Clozapine for Management of Neuropsychiatric Symptoms in Dementia with Lewy Bodies: Case Report and Literature Review

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INTRODUCTION

Dementias are a group of neurocognitive disorders characterized by a significant decline in one or more domains of cognition that interferes with a person's ability to function. According to the Canadian Chronic Disease Surveillance System, more than 402 000 older adults are living with some type of dementia, including Alzheimer disease, in Canada (excluding Saskatchewan).[1] Approximately 76 000 new cases of dementia are diagnosed each year, with a projected total annual health care cost of $16.6 billion for Canadians with dementia by 2031.[1] Dementia with Lewy bodies (DLB) is a type of dementia for which probable diagnosis requires the presence of dementia and at least 2 core clinical features.[2] The core clinical features of DLB are fluctuating cognition, recurrent visual hallucinations, rapid eye movement sleep behaviour disorder, and 1 or more features of parkinsonism, such as bradykinesia, rest tremor, and rigidity.[2] These symptoms can be very disabling and significantly affect quality of life for both patients and their families.

"Lewy body dementia" is an umbrella term for major neurocognitive disorders caused by Lewy body pathology. Parkinson disease dementia (PDD) and DLB are the 2 main syndromes within Lewy body dementia. Together, they constitute the second most common type of degenerative dementia in persons older than 65 years.[3] The major clinical feature that differentiates PDD and DLB is the relative timing of the onset of parkinsonism and dementia. In PDD, motor symptoms precede dementia by at least 1 year, but in DLB, they occur within 1 year of each other.[4] A systematic review estimated that the proportion of individuals with DLB ranged from 0% to 23% among people with dementia.[5]

The evidence base for the pharmacological management of DLB is limited and is founded on evidence related to Parkinson disease psychosis. Despite the paucity of randomized controlled trial data in DLB, acetylcholinesterase inhibitors, atypical antipsychotics such as quetiapine and clozapine, memantine, and nonpharmacological approaches are used for patients with DLB.[4] One major challenge in finding the optimal treatment is the interplay of neurotransmitters such as dopamine, acetylcholine, and serotonin and their opposing effects on neuropsychiatric symptoms such as hallucinations and motor symptoms in DLB. Clozapine is a tricyclic dibenzodiazepine antipsychotic with efficacy in treating hallucinations and psychosis in Parkinson disease without dementia[6] and is less likely than other medications to worsen motor symptoms because of its distinctive dopamine receptor binding.[7] However, the number of case studies describing the use of clozapine in DLB patients is limited.[8] Here, we describe a DLB patient whose severe hallucinations and other neuropsychiatric symptoms were managed by clozapine without adverse effects.

CASE REPORT

In mid-June 2021, a 76-year-old retired male member of the Canadian Armed Forces was admitted to our specialized dementia care unit from an acute care hospital for assessment and treatment of behavioural and psychological symptoms of dementia including hallucinations, aggression, and paranoia.* At the time, he had a diagnosis of unspecified dementia (suspected Lewy body dementia), post-traumatic stress disorder (PTSD), rapid eye movement sleep behaviour disorder, insomnia, type 2 diabetes mellitus, and hypertension.

In 2010, the patient had begun experiencing PTSD symptoms related to his prior military experiences. His symptoms of rapid eye movement sleep behaviour disorder progressed over time, and he was followed by a sleep clinic and a geriatric psychiatry clinic. He was started on quetiapine for his vivid visual hallucinations and olanzapine for

*The authors were unable to obtain patient consent. As per the study institution's research ethics guidelines, all potentially identifiable information not relevant to the case has been omitted.

better control of his neuropsychiatric symptoms, such as poor social communication and agitation.

In early June 2021, he was admitted to a community hospital for violent behaviours with cognitive decline, and a diagnosis of likely Lewy body dementia was made. Before admission, the patient had been hallucinating and claimed that some men were threatening him and his wife. A physician's assessment noted tremors on the patient's left side, with pill rolling and rigidity. Rivastigmine 1.5 mg twice daily was prescribed, along with olanzapine 2.5 mg daily and risperidone 0.25 mg twice daily as needed. His home medications of citalopram 20 mg daily, quetiapine 12.5 mg daily, and clonazepam 0.5 mg at bedtime were continued. Computed tomography showed hippocampus and occipital atrophy, with no acute changes and no hemorrhage. Because of wandering and episodes of falling, the medical team had to restrain him, which was emotionally challenging for his family. Therefore, after about 2 weeks, the family decided to transfer the patient to another hospital.

