# A COMPLICATED CASE OF PSEUDODEMENTIA AND PARKINSONISM TREATED WITH SELEGILINE

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**Abstract**

The term "pseudodementia" has attracted much controversy. An elderly man with major depressive disorder, single episode, was misdiagnosed with dementia and subsequently Parkinson's disease, A trial of high-dose Selegiline at 60 mg per day produced substantial improvement with complete resolution of cognitive impairment and disappearance of signs of "Parkinson's disease".

## Introduction

The term "pseudodementia" has attracted much controversy amongst clinicians. There is belief that this may be a separate clinical entity mimicking dementia and characterized by cognitive problems. On the other hand there is postulation that "pseudodementia" is not an illness but a purely descriptive entity without any diagnostic implications. In this report we will discuss our findings in the case of an elderly Caucasian male who presented at our clinic with symptoms of progressive dementia, as well as possibly Parkinson's disease. It appears that he was initially misdiagnosed with dementia, due to his cognitive limitations, compounded by poor response to sertraline 50 mg daily, followed by a trial of donepezil 20 mg daily and memantine 20 mg daily. Behavioral problems induced by these medications resulted in a clinician adding paliperidone to his treatment. Side effects induced by paliperidone further caused another diagnostic error, resulting in the diagnosis of Parkinson's disease. Treatment with a combination of L-dopa, amantadine and ropinirole produced further deterioration of the patient's symptoms, possibly due to further side effects. Discontinuation of all the medications, followed by initiation of selegiline, titrated to high-dose selegiline therapy at 60 mg per day produced marked cessation of all symptoms, and restoration of cognition. The patient has been stable on selegiline at 40 mg per day for the past year.

## Case report

The patient, a 72-year-old Caucasian male presented at our clinic with complaints of poor motivation, memory problems , insomnia, restlessness, tremor and difficulty with walking. At the time his medications included L-dopa, amantadine, memantine and paliperidone. He was a widower and lived alone. He did not endorse any prior history of neurological problems, strokes, psychiatric disorders or medical ailments. He did have a history of alcohol abuse, but stopped drinking completely approximately one year prior to his visit, at the recommendation of his primary care physician.

He initially presented to his primary care physician with heightened concerns about his difficulties with his memory. He also endorsed poor motivation and inability to "carry on with life". He lived alone, having lost his wife 6 years earlier. He did not have much contact with his extended family, and no longer enjoyed leisure activities including his daily game of golf. A Mini-Mental Status exam administered at the time indicated mild to moderate cognitive impairment. He was given a trial of sertraline 25 mg daily for 2 months with no improvement. This was subsequently increased to 50 mg daily for another month, at which he time he reported further deterioration in his memory and increased restlessness. Paliperidone 3 mg at

night along with donepezil 10 mg daily was then added to his medication regimen. At follow-up he was found to have increased problems with recent and remote memory, motivation, with anhedonia, poor sleep, tremor, mild cogwheel rigidity and a festinant shuffling gait. Lab testing done on the patient including a CBC, CMP, thyroid profile, liver profile, serum folate, B12, vitamin D 3, RPR levels and urine toxicology were all normal. The patient was then referred by his primary care physician to a neurologist for further evaluation and treatment. He was subsequently diagnosed with Parkinson's disease and placed on L-dopa 300 mg daily increased over 2 months to 600 mg daily. No improvement was noted, at which time donepezil was discontinued. Amantadine 200 mg daily was added to his medication regimen, along with memantine 20 mg daily. Surprisingly he was continued on paliperidone increased to 6 mg daily. He was noted to be markedly confused, with further deterioration in his memory, mood, motivation and sleep. Further testing done included repeat liver functions and Serum copper/ceruloplasmin levels which were normal. An MRI of the brain showed mild generalized atrophy, and an EEG which was normal.

He opted to seek a second opinion on the recommendation of his primary care physician, and presented to our clinic with significant complaints of confusion, memory impairment, loss of interest in life, restlessness, difficulties with performing daily activities of living, insomnia and problems with his gait . Upon first examination he was noted to be mildly sedated, a little confused with a monotonous voice. He was fairly well oriented but with significant disturbance in immediate, recent and remote memory. He showed little interest in cognitive testing and a Mini-Mental Status exam indicated 16 out of 30 points. His affect was flat and expressionless and his mood was apathetic and depressed and congruent with his affect. His thought processes were somewhat disorganized and tangential. He was not delusional. He did not exhibit any hallucinations. His insight and judgment appear to be compromised.

Neurological examination revealed no clear deficits in his cranial nerves II through XII. His muscle tone short symmetrical mild cogwheel rigidity. His reflexes were bilaterally depressed. Babinski was negative.

He exhibited a mild pin-rolling tremor. He walked with slow shuffling steps and had some difficulty with his balance. Romberg test was negative and there was no evidence of cerebellar dysfunction.

