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Major Depressive Disorder with Psychotic Features May Lead to MisDiagnosis of Dementia: A Case Report and Review of the Literature

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Abstract

Major depressive disorder (MDD) with psychotic features is relatively frequent among patients with greater depressive symptom severity and is associated with a poorer course of illness and more functional impairment than MDD without psychotic features. Multiple studies have found that patients with psychotic mood disorders demonstrate significantly poorer cognitive performance in a variety of areas than those with nonpsychotic mood disorders. The Mini Mental State Examination (MMSE) and the Dementia Rating Scale (DRS-2) are widely used to measure cognitive functions in research on MDD with psychotic features. Established total raw score cut-offs of 24 on the MMSE and of 137 on the DRS-2 in published manuals suggest possible global cognitive impairment and dementia, respectively. Limited research is available on these suggested cut-offs for patients with MDD with psychotic features. We document the therapeutic benefit of electroconvulsive therapy (ECT), which is usually associated with short-term cognitive impairment, in a 68-year-old woman with psychotic depression whose MMSE and DRS-2 scores initially suggested possible global cognitive impairment and dementia. Over the course of four ECT treatments, this patient's MMSE scores progressively increased. After the second ECT treatment, the patient no longer met criteria for global cognitive impairment. With each treatment, depression severity, measured by the 24-item Hamilton Rating Scale for Depression, improved sequentially. Thus, the suggested cut-off scores for the MMSE or DRS-2 in patients with MDD with psychotic features may in some cases produce false-positive indications of dementia.

Keywords

major depressive disorder; psychotic features; dementia; pseudodementia; Mini-Mental State Examination; Dementia Rating Scale

CASE PRESENSTATION

Background

Major depressive disorder (MDD) with psychotic features is a distinct type of depressive illness in which mood disturbance is accompanied by either delusions, hallucinations, or both. Psychotic features occur in nearly 18.5% of patients who are diagnosed with MDD.[1] The prevalence of MDD with psychotic features increases with age. Over 20 years of

research suggests that patients with psychotic features are more likely to have treatment-resistant depression compared with counterparts who did not have psychotic symptoms associated with their depression.[2,3] Patients with psychotic depression have a greater number of suicide attempts, longer duration of illness, more Axis II diagnoses, and more motor disturbances than those with psychotic features. It is also important to note that patients with MDD with psychotic features have greater overall functional impairment and higher relapse rates than those without psychotic features.[4,5] In addition, geriatric patients with psychotic depression have been found to have more pronounced brain atrophy, higher relapse rates, and greater mortality compared with geriatric patients without delusions or hallucinations.[6] Earlier research found that cognitive function was significantly impaired in patients with psychotic major depression compared with patients with nonpsychotic MDD and healthy comparison subjects.[7]

The term “depressive pseudodementia” continues to be a popular clinical concept, although it has not been incorporated as an individual nosologic category in any classification system. Depressive pseudodementia has been defined as cognitive impairment caused by depression, usually in the elderly, that to some degree resembles other forms of dementia and is at least partially reversible with treatment.[8] Published reports indicate that clinically depressed patients who present with pseudodementia are at increased risk for “true” dementia as early as 2 years after their initial presentation.[9,10] A recent study investigating the long-term outcome of depressive pseudodementia in elderly patients revealed that reversible cognitive impairment in late-life depression is a strong predictor of ensuing dementia.[11]

The standard of care for treating psychotic depression consists of either combination pharmacologic therapy involving an antidepressant and an antipsychotic, or ECT.[12] Depressed patients with psychosis have a poorer response to monotherapy with selective serotonin reuptake inhibitors (SSRIs) or tricyclic antidepressants (TCAs) than patients with nonpsychotic depression.[13] In the mid-1980s, studies showed that only one third of patients with psychotic depression recovered when treated with an antidepressant agent only, compared with one half of such patients who were treated with an antipsychotic agent only. In contrast, two thirds of patients with psychotic depression recovered when they were treated with either ECT or a combination of an antidepressant and an antipsychotic agent.[14] Moreover, numerous studies have shown that ECT treatments with bilateral or right unilateral electrode configuration can be superior to combination drug therapy in the treatment of psychotic depression.[15] A large multicenter, randomized trial investigated the efficacy of bilateral ECT in nonpsychotic depression versus psychotic depression and found a remission rate of 95% in patients with psychotic depression compared with an 83% remission rate in patients with nonpsychotic depression.[16]

