Conversion pseudodementia in the elderly: A review of the literature with case presentation

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INTRODUCTION
The term 'pseudodementia' is widely used in the literature to describe a syndrome that presents with cognitive impairment and other symptoms of dementia. A more careful estimation of the disorder and its subsequent course suggests dementia and pseudodementia have different origins. The neuropsychological impairment of pseudodementia is considered reversible and there is no apparent primary neuropathological process that leads to the genesis of this disturbance.

The use of the term ‘pseudodementia’ is not accepted by all authors. Some consider that the term provokes confusion and lacks diagnostic value. There is much concern about the clinical picture of pseudodementia in certain patients suffering from depression, particularly in old people. The fact that severe affective disorders could lead to cognitive impairment has been known from the 19th century. The symptoms of cognitive impairment that develop during the course of a depressive disorder are attributed to the decreased concentration and inattention of patients. It is expected that the symptomatology of pseudodementia remit with appropriate treatment and the resolution of the depressive illness. Consequently, some authors suggest an empirical trial of antidepressant medication or the use of electroconvulsive therapy (ECT) in cases in which no sufficient evidence exists to make a positive diagnosis of dementia. Interestingly, there is some evidence that elderly patients with depressive pseudodementia are more likely to develop irreversible dementia during follow up than non-demented elderly depressed patients.

Depressive symptomatology is also likely to be present in prodromal stages of Alzheimer’s disease (AD) and in other types of dementia, either as a demoralized reaction of a patient who understands that he/she suffers from a major illness or as a result of a common pathophysiological mechanism of both conditions. Recent studies support that when depression develops as part of the brain changes associated with AD, it may further accelerate the progression of the neuropathological changes in AD.

Depression in patients with dementia increases morbidity, contributes to excess disability and subjective suffering, family burden and premature institutionalization. Finally, in some cases, the two conditions coexist, so it is possible that a patient with a history of depression will develop dementia. In fact, there is recent evidence that depressive disorders are risk or even predisposing factors for the development of AD and other dementia disorders.
Despite the emphasis placed on depression as a cause of pseudodementia, Kiloh, in an excellent study in 1961, underscored the existence of many other mental disorders related to the symptomatology of pseudodementia. Apart from depression (both endogenous and reactive), Kiloh recognized Ganser syndrome, delirium, mixed manic episode and paraphrenia as underlying disorders in cases of pseudodementia. He also pointed out the existence of amenable hysterical mechanisms in certain cases.

Wells, in a study of 10 patients, denoted the existence of many different disorders that can be revealed as pseudodementia. Apart from depression, Wells observed that post-traumatic neuroses and personality disorders are related to the clinical picture of pseudodementia. He also proposed certain criteria for the differential diagnosis from organic dementia. According to Wells, a previous history of mental disease, short duration and the rapid progression of symptoms, subjective complaints of cognitive deficits, ‘don’t know’ answers, variable and inconsistent performance and apparent cognitive dysfunction are characteristic of pseudodementia.

The term ‘conversion pseudodementia’ was introduced by McEvoy and Wells in 1979 for the description of a case of a 44-year-old woman with cognitive impairment. Her symptomatology was first attributed to presenile dementia. During her hospitalization, careful clinical observation and evaluation revealed incongruity between her behavior in unstructured situations and conversations and her performance on formal mental state evaluation. This observation, in combination with the re-establishment of cognitive function, led the authors to formulate the diagnosis ‘pseudodementia due to conversion reaction or conversion pseudodementia’. In fact, it was Wernicke, as cited by Bulbena and Berrios, who first used the term ‘pseudodementia’ to refer to ‘chronic hysterical states mimicking mental weakness’.

However, in everyday clinical practice, extensive laboratory tests, neuropsychological assessment and brain imaging are required to exclude a reversible cause of dementia symptoms. It is known that a number of conditions, such as drug administration, metabolic disturbances, B₁₂ vitamin insufficiency, normal pressure hydrocephalus and brain lesions are accountable for the clinical picture of dementia in certain cases. It has been reported that potentially reversible causes make up 13.2% of all cases of dementia. Depression accounts for almost 5% of dementia syndromes. Appropriate treatment of these patients is expected to result in cognitive improvement. When a thorough clinical and laboratory evaluation is completed, and no apparent reversible organic cause has been detected, the clinician should consider an underlying mental disease as the cause of cognitive impairment.

In younger subjects who present symptomatology compatible with dementia, suspicion of the existence of a reversible cause is easier to be placed, because the incidence of presenile dementia is low. Diagnosis in the elderly can be difficult, because they are much more likely to suffer from dementia. In both age groups, a thorough clinical evaluation is required before a mental disease is considered to be the underlying cause for the symptoms of dementia. In the elderly, clinicians should always consider depression as a cause of dementia symptoms and bear in mind that atypical presentations are common in this age.

