FUNCTIONAL DECLINE IN A PATIENT WITH ALZHEIMER'S DISEASE WITH BEHAVIOURAL AND PSYCHOLOGICAL SYMPTOMS OF DEMENTIA: RE-EVALUATING THE DIAGNOSIS AND MANAGING CAREGIVER CONCERNS

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ABSTRACT

Madam C, an 81-year-old lady, with a history of Alzheimer's disease (AD) with behavioural and psychological symptoms of dementia (BPSD) was admitted to the community hospital (CH) for rehabilitation following functional decline from newly diagnosed drug-induced parkinsonism (DIP). The concurrent issues of worsening function and ongoing behavioural issues contributed to significant caregiver stress. This case illustrates how family physicians help transition patients to community care: by re-evaluating diagnosis and managing caregiver concerns.

Keywords: Geriatrics, Dementia

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INTRODUCTION

Family physicians (FPs) often interact with patients who present with undifferentiated symptoms. Our generalist perspective to patient care allows us to adopt a biopsychosocial approach to identify hidden agenda and make a comprehensive diagnosis of the patient's problems.[1]

Whilst providing care for a patient with dementia, it is also important to provide care for their caregiver. Caregiver burden increases with dementia severity and the presence of BPSD.[2] FPs are well placed to provide personal, comprehensive, and coordinated care to patients with dementia and their caregivers.

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CASE STUDY

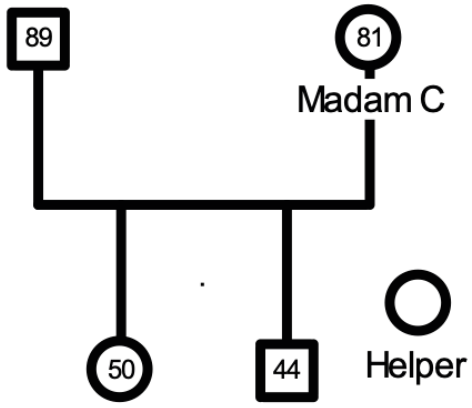
Madam C is an 81-year-old Chinese female who was admitted to a tertiary hospital for functional decline with parkinsonism features after recent discharge from post-subarachnoid haemorrhage rehabilitation.

During her tertiary hospital admission, she was diagnosed with:

1. Functional decline secondary to DIP
- Causative agent: risperidone (medication ceased)
- Function:
    - Sit-to-stand and basic ADL: 2-person maximum assistance
    - Ambulation: unable to ambulate with assistance
2. Mixed delirium, background of AD with BPSD
- Precipitated by left knee osteoarthritis flare, psychotropic medication use, hospital environment
- CT brain: no acute abnormalities, old lacunar infarcts present
- Withheld fluvoxamine 50 mg ON due to drowsiness
- Reduced Epilim Chrono to 300 mg ON (previously 300 mg BD)
- Started quetiapine 12.5 mg BD due to BPSD
3. Vitamin D insufficiency

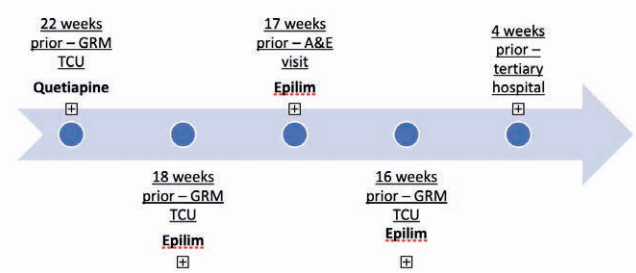
Madam C was transferred to the CH for rehabilitation. Upon admission to CH, Madam C did not have joint pain nor did she report any hallucinations. On examination, supine BP was 131/68 mmHg without postural hypotension. Her BMI was 21.4kg/m^2. Neurological examination revealed bradykinesia, cogwheeling, and rigidity in both upper limbs. Power was 4 in all limbs. Hypomimia was present. There were no cerebellar signs or resting tremor. Visual acuity was 6/9 bilaterally. There was no vertical gaze palsy. Madam C required 2-person maximum assistance for bed mobility and sit-to-stand and was unable to ambulate. Madam C was alert during the assessment and did not display inattention. Mini Mental State Examination score was 16/30.

Background



Prior to the recent admissions, Madam C was pre-morbidly ADL independent and community ambulant with a walking stick. Her BPSD manifested as persecutory delusions and physical assaults towards her son. Her BPSD medications were changed over the course of six months (illustrated in **Figure 1**).