With his full informed consent his medications were tapered off and discontinued over 2 weeks. After careful deliberation regarding a robust SSRI retrial vs Selegiline, he opted for treatment with Selegiline. He was placed on a tyramine free diet, and advised to maintain complete sobriety with his use of alcohol, which he had stopped completely approximately 6 months prior to his visit with us. He was then placed on selegiline 20 mg daily and followed up in 2 weeks with no significant improvement in his cognition or mood. It was noted that his affect was more appropriate, and his rigidity and tremor had decreased. Selegiline was increased to 40 mg daily and further follow up visit arranged in 2 weeks with no significant changes. Subsequently he was placed on selegiline at 60 mg daily and seen in another 2 weeks. At that time he was noted to be quite alert and active and cheerful. His cognitive functioning seemed to have returned to normal with a score of 28 out of 30 on the Mini-Mental status examination.

Cogwheeling rigidity, tremor and shuffling gait had disappeared. He was sleeping much better and showed good motivation. He continued to maintain sobriety, was able to perform all activities of daily living, and develop a more social lifestyle including returning to his leisure activities, especially golf.

In follow up at 1 month intervals for the next 1 year he continued to be stable on selegiline, reduced to 40 mg daily with complete sobriety from alcohol and no other medications.

## Discussion

This case illustrates the difficulties faced by clinicians with stereotyping older patients with a recognizable neurological disease. The patient's major depressive disorder was misdiagnosed with dementia due to inadequate trial of sertraline, followed by side effects induced by paliperidone and donepezil. Unfortunately, his symptoms then appeared to be more consistent with Parkinson's disease due to adverse side effects of his medications. Treatment with L-dopa, amantadine, memantine produced no benefit. Discontinuation of all the medications followed by the judicious use of high-dose selegiline with a precaution of tyramine free diet

produced significant resolution of all his symptoms and an ability to return to "normal functioning" and enjoyment of daily life.

**Conclusion**

Clinicians should be very aware of the need to do a thorough medical examination and history taking to make a proper diagnosis in our ever-increasing geriatric population. Inadequate trial of a medication, in this case sertraline, can lead to a wrong diagnosis of dementia, further compounded by another wrong diagnosis of Parkinson's disease due to iatrogenic side effects of medications. The term "pseudodementia" may not adequately describe a medical illness, but is clearly a syndrome or condition encountered by us, the proper diagnoses and treatment of which, can rapidly restorative patient to normalcy and reduce overall morbidity.

Statements

**REFERENCES**

1 Post F. The management and nature of depressive illness in late life: a follow

through study. Br J Psychiatry. 1972;121:393-404.

2 Murphy E. The prognosis of depression in old age. Br JPsychiatry. 1983;121:

393-404.

3 Knoll J. Selective inhibition of B type monoamine oxidase in the brain: a drug

strategy to improve the quality of life in senescence. In: Weverling JA, ed.

4 Georgotas A, Friedman E, McCarthy M, Mann J, Krakowski M, Siegel R, Ferris

Shoulson I. Deprenyl and tocopherol antioxidative therapy of parkinsonism

5 Elsworth JD, Glover V, Reynolds GP, Sandler M, Lees AJ, Puapradit P, Shaw

KM, Stern KM, Kumar P. Deprenyl administration in man: a selective monoamine oxidase B inhibitor without the 'cheese effect.' Psychopharma

6 Dobie DJ. Depression, dementia, and pseudodementia. Semin Clin Neuropsychiatry. 2002;7:170-186.

Lamberty GJ, Bieliauskas LA. Distinguishing between depression and dementia in the elderly: a review of neuropsychological findings. Arch Clin Neuropsychol. 1993;8:149-170.

6 Weytingh MD, Bossuyt PM, van Crevel H. Reversible dementia: more than 10% or less than 1%? A quantitative review. J Neurol. 1995;242:466-471.

7 Clarfield AM. The decreasing prevalence of reversible dementias: an updated meta-analysis. Arch Intern Med. 2003;163:2219-2229.

8 Hejl A, Hogh P, Waldemar G. Potentially reversible conditions in 1000 consecutive memory clinic patients. J Neurol Neurosurg Psychiatry. 2002; 73:390-394.

9 Wells CE. Pseudodementia. Am J Psychiatry. 1979;136:895-900.

Rosenstein LD. Differential diagnosis of the major progressive dementias and depression in middle and late adulthood: a summary of the literature of the early 1990s. Neuropsychol Rev. 1998;8:109-167.

10 Tractenberg RE, Weiner MF, Patterson MB, et al. Comorbidity of psychopathological domains in community-dwelling persons with Alzheimer's disease. J Geriatr Psychiatry Neurol. 2003;16:94-99.

11 Cummings JL, Ross W, Absher J, et al. Depressive symptoms in Alzheimer disease: assessment and determinants. Alzheimer Dis Assoc Disord. 1995;9:87-93.

12 Snowden M, Sato K, Roy-Byrne P. Assessment and treatment of nursing home residents with depression or behavioral symptoms associated with dementia: a review of the literature. J Am Geriatr Soc. 2003;9:1305-1317.