**REVIEW OF THE LITERATURE**

In order to review the literature on conversion pseudodementia, we performed a search of the Medline, PsychINFO and EMBASSE databases. We used pseudodementia, conversion pseudodementia, hysterical pseudodementia, hysteria and conversion disorder as key words for our search. This review of the literature from 1961 to 2004 revealed only a few reported cases of pseudodementia as a conversion reaction. In the case of old patients, the most complete recording has recently been published by Hepple. This author studied 10 patients, of whom six were old, with a mean 13 year follow up. The patient sample was collected prospectively over a 12 year period of clinical work. Hepple recognizes progressive cognitive impairment, regression and patients’ dependency on their carers, beginning in late middle or earlier old age, as basic characteristics of conversion pseudodementia syndrome. None of these cases could be given a clear diagnosis of organic dementia, delirium, depression or psychosis. All patients had a long period of survival from the beginning of their symptoms. Hepple concluded that the personality of patients was characterized by borderline and narcissistic traits and perfectionism, whereas in their past psychiatric history there was a tendency for depressive symptoms.
Another five cases have been reported by Kirby and Harper. These authors suggested that the hysterical (conversion) pseudodemented behavior of patients is a result of an adjustment or developmental crisis. They considered that the changes that accompany aging, such as physical, cognitive and environmental changes, can cause severe stress to some old people. When problems are beyond the resources and capabilities of these people, it is possible that hysterical reactions arise as a result of maladaptive functioning. There is also one case reported by Good that assembles certain typical characteristics of conversion pseudodementia. Good described the case of a 69-year-old woman with a sudden onset of confusion and disorientation. Extensive clinical and laboratory investigation revealed no evidence of organic disease and the patient’s symptoms improved significantly after 3 weeks of hospitalization, without any specific treatment.

The only effort to estimate the incidence of conversion pseudodementia until now was made by Liberini et al. in 1993. These authors studied a sample of 467 patients and found six cases of cognitive impairment attributed to conversion pseudodementia. Over a 3 year follow-up period, the diagnosis was confirmed in five of these cases. According to the authors, cognitive impairment may represent a new type of conver- sive (hysterical) syndrome of old patients in modern societies that tends to replace the classic picture of symptoms of bodily conversion.

In all cases, the diagnosis of conversion pseudodementia was made after exclusion of other causes and was confirmed by a follow-up period of at least 1 year. Researchers agree that the duration of the syndrome is a basic diagnostic element. The syndrome may continue for considerably longer than organic dementia, which would lead to death; in contrast, cognitive impairment may soon improve significantly, which, again, is not compatible with organic dementia. The discrimination from pseudodementia due to depression is made on the basis of the absence of a depressed feeling, a poor response to antidepressant medication that is often administrated to patients and the continuous, constant course of conversion pseudodementia, or in contrast, a spectacular and rapid improvement that cannot be explained as a natural course of a depressive episode.

With regard to prognosis, there are differences between the reported cases. In some patients, there was significant improvement of cognitive function, whereas others lost their functionality and became dependent on their family, relatives or other caregivers. Furthermore, the number of cases is small, so definite conclusions cannot be made at present. Hepple concluded that prognosis appears to be poor and inappropriate hospitalization or institutionalization may lead to further retreat into pseudodementia and dependency.

The cases of pseudodementia in younger patients resemble those of old people; however, in these cases there are fewer diagnostic difficulties because presenile organic dementia is infrequent in younger patients. Thus, the appearance of such symptoms in a young person will raise the suspicion of the existence of pseudodementia. The prognosis is usually better in younger patients than in old patients; however, in some cases, the syndrome runs through a chronic course and leads patients becoming utterly dependent on their caregivers.

Apart from these formal cases of conversion pseudodementia, there are other cases that exhibit some of the characteristics of the syndrome without cognitive impairment. Padoani and De Leo reported cases of three patients with serious and persistent regressive behavior that resembled dementia, without evidence of cognitive decline. All three patients were less than 65 years of age and the follow-up period was 5 years. The initial diagnosis was AD and physical examination and laboratory tests revealed no other medical condition. The onset of symptoms was acute, the development rapid and the syndrome resulted in the loss of autonomy and the complete dependence of the patients on their family, relatives or other caregivers. Neurological symptoms were also present, such as slowness, impaired gait and incontinence. Although at the onset of the syndrome depressive and anxious symptomatology was marked, during the complete development it was replaced by abulia and dysphoria, even aggressiveness. The result was affective and social withdrawal. The patients’ scores in the Mini-Mental State Examination (MMSE) remained 30/30. The authors suggested certain common points between conversion pseudodementia and the syndrome they described. They considered the appearance of secondary gain (i.e., other people’s support and care, avoidance of responsibility, undeserved compensation) and the existence of stressful, triggering life events prior to onset to be common in both...
disorders. They also suggested that both disorders were the result of maladaptive adjustment strategies in cases of individuals with insufficient coping capacities.28