It is very important to note that patients with psychotic depression may be excluded from controlled clinical trials because they may lack the capacity to provide informed consent. Alternatively, patients with severe psychotic, or even non-psychotic MDD, may be excluded from clinical trials because of cognitive impairment as measured by cognitive tests (depending on the study methodology). A survey of recent clinical trials in ECT uniformly showed that a diagnosis of dementia was a criterion for exclusion from study participation.[17-19] This raises the question of how to operationalize the identification of dementia. In addition to a pre-existing clinical diagnosis of dementia, a possible diagnosis of dementia may be based upon a pre-defined cut-off score on a neuropsychologic instrument such as the Mini Mental State Examination (MMSE)[20] or the second edition of the Dementia Rating Scale (DRS-2).[21] Published cut-off scores on the MMSE of 24 and on the DRS-2 of 137 are used to suggest possible global cognitive impairment and dementia, respectively, and are used in clinical ECT investigations to exclude patients.

In this case report, we illustrate how the application of either the MMSE or the DRS-2 in patients with psychotic depression may identify cognitive impairment and suggest that this impairment may be related to the psychotic depression rather than a dementing illness.

Case Description

The patient was a 68-year-old Caucasian woman with a history of MDD, recurrent, severe with psychotic features, as well as anxiety disorder NOS. She presented to the emergency room (ER) with concerns that she could neither speak nor move. She was also obsessed with being unable to swallow or eat. Her husband provided most of the history, but during her initial interview in the ER, the patient was speaking without difficulty. She reported delusions that her "tongue and legs were gone." The patient's husband reported that she had lost 8 pounds in the prior month due to appetite loss and that he "had to force her to eat." The patient's medications at the time of admission included fluoxetine (30 mg/day), aripiprazole (2.5 mg every evening), haloperidol (1 mg/day), lorazepam (1 mg three times a day), and temazepam (30 mg at bedtime). The patient was 5 feet, 4 inches tall and weighed 175 pounds (body mass index of 30.0).

The patient's first major depressive episode (MDE) occurred at the age of 59, and her husband reported that, in the ensuing years, his wife had very few depression-free intervals. Previously unsuccessful psychopharmacologic interventions included sertraline, olanzapine, clorazepate, oxazepam, and trazodone; she had not had ECT. The patient had no current substance abuse or history of substance abuse. She had made numerous suicide attempts, mostly by overdose, for which she was hospitalized on an inpatient psychiatry unit both in 2001 and 2002. Family history was notable for a brother with longstanding depression who had received ECT. Her medical evaluation revealed no abnormalities. No brain imaging was done.

The most recent evaluation of the patient's age-appropriate level of cognitive and psychosocial function occurred approximately 9 months prior to the current episode. At that time, she was still socializing with friends and able to independently cook and balance her checkbook. This euthymic interval lasted for several months before her sudden decline in cognitive function and worsening depression.

At the time of this admission, the patient was considered an appropriate candidate for ECT and was also assessed for participation in an ECT clinical trial. She was started on low-dose venlafaxine (37.5 mg/day) prior to her first ECT treatment, which was to be titrated up to a therapeutic dosage by the end of her ECT treatments. The patient received her ECT sessions three times a week (i.e., Monday, Wednesday, Friday). Her venlafaxine dosage was titrated to 75 mg in the morning and 150 mg in the evening by her fifth ECT treatment. Her MMSE scores and the 24-item Hamilton Rating Scale for Depression (Ham-D_{24}) scores were determined prior to each of her first five ECT treatments.[22] The DRS-2 was also administered to the patient prior to her first ECT treatment.

The DRS-2 is a commonly used test that measures cognitive functions that are affected by frontal-subcortical deficits.[21] The DRS-2 is divided into five subscales that measuring attention, initiation/perseveration, construction, conceptualization, and memory; total and subscale scores are obtained. The estimated administration time of the DRS-2 is between 20 and 45 minutes. Normative data for the DRS-2 in healthy subjects showed that normal total scores on the DRS-2 range from 137 to a maximum obtainable 144, although different normative data have been obtained depending on age and education level.[23] Scores below 137 indicate the presence of a dementia-like illness.

