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(CASE REPORT)

Psychiatric Symptoms as Indicators of Neurodegenerative Processes: Lewy Body Disease as an Example of the Essential Collaboration Between Neurology and Psychiatry

Wafaa Mansouri *, Jalal Salim, Abdelilah Nait Abbou, Yahya Amara, Khalid Mouhadi and Mohamed Kadiri

Department of Psychiatry, Mohammed V Military Training Hospital, Faculty of Medicine and Pharmacy, Mohammed V University, Rabat, Morocco.

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Abstract

Introduction: Psychosis encompasses a group of psychiatric disorders within the spectrum of psychotic disorders, primarily characterized by a significant disruption in contact with reality. The typical age of onset for psychotic disorders ranges from late adolescence to early adulthood, approximately between 15 and 35 years. However, in some cases, psychotic symptoms may emerge after the age of 40, at which point the condition is termed late-onset psychosis. This unusual clinical presentation should prompt, as a first-line consideration, a search for an underlying organic etiology, most notably a neurodegenerative disorder. Among these, Lewy body disease (LBD) serves as a significant differential diagnosis due to its frequent association with early visual hallucinations, pronounced cognitive fluctuations, and extrapyramidal signs.

Case Description: The patient is a 58-year-old man presenting to psychiatric services for the first time with symptoms of anxiety and depression, reportedly beginning during the COVID-19 pandemic. He complained of unfounded fears, social withdrawal, low mood, and sleep disturbances. He was prescribed antidepressants and anxiolytics and referred for a neurological consultation.

The neurological examination, cranial CT scan, and standard blood tests were unremarkable. The clinical course remained stable until, four years later, the patient suddenly exhibited childlike and disinhibited behavior, developed psychomotor instability, and experienced worsening sleep disturbances. A therapeutic shift to antipsychotic medication was implemented. The psychiatric picture then evolved to include visual and auditory hallucinations, as well as delusions of persecution and abandonment. A new neurological assessment was requested.

The neurological examination at that time revealed a Parkinsonian syndrome characterized by tremor and akinetic-rigid features (pre-existing but exacerbated by neuroleptic treatment), along with deficits in working memory and fluctuating attention and alertness. A comprehensive metabolic and infectious workup for dementia yielded negative results. A brain MRI showed grade 1 vascular leukoencephalopathy according to the Fazekas scale. A diagnosis of Lewy body disease (LBD) was subsequently established.

The patient was started on 600 mg of quetiapine per day and 50 mg/day of piribedil (a dopamine agonist), resulting in an almost complete resolution of symptoms.

Discussion: Late-onset psychosis affects individuals older than 40 and is secondary in approximately 60% of cases, often due to neurodegenerative disorders, medications, or toxic exposures. Lewy body disease (LBD) is a neurodegenerative condition characterized by a classic triad of symptoms: fluctuating cognitive impairment, early visual hallucinations, and Parkinsonian syndrome. A diagnosis of probable LBD is established when the triad

* Corresponding author: Wafaa Mansouri

of dementia, parkinsonism, and psychiatric symptoms is present. Distinguishing between these clinical entities relies on a careful analysis of the nature and characteristics of hallucinations and delusions, the progression of symptoms over time, and their association with neurological signs. In our case, psychiatric symptoms were the predominant clinical feature for several years, during which neurological examinations showed no significant findings. Parkinsonian signs and cognitive decline developed gradually over time. Ultimately, the sudden worsening and complexity of the psychiatric presentation led us to reconsider the initial diagnosis in close collaboration with our neurology colleagues.

Conclusion: Late-onset psychosis requires particular attention due to the possibility of an underlying secondary cause, its higher morbidity and mortality compared to primary psychotic disorders, and the need for tailored therapeutic strategies based on the etiology. Our case—Lewy body disease (LBD) with psychiatric onset—illustrates a clinical situation that necessitates close collaboration between neurologists and psychiatrists.

Keywords: Late-onset psychosis; Lewy body disease (LBD); Neurodegenerative disorders; Visual hallucinations; Parkinsonism.