Upon admission to our institution's specialized dementia care unit, in mid-June, a physical assessment showed parkinsonian symptoms of cogwheel rigidity, unilateral hand tremor at rest, unsteady gait necessitating use of a walker for ambulation, and decreased blink rate. Despite the unsteady gait, the patient had no underlying signs of dizziness or syncope. His vital signs were normal, and initial white blood cell (WBC) count and absolute neutrophil count (ANC) were within normal limits (10.8×10^9/L and 7.3×10^9/L, respectively). His scheduled medications at the time of admission to our unit included olanzapine 2.5 mg daily, risperidone 0.25 mg twice daily, quetiapine 12.5 mg daily, rivastigmine 1.5 mg twice daily,and citalopram 20 mg daily.

Magnetic resonance imaging showed moderate diffuse brain atrophy with ventricular prominence and microangiopathic changes, but no ischemia or hemorrhage. A Montreal Cognitive Assessment test was done, in which the patient scored 9 out of 30, with cognitive deficits mainly in the domains of visual–spatial function, executive function, and memory.

The olanzapine and risperidone were discontinued, and quetiapine 12.5 mg 3 times daily as needed was prescribed. Given multiple "code white" episodes for violent and aggressive behaviours, the quetiapine dose was increased to 25 mg twice daily. Multiple breakthrough doses of parenteral lorazepam and loxapine were needed to control his behavioural and psychological symptoms of dementia in the following weeks. During the code white episodes, the patient was verbally and socially inappropriate toward staff members and showed physical aggression by lifting tables above his head and breaking objects. Given the safety risk to staff, the patient was not eligible to participate in recreational activities as part of his nonpharmacological treatment plan. With higher doses of quetiapine, no excessive sedation was observed, but the patient's gait was less steady after titration to the 100-mg daily dose.

With the updated daily dose of quetiapine 100 mg, a change to rivastigmine patch 13.3 mg, and initiation of trazodone 100 mg daily, the patient continued experiencing distressing visual hallucinations, delusions, incoherent speech, restlessness, fluctuations in mental function, periods of lethargy, and memory problems. His impaired cognition and psychotic symptoms had negatively affected his decision-making and activities of daily living, and his PTSD complicated the final diagnosis. Two weeks after admission to our institution, a diagnosis of probable DLB was made. The patient met the full diagnostic criteria for DLB, with the following 4 core clinical features: fluctuations in attention and alertness, prominent and well-formed visual hallucinations, parkinsonism, and rapid eye movement sleep behaviour disorder.

In July 2021, a neurological assessment of the patient's motor function showed cogwheel rigidity and paratonia throughout the upper and lower extremities, along with bilateral tremors in the upper limbs at rest. He exhibited an unsteady gait that necessitated the continued use of a walker. Quetiapine was discontinued, and clozapine was initiated at 6.25 mg daily, with very slow titration up to 50 mg daily. At this time, the patient was also receiving rivastigmine 13.3 mg transdermally daily and trazodone 50 mg 3 times per day. Citalopram was tapered down and discontinued in early August 2021. To monitor for agranulocytosis, the patient was registered with the Clozapine Support and Assistance Network, and weekly blood tests were arranged. His WBC count and ANC remained within normal limits. He tolerated clozapine very well and did not experience adverse effects of sedation, hypersalivation, or weight gain. Neither parkinsonian symptoms nor extrapyramidal symptoms (EPS) were observed. Approximately 2 weeks after initiation of clozapine, he was noticeably calmer and less agitated, and he was experiencing fewer hallucinations. Nonpharmacological strategies such as involvement in creative arts, painting and music therapy, yoga, physiotherapy, and occupational therapies were added to his treatment plan to decrease his agitation, as well as to improve his gait stability, function, and overall quality of life. These strategies also helped to improve his sleep, with minimal use of melatonin 5 mg at bedtime as needed for insomnia.

By November 2021, the patient's cognitive status had improved significantly on a stable dose of clozapine 50 mg daily. He still experienced some cogwheel rigidity, but it did not worsen during the titration of clozapine. The patient did have a fall that month and was found to have some mild orthostatic hypotension, which prompted a decrease in his candesartan dose. Subsequent assessments showed that he was no longer experiencing delusions and was having no periods of altered perception during the day, no periods of disorganized speech, and less frequent periods of restlessness with fluctuating mental function throughout the day. There

were no further occurrences of aggression or violent behaviour. His hallucinatory episodes improved, although he did have occasional hallucinations briefly in the evenings. These were not distressing to him, and he was easily redirected by nurses and other staff without the need to administer breakthrough medications. He was very cooperative with care, and his motor symptoms such as cogwheel rigidity and tremors remained essentially the same as before clozapine was initiated. His symptom control also led to improved quality of life, and he has been participating in social activities inside and outside the facility, such as going on bus trips and city tours. A timeline of changes in the patient's psychoactive medications is presented in Figure 1.