Figure 1: Timeline of change of BPSD medications



Madam C lives with her son and a newly hired helper in a 5-room HDB flat. There are no ramps or grab bars in the flat. Madam C is a retired teacher and lives off her pension. Her son was previously a freelance photographer but has not worked since the pandemic. Madam C's daughter lives overseas and supports the family financially.

Background Medical History

1. AD with BPSD

- AD diagnosed three years prior – on follow-up with a geriatrician
- Previously taking donepezil – stopped due to nightmares
- BPSD started six months prior

2. Recurrent falls (nine falls in past year) attributed to poor safety awareness from AD

- Complicated by recent subarachnoid haemorrhage
- No history of fragility fracture

Medication List (upon arrival to CH)

PO Epilim Chrono 300 mg ON
PO quetiapine 12.5 mg BD
PO colecalciferol 1,000 units OM
PO calcium carbonate 1.25 g OM
PO paracetamol 1 g TDS PRN
Topical ketoprofen plaster 1 patch BD PRN

Caregiver's Revelation

Madam C's son conveyed his concerns about Madam C's deterioration in function from premorbid. He was concerned the parkinsonism was not improving despite cessation of the causative medication. He was experiencing caregiver stress due to her underlying BPSD and was worried that the recent change in medications could lead to worsening of BPSD. He was also concerned that she would not regain function thus adding to caregiver burden. He wanted to return to work but was worried that the helper would not be able to handle Madam C alone.

Gaining Insight: What Are the Issues?

1. Is the functional decline solely due to DIP?
2. How do we balance managing patient's underlying BPSD and her son's concerns and expectations of BPSD management?
3. How can we manage her son's caregiver stress whilst supporting the patient to return home safely with adequate resources?

STUDY THE MANAGEMENT: HOW DO WE APPLY THIS IN OUR CLINICAL PRACTICE?

1. Is the functional decline solely due to DIP?

Functional decline in older adults with dementia is often multifactorial, involving an intricate interplay of neurological, pharmacological, and systemic factors. Diagnosing the exact cause requires a comprehensive evaluation to differentiate between reversible conditions and underlying neurodegenerative processes.

Role of DIP

DIP is an adverse effect of dopamine receptor-blocking agents, particularly antipsychotics, which are frequently used to manage BPSD. DIP manifests with parkinsonian features such as bradykinesia, rigidity, and tremors, which resemble idiopathic Parkinson's disease (PD) but are often symmetrical and associated with less pronounced resting tremor.[3]

Risperidone, an antipsychotic used in Madam C's treatment, is well-documented to have a higher propensity for causing DIP compared to other second-generation antipsychotics.[4] Its strong D2 receptor binding affinity and slower dissociation

contribute to this risk. An epidemiological study comparing antipsychotics found that quetiapine and clozapine had the lowest rates of DIP, making them preferable for elderly patients prone to motor side effects.[5] The decision to switch Madam C from risperidone to quetiapine aligns with this evidence-based understanding.

DIP typically resolves within weeks to months of discontinuing the offending drug, but in up to 20 percent of older adults, symptoms persist beyond this timeframe, suggesting an unmasking of pre-existing subclinical neurodegeneration, such as PD or dementia with Lewy bodies (DLB).[6]

Differentiating DIP from Neurodegenerative Parkinsonism

Distinguishing DIP from idiopathic PD or other neurodegenerative parkinsonian syndromes is challenging but critical for tailoring treatment. Clinical clues include:

- History of symptoms: DIP is defined as the presence of parkinsonism without a history of parkinsonism before the use of the offending drug and onset of parkinsonian symptoms during use of the drug.[7] If parkinsonism symptoms predate the use of the drug, PD is the more likely diagnosis.
- Symmetry of symptoms: DIP typically presents with bilateral and symmetric motor features, while PD often begins unilaterally.
- Resting tremor and response to levodopa: PD is more likely to include resting tremor and a robust response to levodopa, whereas DIP shows less pronounced tremor and inconsistent levodopa responsiveness.[3]
- Non-motor symptoms: Non-motor symptoms, such as REM sleep behaviour disorder, hyposmia, and autonomic dysfunction, are more specific to PD and less common in DIP.[8]

Advanced imaging techniques, such as dopamine transporter (DaT) scans, can help differentiate DIP from neurodegenerative parkinsonism. DaT scans typically show preserved striatal dopamine uptake in DIP but reduced uptake in PD and DLB.[3] While not performed in Madam C's case due to limited availability, such tests could have clarified her diagnosis.