1. Introduction

Certain neurological disorders may initially manifest through psychiatric symptoms, complicating and often delaying the diagnostic process. Among these, Lewy body disease (LBD) stands out as a crucial differential diagnosis, particularly in the presence of atypical cognitive and behavioral presentations. LBD is a neurodegenerative disorder characterized by a symptomatic triad of fluctuating cognitive impairments, early visual hallucinations, and Parkinsonian features. This core symptomatology is frequently accompanied by REM sleep behavior disorder (RBD), autonomic dysfunction, and marked neuroleptic sensitivity. Visual hallucinations, among the most common early symptoms of LBD, have been associated with specific alterations in brain regions involved in visual-perceptual, attentional, and executive functions, as demonstrated by voxel-based morphometry (VBM) analyses and recent neuropsychological meta-analyses [1]. Their prevalence is estimated at over 80% in LBD patients, significantly contributing to caregiver distress and severely impacting quality of life [2]. These hallucinations, often complex, may recur from the early stages of the disease and are frequently experienced as disturbing or even distressing by patients. Their persistent and detailed nature distinguishes them from the transient hallucinations observed in other conditions [3]. Moreover, the frequent underrecognition of LBD within the medical community and the polymorphic nature of its clinical presentation contribute to delayed diagnosis. In the early phases, psychiatric symptoms may dominate the clinical picture and be misinterpreted as primary psychotic disorders, particularly when hallucinations are isolated or accompanied by delusional ideation or anxiety. We present here the case of a patient whose clinical evolution exemplifies the diagnostic complexity of LBD, highlighting the challenges of early recognition and the importance of an integrative approach that combines psychiatric, neurological, and neuropsychological data.

2. Clinical Observation

The patient is a 58-year-old married man and father of two children. He is illiterate and works as a gardener. He has no prior psychiatric or somatic history, particularly no known neurological conditions. He is a chronic smoker with a history of 35 pack years.

In mid-2020, he was referred for psychiatric consultation due to work incapacity, persistent sadness, insomnia, and anxiety, evolving over several months. Based on this presentation, a diagnosis of major depressive disorder with anxious distress was established, leading to the initiation of treatment with sertraline (a selective serotonin reuptake inhibitor) and prazepam (a benzodiazepine).

Over the following four years, the clinical course was marked by stabilization and a good therapeutic response.

Sudden psychomotor instability, insomnia, and the emergence of disinhibited behavior abruptly altered the clinical picture, raising suspicion of a manic switch. The patient was hospitalized; sertraline was discontinued, and treatment with levomepromazine (100 mg) followed by olanzapine (10 mg) was initiated. Due to a lack of significant clinical improvement and the persistence of agitation, a new neurological evaluation was requested.

Brain imaging (CT scan showing no significant abnormalities; MRI revealing grade 1 vascular leukoencephalopathy according to the Fazekas scale) did not allow for a clear etiological diagnosis at this stage. However, the work-up revealed an atrioventricular block, which necessitated the implantation of a dual-chamber pacemaker, indicating involvement of the autonomic nervous system.

During hospitalization, clinical features suggestive of Lewy body disease (LBD) emerged: marked cognitive fluctuations, impaired attention and alertness, childlike behavior, complex visual and auditory hallucinations, sleep disturbances consistent with REM sleep behavior disorder (RBD), and an akinetic-rigid parkinsonian syndrome, predominantly on the left side.

The case was presented at a neuroscience multidisciplinary meeting for diagnostic and therapeutic clarification. The final diagnosis retained was Lewy body disease. The patient was treated with quetiapine at a dose of 600 mg per day, which led to stabilization of mood, psychomotor behavior, and sleep, as well as resolution of hallucinations within two weeks.

Fifteen days later, the patient was re-hospitalized to initiate piribedil (a dopamine agonist), which was gradually titrated up to 50 mg/day. This resulted in a reduction and eventual disappearance of tremors, with no adverse effects observed. Cognitive symptoms, however, persisted.

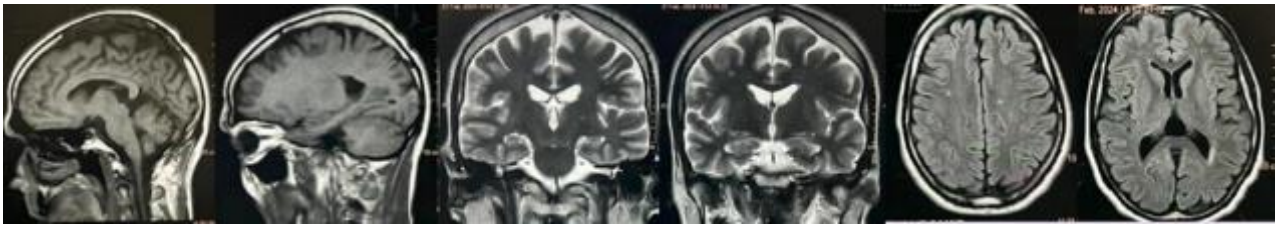

Figure 1 Brain MRI in T1, T2, and FLAIR sequences showing Fazekas grade I vascular leukoencephalopathy, with no other parenchymal lesions, cortical or subcortical atrophy, or ventricular system abnormalities

3. Discussion

The diagnostic complexity of Lewy body disease (LBD) lies in its predominance—sometimes exclusive—of initial psychiatric manifestations. This case highlights the risk of diagnostic delay or misdirection when symptoms are viewed in isolation without considering a potential neurological origin. Early visual hallucinations, cognitive fluctuations, and marked neuroleptic sensitivity should alert clinicians and prompt a comprehensive neurological assessment [2,4,5].