In February 2022, a neurological reassessment noted that the patient was functionally improved compared with his prior assessment (before initiation of clozapine treatment). He could stand easily from a seated position by pushing up from the chair. With his walker, he continued to take short steps with some foot dragging, but there was no gait festination, no turning en bloc, and no gait freezing. Physical examination showed asymmetric parkinsonism, with the right side being more rigid, tremulous, and bradykinetic than the left.

DISCUSSION

To date, Health Canada has not approved any medications for DLB, and all current treatments are therefore used off-label. For example, acetylcholinesterase inhibitors and memantine may improve cognitive and neuropsychiatric symptoms in DLB.[9] If antipsychotics are needed for severe or distressing psychotic symptoms, the typical antipsychotics are avoided in patients with DLB because these drugs have higher dopamine antagonism, with a higher risk for motor symptoms and EPS. Clinicians often select clozapine and quetiapine for patients with PDD and DLB because these drugs have lower affinity for D2 receptors, but the evidence for their efficacy in DLB is low.[8,10] The use of clozapine is limited because of the idiosyncratic and very low but serious risk of agranulocytosis (0.38% in more than 99 000 patients[11]), which requires frequent blood monitoring.[12] The efficacy of clozapine in treating psychotic symptoms in patients with Parkinson disease without worsening of motor symptoms has been demonstrated in randomized clinical trials.[13-15] Clozapine exhibits weak D2 antagonism, strong serotonin-2 receptor (5-HT2A) antagonism, and fast dissociation from the D2 receptor, leading to its favourable motor profile.[16] However, clinical trials have not been conducted with clozapine in patients with the sole diagnosis of DLB. There is also little evidence for the efficacy of nonpharmacological approaches, because research involving patients with DLB has been limited. Patient- or caregiver-focused education and training to manage psychiatric symptoms, the elimination of triggering environmental stimulants, exercise (both motor and cognitive), and cognitive behavioural therapy are commonly used, despite the limited evidence.[2,17] This case report aims to shed light on some of the pharmacological challenges in treating patients with DLB and adds