Table 1 shows the patient's baseline values for the DRS-2, MMSE, and Ham-D_{24}, as well as changes on the MMSE and Ham-D_{24} over the course of treatment. The patient's DRS-2 raw score of 120 and subscale scores of 4 on the Age-Corrected Mayo Clinic's Older Americans Normative Studies (MOANS) Scaled Score (AMSS)[24] and 3 on the Age- and Education-Corrected MOANS Scaled Score (AEMSS), taken prior to her first ECT treatment, qualified her for a diagnosis of moderate to severe dementia (Table 1).[21] The patient's initial MMSE total score of 21, assessed prior to her first ECT treatment, also suggested that global cognitive impairment was present. However, after the patient's first two ECT treatments, her MMSE score increased to 25. After two additional treatments, her MMSE score increased to 28, indicating a likely false positive initial determination of global cognitive impairment as the etiology of her decreased functional and delusional state. The patient's energy, appetite, and functional state markedly improved after her first two treatments. Her expressions of nihilistic delusions were also markedly decreased in frequency. Her mood and outlook continued to improve over the course of her first four ECT treatments, as reflected by continued decreased depression severity as determined by the Ham-D_{24}.

Discussion

The MMSE and DRS-2 are routinely used by clinicians and researchers to determine global cognitive function and establish operational criteria for excluding or including patients with dementia in controlled clinical trials.[20] Previous studies have found that global cognition and instrumental activities of daily living (IADL) can both be impaired in severely depressed patients.[25] In contrast to younger patients with severe depression, older adults with severe depression are more likely to display significant cognitive impairments associated with depressive symptomatology.[2] These cognitive changes can contribute to the severity of psychiatric symptoms and disability that older depressed patients face and likely reflect compromise of certain neural circuits, directly linking cognitive impairments to late-life depression.[26]

Based on the case report described here, we propose that performance on the MMSE and DRS-2 should not be the only measures used to determine if a patient with MDD with psychotic features also has possible dementia, which will result in exclusion from a clinical trial. We have described the therapeutic value of ECT treatment in a patient with psychotic depression whose initial scores based on standardized cut-off values on both the MMSE and DRS-2 categorized her performance in the moderate to severe dementia range. The severe depression and psychotic state of the patient described in this case report would have incorrectly been attributed to cognitive deficits associated with dementia if the MMSE and DRS-2 instruments were interpreted solely based on accepted manualized cut-off scores specific for each instrument. Thus, further research is warranted regarding performance on the MMSE and DRS-2 measures in patient cohorts with MDD with psychotic features to determine their psychometric properties and appropriate scores to define normal and abnormal performance.

COMMENTARY

David A, Kahn, MD

Wagner and colleagues describe an elderly patient with psychotic depression who met screening criteria for possible dementia based on low scores on the MMSE and DRS-2, but who improved after a course of ECT to the point where her MMSE was almost normal (28 out of a possible 30). The DRS-2 was not repeated, which would have been of interest. Their article makes the focused point that, if we use standard screens for dementia in patients who have psychotic depression, we should take the cutoff scores with a grain of salt. This is the case whether we are screening for suitability for inclusion in research or to make a clinical

diagnosis of dementia. The authors discuss the phenomenon of state-specific, reversible cognitive impairment in severe depression, known as pseudodementia, a common finding in elderly patients with psychotic depression. While a possible harbinger of future real dementia, "pseudodementia" is not the same thing—a key point in our education of patients and families who fear worsened cognition from ECT.

Cognitive effects of ECT on retrograde, autobiographical memory are widely known, so it may come as a surprise to patients, and even to ourselves, that certain aspects of cognitive functioning may improve with ECT treatment. Perhaps the best known public examplar of this phenomenon was the great pianist Vladimir Horowitz, who suffered from crippling depressions that left him unable to perform: "My octaves are no good now; they used to be, but not now."[27] (p. 387). As described in numerous biographies, he returned to the stage triumphantly following courses of ECT in the 1960s and 1970s.[27] His mind recovered a repertoire of over 400 pieces; a testament to the net benefit of ECT to restore, not destroy, brain function while alleviating depression. It was amazing to see Horowitz internationally televised from Moscow a decade later, twirling through Mozart and Rachmaninoff.