Although visual hallucinations are hallucinatory, they occur in over 70% of patients with LBD and serve as a key warning sign. In early stages, they may cause confusion, anxiety, or avoidance behaviors, as demonstrated in Yumoto & Suwa's qualitative study, which emphasized patients' subjective experiences and coping strategies, such as modifying their home environment to manage distress [3,5]. Moreover, these hallucinations have been associated with morphological abnormalities in the occipitotemporal, parietal, and frontal brain regions, as shown in voxel-based morphometry meta-analyses, supporting their neurodegenerative origin [1].

The use of antipsychotics, especially first- and second-generation neuroleptics, is known to worsen motor symptoms in patients with LBD. Due to dopaminergic hypersensitivity, about 50% of patients develop severe adverse effects, including rigidity, catatonia, or even neuroleptic malignant syndrome [4,5,6]. In our case, the worsening of Parkinsonian symptoms following antipsychotic administration strongly suggests an iatrogenic mechanism. Current guidelines recommend extreme caution in prescribing neuroleptics for this population [4,7].

This case clearly illustrates the iatrogenic risk: that the introduction of antipsychotics exacerbated the motor profile. A recent case report also highlights this issue, where intolerance to conventional antipsychotics was overcome with pimavanserin. This selective 5-HT2A receptor inverse agonist has shown efficacy without worsening motor symptoms [8]. Alternatives such as electroconvulsive therapy (ECT) have also demonstrated promise, especially for severe or treatment-resistant depressive or psychotic symptoms, with positive outcomes reported in over 75% of cases according to a recent systematic review [9].

Furthermore, the emergence of cardiac dysautonomia—in this case, an atrioventricular block—supports the hypothesis of widespread autonomic nervous system involvement. Nagahama et al. showed that autonomic symptoms—including sleep-wake rhythm disturbances and orthostatic hypotension—are common even in moderate stages of LBD, contributing to overall morbidity and clinical decline [2]. These signs are now considered relevant prodromal features and are incorporated into screening tools like the DCARD checklist, which has recently been validated for early detection of LBD in memory clinics [10].

From a cognitive perspective, neuropsychological profiles in LBD are distinctly different from those seen in Alzheimer's disease (AD). Executive, attentional, and visuospatial deficits are early and prominent in LBD, while memory impairments tend to be more modest in the initial phases. This has been confirmed by Pezzoli et al. in a meta-analysis of 35 neuropsychological studies, which identified significant deficits in immediate memory, visual attention, and executive functions among patients with hallucinations [1]. Additionally, depressive symptoms tend to be more frequent and severe in LBD compared to AD, worsening progressively as patients approach death, as shown in a recent five-year longitudinal study [11]. Therefore, differential neuropsychological assessments are vital for guiding diagnosis.

Finally, recognizing hallucinations as neurological symptoms—rather than purely psychiatric phenomena—is a crucial step toward acceptance of the diagnosis by both the patient and their family. As emphasized by Yumoto and Suwa, psychological support and family education are essential to reduce isolation and promote adaptation [3].

4. Conclusion

This case highlights the importance of a multidisciplinary clinical approach that combines psychiatry and neurology when assessing atypical or treatment-resistant psychiatric symptoms. Early signs of Lewy Body Dementia can mimic primary psychiatric disorders—especially psychotic or mood disorders—making diagnosis particularly challenging. Recognizing key features such as complex visual hallucinations, cognitive fluctuations, and heightened sensitivity to antipsychotics is essential to avoid inappropriate treatments and their potential harmful effects. This serves as a reminder to clinicians, particularly psychiatrists, to stay alert in diagnosing unusual, fluctuating, or resistant psychiatric symptoms and to consider the possibility of an underlying neurological condition. In this context, early screening tools, refined neuropsychological evaluations, and appropriate additional tests can aid in quicker, more accurate diagnosis and targeted management.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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