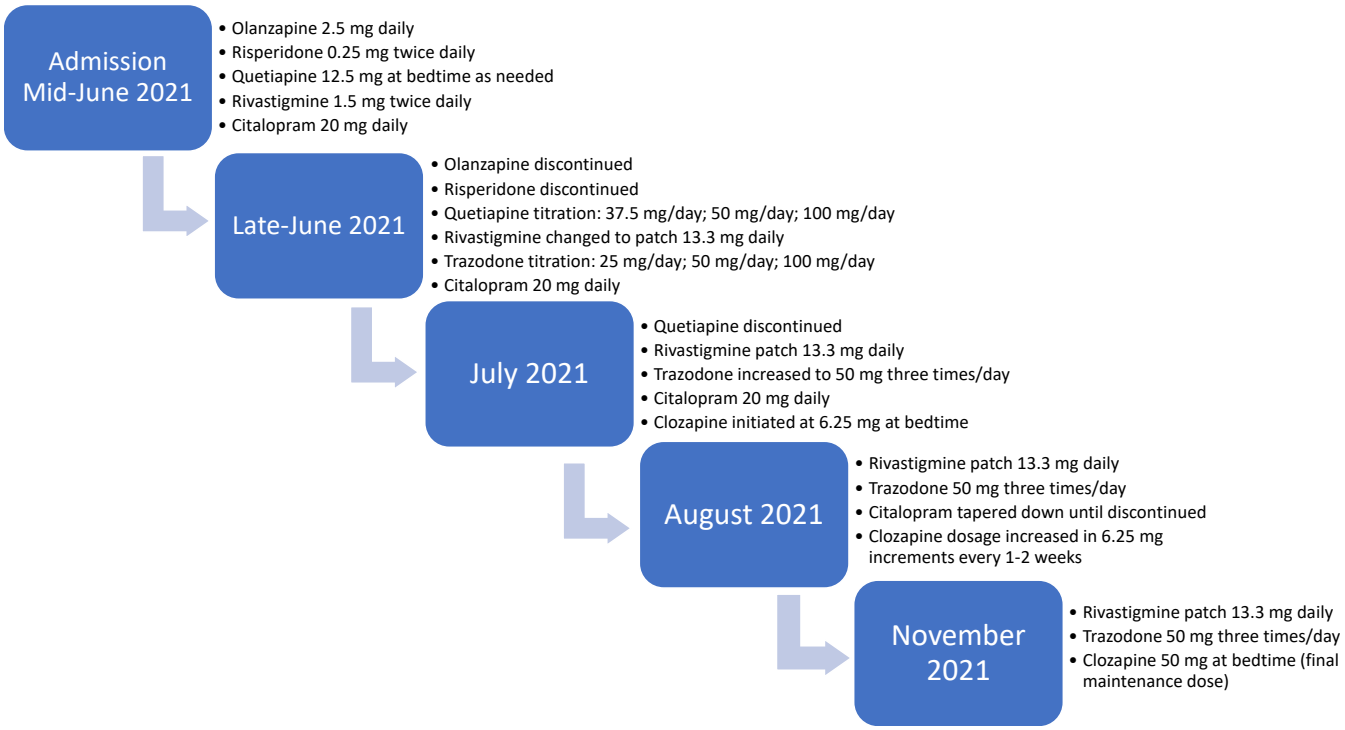

FIGURE 1. Timeline of changes in the patient's psychoactive medications.

to the evidence for the efficacy and safety of clozapine when the patient is monitored appropriately.

We conducted a literature search of randomized clinical trials, systematic reviews, meta-analyses, case reports, and retrospective chart reviews comparing clozapine with placebo, other pharmacological options, and nonpharmacological treatments for DLB. We searched several databases, specifically TRIP PRO, the Cochrane Database of Systematic Reviews, Embase, PubMed, MEDLINE, and PsycINFO, as well as the grey literature, from January 1993 to May 2022, with keywords for the population and interventions of interest: (clozapine OR Clozaril) AND (Lewy* OR dementia). Six case studies (5 in English and 1 in Dutch) documented the treatment of DLB patients with clozapine (Table 1).[18-23] We also found several systematic reviews based on these few case studies on the use of clozapine in DLB and therapeutic alternatives for DLB and PDD. Patient demographic characteristics, prior or concomitant use of acetylcholinesterase inhibitors or dopaminergic or antipsychotic drugs, and the outcomes of these 6 case studies (describing a total of 7 patients) are presented in Table 1. The primary goal of therapy in all of these case studies was to reduce the patient's symptoms, such as hallucination, delusions, and aggression, that were not mitigated with previous medication regimens. Neuroleptic sensitivity in 2 patients (described in a single case report) resulted in discontinuation of clozapine due to worsening hallucinations and confusion.[20] In the other 5 case studies, the goals of therapy were met, as the resolution of patients' hallmark symptoms was clinically significant and patients did not experience increased risk of falls or orthostatic hypotension due to clozapine. Among these 5 patients, 2 patients experienced EPS, which were treated with concomitant daily levodopa[21,22]; a third patient's EPS were minimal and did not interfere with his daily activities.[23] None of the 5 patients in these cases were reported to have experienced neuroleptic sensitivity syndrome or concerning changes in WBC count or ANC as a sign of agranulocytosis due to the use of clozapine. Agranulocytosis is a dangerous but reversible hematological condition that can be managed, if identified early, by regular blood monitoring. Patients may be rechallenged with clozapine once neutrophil counts return to normal.[24] Although there is no evidence derived from randomized clinical trials, these 6 case reports present important points about the efficacy of clozapine. We believe that clozapine can be safely used to improve the neuropsychiatric symptoms of DLB, if monitored appropriately.

The case that we have reported here highlights the efficacy and safety of clozapine in treating DLB core symptoms, such as cognitive fluctuation and hallucinations, without worsening motor symptoms or inducing side effects such as falls, neuroleptic sensitivity, or agranulocytosis. Because clozapine has α-adrenergic blockade and a higher risk of dizziness and orthostatic hypotension, especially in the geriatric population, we monitored the patient's orthostatic blood pressure routinely. Given the patient's history of constipation, he continued taking laxatives, and his bowel movements were monitored daily to allow mitigation of the additive anticholinergic side effects of clozapine. Upon admission to our specialized dementia care unit, the patient's risperidone and olanzapine were discontinued and replaced by quetiapine, given that his parkinsonian symptoms were likely due to the higher dopamine antagonism of risperidone and olanzapine relative to quetiapine.[9] Also,