A number of studies have evaluated cognitive effects of ECT in detail. At least two meta-analyses have aggregated these results, useful summations since the individual studies are small and vary widely in their testing approaches to fine-grained distinctions between different aspects of cognition such as encoding, learning, retention, and retrieval, as well as treatment variables such as modality, energy dose, and waveform.

First, in 2010, Semkovska and McLoughlin[28] pooled data from 82 studies of patients 18 years of age and older. They included studies that provided at least one reported mean and standard deviation from standardized cognitive testing or a test of significance of difference within subjects, as well as measurements both pre- and post-treatment. They grouped data into relatively homogeneous pools, and created definitions for time course of post-ECT recovery, dividing it into subacute (0–3 days after complete of ECT course), short-term (4-15 days), and long-term (> 15 days).

The results of this study were as follows. Global cognitive status as measured by the MMSE was slightly impaired subacutely, but improved thereafter over baseline. Processing speed was mildly impaired subacutely, recovered to baseline short-term, and then improved over baseline long-term. Attention and working memory (digit span forward and backward, mental control, spatial span) were unchanged or slightly improved. Verbal memory (word lists, story memory, paired associates) showed subacute impairment but at long-term was showed slight improvement. Visual memory for figure reproduction recall showed small subacute impairment, but improvement over baseline at long-term follow-up. Executive functioning tests included the Trail Making Test Part B for set-shifting, the Stroop Color-Word condition for mental flexibility in speed and quality of performance, and the Semantic and Letter Fluency test for organizing thinking. These tests showed medium to large subacute impairment, recovery of baseline in the short-term and either maintenance of baseline performance or small to medium improvement in the long-term. Vocabulary and IQ, measured in a few studies as indicators of overall intellectual ability, were unchanged. The authors also analyzed variations in how ECT was administered. Not surprisingly, electrode placement was a factor, with bilateral (bitemporal placement) ECT producing greater subacute and short-term impairment compared with unilateral ECT in verbal and non-verbal recall. At the same time, bilateral ECT was associated with greater improvement over baseline than unilateral ECT in MMSE short-term and one test of verbal learning long-term. Differences in waveform and frequency of administration did not appear to influence cognition. The authors concluded that ECT caused significant impairment in the first few days after treatment, but that, compared with baseline, these deficits resolved during the next

2 weeks, and some functions actually improved over baseline after that. There were no persistent cognitive deficits resulting from ECT beyond 15 days. The authors noted that it is well established that major depression itself is associated with cognitive deficits. After ECT, some baseline deficits persisted, while some improved; none worsened.

Tielkes et at.[29]conducted a meta-analysis of ECT's cognitive effects in the elderly, evaluating 15 studies performed between 1980 and 2006 in patients 55 years of age and older that included at least one instrument for cognitive measurement before and after treatment. A few of these studies were also included in the Semkovska and McLoughlin analysis, but many were not due to less rigorous data collection. Methods of measurement—timing and instruments—varied widely, as did exclusion criteria of patients with known cognitive disorders. Most of the studies sused only the MMSE. At baseline, most studies reported mild to moderate cognitive dysfunction due to depression; all showed improvement in mood after ECT.

Results within 2 weeks post-ECT showed that global cognitive functioning improved in patients who had demonstrated cognitive impairment or dementia pre-ECT, defined as MMSE under 24. However, in patients with a pre-ECT MMSE of 24 or higher, cognitive function was stable pre- and post-treatment. The memory subscale of MMSE declined but other subscales improved or stayed the same. Older patients who had had more previous ECT treatments were more vulnerable, and bilateral treatment was more likely to impair memory than unilateral treatment. At longer term follow-up of up to 1 month, the unilateral group showed a trend toward global improvement. One study showed significant improvement in naming, learning, and delayed recall.[30] Another showed that ECT improved speed of processing, memory, and perception; improvement in depression was associated with improvement in verbal learning memory, processing speed, and executive functioning.[31] The meta-analysis showed that global cognitive functioning was largely stable during maintenance ECT, but that there were some focal decreases, particularly in verbal fluency, during the week after each treatment.[29]

