adaptation skills training, cognitive remediation therapy, family interventional therapy, self-management training, lifestyle interventions, and family psycho-education, with pharmacological treatment to our patients, and the compliance rate of treatment improved.[15] The use of antipsychotics in older adults can be difficult due to the various, and often serious, side effects. These include sudden death. Contemporary guidelines suggest that antipsychotics should be prescribed cautiously using a low dosage and over a short time.[16] There is no doubt that antipsychotic drugs are a mainstay of treatment. Initially, drug treatment should be started at very low doses and gradually increased. Patients who present with late-onset disease usually respond to dose amounts that are one-quarter to one-half those given to patients with early-onset disease. Patients with very late-onset cases may respond to doses as low as tenths of those used in young adults. In our patient we started antipsychotic haloperidol 1.5 mg in a low dose and gradually increased it up to 3 mg per day. Her psychotic symptoms almost disappeared after 3 months of follow-up period. As long as the side effects and effectiveness of the medication are closely monitored, she should continue to receive antipsychotic medications. Metabolic changes and drug interactions are more likely to occur in elderly patients.[17] In our patient, we avoided giving atypical antipsychotics as she has dyslipidemia, and we were able to get positive results with typical antipsychotics in low doses.

CONCLUSION

There are several causes of late-onset psychosis, including systemic medical diseases, neurodegenerative disorders, and substance abuse disorders. An etiological opinion can be drawn by clinicians after carrying out an in-depth collateral history and physical examination, as well as monitoring laboratory tests and neuroimaging. It is crucial to initiate medication carefully with the lowest dosage for short-term efficacy and monitor the adverse effects of antipsychotic medications. There is still a need for more research. However, it appears that subtle cognitive deficits, social isolation, and sensory impairment are associated with a significantly increased risk of psychosis in elderly patients who are genetically predisposed. As a result, the treating clinician should take these factors into account when developing an appropriate treatment plan. In addition to antipsychotic medication, anticholinesterase inhibitors may also be effective in treating these patients.

Conflict of interest

There are no conflicts of interest.

Author contribution

JAS- material preparation, conception and design, or acquisition of data, or analysis and interpretation of data, SFQ-draft of the manuscript was written, drafting or revising it critically for significant intellectual content. HHM & AME-Critical review and editing of the final manuscript were done.

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