TABLE 1. Case Studies Investigating the Use of Clozapine for Patients with Lewy Body Dementia

Study	Age (yr) and Sex	Cognitive Screening Test	Prior Medications	Clozapine Initial–Final Dose[a] (mg/day)	Concomitant Medications (mg/day)	Outcomes and Efficacy
Chacko et al. (1993)[18]	57, female	NR	Levodopa, selegiline, pergolide	25–75	None	Resolution of hallucinations, improved mood, and improved social communication
Geroldi et al. (1997)[19]	74, female	MMSE 19/30	Haloperidol	12.5–37.5	None	Satisfactory reduction of visual and tactile hallucinations
Burke et al. (1998)[20]	71, male 69, male	MMSE 27/30 MMSE 16/30	Selegiline, levodopa Pergolide, levodopa	6.25–12.5 6.25	None None	Clozapine discontinued because of increased confusion, hallucination, agitation, and behavioural symptoms
Majic et al. (2010)[21]	73, female	MMSE 20/30	Quetiapine, pipamperone, donepezil	200	Levodopa 375, donepezil 10	Resolution of hallucination and delusions, worsening of parkinsonism
Archie et al. (2013)[22]	77, male	NR	Quetiapine, rivastigmine	75	Levodopa 100	Resolution of hallucination and delusions
Bhamra et al. (2018)[23]	75, male	MMSE 23/30	Donepezil, rivastigmine	6.25–18.75	Rivastigmine patch 13.3	Resolution of hallucinations and agitation, improved mood

MMSE = Mini-Mental State Examination, NR = not reported.
[a]Where only one dose appears, the patient's dose did not change over the course of treatment.

olanzapine has anticholinergic side effects and a higher risk for metabolic syndrome that could affect the patient's diabetes. By approximately 12 months after clozapine initiation, the patient's positive outcomes persisted, and his mild, infrequent hallucinations and agitation had resolved. In compliance with Health Canada drug monitoring requirements,[25] blood monitoring was conducted weekly for the first 6 months and biweekly for another 6 months, during which time his WBC count and ANC were consistently within the normal limits (4–11 × 10^9 /L for both); this monitoring will continue in the future. Because of disease progression, his complete dependence on others for activities of daily living, and the difficulty of caring for him at home, there is currently no plan to discharge this patient. However, in cases where the patient can be discharged, the drug manufacturers offer a compassionate program for off-label use of clozapine, which assists patients with medication costs and a monitoring plan.[26] Given the serious risk of agranulocytosis associated with clozapine, patients, their health care providers, and their dispensing pharmacists must be enrolled in a registry specific to 1 of the 3 marketers of clozapine in Canada.[25] Alternatively, Veterans Affairs Canada has coverage for veterans, depending on their pension conditions.[27]

Pimavanserin is a selective serotonin 5-HT2A receptor inverse agonist that was recently approved by the US Food and Drug Administration (but is not marketed in Canada) for the treatment of hallucinations and delusions associated with Parkinson disease psychosis.[28] The effects and maximal benefit of this drug are not immediate and can take 2–6 weeks to achieve, which may not be ideal in acute care settings.[29] As of summer 2023, patients are being recruited to an open-label trial that will compare quetiapine and pimavanserin among patients with psychosis due to PDD or DLB.[30]

Ours was an observational case report with no control group for comparison. As with most case reports, unmeasured confounding, selection bias, and recall bias limit the generalizability of our findings. In 1 previous case report, the authors were able to conduct a variety of objective assessments such as the Mini-Mental State Examination before and after initiating clozapine to evaluate the outcomes,[23] but this type of testing was not applicable in our clinical setting. Therefore, our outcomes might reflect measurement and interviewer biases. Despite these caveats, this case report supports the use of clozapine for treating patients with DLB. There is a need for further research, through larger case series and placebo-controlled trials, to better assess the place of clozapine and other treatment options in guidelines for managing DLB.

CONCLUSION

In the patient case presented here, clozapine was an effective treatment option for persistent psychosis and aggression in DLB. Through its anticholinergic activity, this drug has a multitude of possible side effects that may significantly affect an older patient like the one described here, such as constipation and urinary retention.[25,26] Therefore, consideration must be given to identifying the side effects to which the person may be vulnerable, and appropriate monitoring plans should be put in place to monitor for and reduce the risks of these side effects. The use of low doses and slow titration remains appropriate and is the safest way to monitor for emerging side effects and to identify the lowest therapeutic dose.

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