An interesting study by Bayless and colleagues in 2009[32] evaluated cognitive function before ECT compared with 2 to 3 weeks post-ECT in 20 patients with psychotic depression. Mean ratings of depression, positive symptoms, and negative symptoms all improved markedly. Average ratings of cognitive function also improved. While 30% of the sample qualified as impaired on the testing battery used in this study pre-ECT, only 10% were impaired afterwards. Cognitive function improved significantly on many subscales, most notably those related to attention and language. Interestingly however, linear regression analysis showed that cognitive improvement correlated most with improvement in negative symptoms and not with change in depression or psychosis, suggesting that ECT's effects on cognition may involve areas of the brain apart from those directly related to the symptoms we think of as core features of psychotic depression.

Discussions by some authors mentioned uncertainty as to whether ECT leads to cognitive improvement by inducing remission from depression, or by specific cognitive-enhancing effects on brain function apart from antidepressant activity. As far as I am aware, there is no meta-analysis comparing cognitive function during and after depressive episodes that remit with medication, ECT, and placebo, which would be a good test of modality-specific effects on cognition, compared with disease-state effects.

Both of the meta-analyses as well as the individual studies cited above did not address the most persistent complaint about ECT, long-term retrograde amnesia for personal memories prior to the course of treatment. Research in this area has been flawed by lack of controls for autobiographical memory—what is the "normal" rate of forgetting personal information?

Recent studies have used tests of impersonal verbal and visuo-spatial information taught shortly before ECT and retested later, and have compared results with those of matched controls without depression. Using this technique, O'Connor et al. were able to show that retrograde memory, but not anterograde memory, was somewhat impaired.[33] The study was not designed to evaluate long-term personal memory or unilateral/bilateral differences. It would have been interesting if the researchers had included a second control sample of depressed patients who were receiving medication instead of ECT.

In a large study comparing waveforms and modalities, Sackeim and colleagues evaluated both recently taught information and longer term personal memory for public events.[34] They showed that an ultra-brief pulse stimulus (0.3 millisecond), given unilaterally, resulted in far less retrograde memory loss and subjective distress than bilateral standard brief pulse (0.15 millisecond) or bilateral ultra-brief pulse ECT—not a surprising result. However, the unilateral ultra-brief pulse stimulus was also markedly less injurious to long-term and recent retrograde memory than unilateral ECT given with standard brief pulse stimulus, an important new finding about minimizing side effects within the unilateral modality. Differences persisted for the 6 months of follow-up. Older patients, and those receiving more treatments, consistently performed more poorly in all conditions.

The bottom line is that global and granular measures of present-state cognitive functions, including the ability to learn and utilize new material, improve in patients who receive ECT, including the elderly and those with psychosis. Retrograde memory, less often assessed in formal studies, has been shown to be impaired to varying degrees depending on the laterality and waveform as well as the age of patients and extent of treatment. In counseling patients and families about what to expect from ECT, and in obtaining their informed consent, we can convey not only optimism about the effects of ECT on depression, but also on many aspects of day-to-day cognitive functioning that have been weakened by depression. This optimism is tempered by the possibility of some loss of prior memory, which can be reduced by using the most modern technique of ultra-brief pulse, unilateral stimulus. The case report by Wagner and colleagues illustrates an outcome in which the patient passed from "pseudodementia" to normal anterograde global cognitive functioning over the course of ECT. A review of available evidence happily suggests that this outcome is the rule, not the exception.

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Table 1

Scores on MMSE, DRS-2, and Ham-D_{24} over the course of ECT

	Pre ECT Session				
	1	**2**	**3**	**4**	**5**
MMSE	21	22	25	28	28
DRS-2	120 (raw score) 4 (DRS-2 AMSS) 3 (DRS-2 AEMSS)				
Ham-D_{24}	45	43	41	34	18

ECT = electroconvulsive therapy, MMSE = Mini Mental State Examination, DRS-2 = Dementia Rating Scale, Second Edition, $HRSD_{24}$ = 24-item Hamilton Rating Scale for Depression; AMSS = Age-Corrected Mayo Clinic's Older Americans Normative Studies (MOANS) scaled score, AEMSS = Age- and Education-Corrected MOANS scaled score

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