Contributors to Madam C's Functional Decline

1. **Underlying idiopathic PD:** Upon further history-taking, Madam C had been having left upper limb tremors for the past year and was noted to be "shuffling" her feet. These symptoms preceded quetiapine and risperidone use.
2. **Hospital-associated deconditioning:** Prolonged hospitalisation following her subarachnoid haemorrhage likely exacerbated physical and cognitive impairment. Evidence shows that older adults lose up to 5 percent of muscle mass per day during bed rest, significantly impairing mobility and function.[9]

Recognising the multifactorial nature of Madam C's functional decline, her management was individualised to address each potential contributor. The initiation of levodopa therapy, based on the suspicion of idiopathic PD, led to significant improvements in rigidity and ambulation. Levodopa, as a dopamine precursor, is the gold standard treatment for PD, with studies confirming its efficacy in improving motor symptoms.[10]

This therapeutic response, combined with structured rehabilitation, underscores the importance of iterative reassessment in complex cases. Functional improvement, to 2-person moderate assistance for basic ADLs, sit-to-stand, and ambulation with walking frame, was achieved despite the challenges of overlapping diagnoses, reaffirming the need for a multidisciplinary approach to care.

2. **How do we balance managing patient's underlying BPSD and her son's concerns and expectations of BPSD management?**

BPSD, affecting over 90 percent of individuals with dementia during their disease course, are a major cause of caregiver distress and institutionalisation.[11] Madam C's well-controlled BPSD during her initial rehabilitation period demonstrated the effectiveness of low-dose quetiapine combined with non-pharmacological measures and caregiver education.

However, her BPSD temporarily worsened during an episode of hyperactive delirium, triggered by a urinary tract infection. Delirium, particularly in dementia patients, is a common consequence of infections. Non-pharmacological strategies such as reorientation, use of music, and daytime cognitive activities have been shown to reduce the severity and duration of delirium episodes without the risks associated with pharmacological treatments.[12]

Madam C's son, concerned about the potential recurrence of BPSD at home, requested medication adjustments. While pharmacological management is sometimes necessary, it is essential to weigh the risks of polypharmacy and side effects, particularly in dementia patients. The morning dose of Epilim chrono was restarted in consultation with her geriatrician. Madam C's symptoms stabilised, illustrating the value of collaborative, evidence-based decision-making in managing BPSD.

3. **How can we manage her son's caregiver stress whilst supporting the patient in returning safely home with adequate resources?**

Caregiver burden is a critical consideration in dementia care, particularly when managing complex cases involving both functional decline and BPSD. Studies consistently demonstrate that caregiver burden increases with the severity of dementia and is strongly associated with reduced quality of life for both patients and caregivers.[2] Madam C's

son's stress revolved around providing care for his mother. His initial Zarit Burden Interview (ZBI) showed moderate to severe burden.

In this case, the multidisciplinary team addressed caregiver stress by implementing a comprehensive discharge plan, including:

- **Caregiver Training:** Evidence shows skill-building interventions, such as hands-on training for managing BPSD and ADLs, significantly improve caregiver competence and reduce stress.[13]
- **Environmental Modifications:** The installation of grab bars and ramps, facilitated through the HDB EASE programme, has been shown to enhance home safety and prevent falls, critical for patients with impaired mobility.[14]
- **Community Resources:** Referring caregivers to online platforms such as DementiaHub.SG and support groups provides ongoing education and emotional support, which are essential for sustained caregiving.
- **Respite and Daycare Services:** Enrolment in dementia daycare programmes provides structured patient activities while allowing caregivers to work or rest, a strategy linked to reduced caregiver burden and improved mental health outcomes.[13]

The tailored plan successfully reduced Madam C's son's caregiver burden, as evidenced by his improved ZBI at discharge. This underscores the importance of an interdisciplinary approach in addressing the needs of both patients and their caregivers.

CONCLUSION

This case highlights the role of the FP as a medical expert and collaborator in re-evaluating and managing the biological interplay of functional decline from anti-psychotic use in a patient with advanced dementia with BPSD. The most appropriate psychotropic agents at the lowest possible dose were used, in collaboration with the inputs of the interdisciplinary team and the geriatrician. It also highlights how an FP bridged communications with the patient's son, enabling a smooth transition with safety netting for the patient as she transitioned into the community.

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LEARNING POINTS

- **Importance of re-evaluating diagnosis: Continuous re-assessment of patients with dementia is crucial, as new symptoms may point to additional diagnoses, which can significantly influence management.**
- **Balancing symptom control and caregiver concerns: Effective communication with caregivers is essential for managing expectations and addressing concerns, especially when balancing the control of BPSD with the patient's overall health and function.**
- **Multidisciplinary approach to caregiver burden: A tailored, team-based approach that includes practical training, resource provision, and emotional support is essential for reducing caregiver burden and facilitating safe patient discharge.**