Eastwood and Kennedy reported four cases of old women with behavior disturbances and fluctuating psychomotor activity, from excessive activity to withdrawal and drowsiness.29 Core symptoms were dysphoria, anxiety, confusion and approximate answers. Estimation of cognitive function during the first days of hospitalization revealed cognitive impairment. All patients received antidepressant medication, but in only two patients did the symptomatology improve. In the long term, only one patient maintained her function, whereas the others required permanent care. The authors concluded that the patients exhibited a form of pseudodementia that was probably related to personality and marital relationships. They considered that the prognosis was unfavorable, at least in the short term.29

Howells and Beats suggested that situations of emotional dependency can be revealed in the form of such an unusual clinical picture and underscored the role of deficit premorbid personality.30 These authors considered that clinicians should make a full etiological inquiry that was not only organic, but also psychosocial in order to understand the nature of the atypical symptomatology.30

With respect to treatment, researchers consider that hospitalization or institutionalization is not advisable because it may worsen the symptomatology of regression and may increase the dependency of old patients, while simultaneously preventing sufficient clinical evaluation.22,28,31 However, even in suspected cases of amenable depressive disorder, antidepressant medication should be administered.1 Hepple suggests a cognitive and analytic psychotherapeutic approach that will at least prevent or moderate the development of the syndrome, which, in full growth, does not improve with therapeutic interventions.22

As for the prognosis of pseudodementia, researchers agree that it is determined by the course of the amenable mental disorder, because it is a heterogeneous syndrome that may be a clinical manifestation of several mental disorders.7,32 With regard to conversion pseudodementia, it is expected that the syndrome will have the course of a conversion disorder. The data presented in the literature globally report a poor prognosis, with persistent symptomatology for years. The prognosis is better in cases of coexisting depressive symptoms, whereas the coexistence of personality disorder is considered to be an aggravating factor.33 However, until now, there are conflicting results reported in the literature regarding the outcome of the syndrome in the long term. Obviously, systematic recording and investigation of all new cases of conversion pseudodementia are required to make the identification of certain prognostic factors possible and to extrapolate conclusions regarding the long-term course of the syndrome.

CASE PRESENTATION

A 73-year-old male was admitted to our unit presenting with symptoms of sleep disturbance, psychomotor agitation and aggressiveness, mainly against his wife. The onset of the symptomatology was acute and the development rapid, within 20 days.

According to his medical records, the patient had been hospitalized in our unit twice, once 5 years and then 6 months ago, for the management of two distinct episodes of masked depression. His symptomatology included psychomotor anxiety and many unspecified physical complaints with an intense preoccupation with these complaints. These symptoms had been more prominent than the depressed mood itself.

During both periods of hospitalization, a thorough physical examination, extensive laboratory tests, full blood count and computed tomography (CT) of the brain had been performed, revealing no pathological findings. The patient was prescribed mirtazapine at a dose of 30 mg/day and, in both cases, his symptoms underwent complete remission. The patient had always been compliant with his treatment. At the time of current admission, the patient was continuing to take mirtazapine.

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According to his history, his behavior had been characterized by vague hypochondriac complaints. His personality traits included insistence, meticulousness and rigidity. The family background of the patient was of considerable interest, because his older daughter suffered from psychosis and occasionally partook in serious self-destructive behavior such that she had been hospitalized several times in the past in our unit. As a result of the severity of her disorder, she had been placed in an institute.

During his own hospitalization, the patient’s behavior was characterized by withdrawal and regression. He used to pass his hours in bed groaning, looking
around as if he was lost without speaking or answering any questions, showing that he had difficulty understanding anything that was said. An interview with him was impossible. He kept saying that he wanted to pass through ‘general examinations’. He did not cooperate with blood sampling. The patient suffered sleep disturbances and was eating with difficulty, under pressure from his wife. After a few days, he became spatially disoriented. He left the unit twice and was found wandering around the hospital and in other units without being able to find the way back or to ask for help. On another occasion, the patient was retained by other patients and by the personnel of the unit while he was attempting to leave. Sometimes his behavior was disorganized, as when he lay down on a stretcher at the end of the corridor, far away from his ward, stubbornly refusing to get up. On another occasion, he entered the office saying that he was in his house and began to take away many of the objects in the office. When his wife and staff tried to explain to him that he was wrong, he simply sat for a while in a chair, then turned around and left. He caused damage in his ward in an outbreak of rage, without any obvious reason for his rage. Usually, the patient was aggressive towards his wife, despite the fact that he was having difficulties in doing anything without her encouragement. He was completely dependent on her.

Physical and neurological examination of the patient did not reveal any pathological signs, so a new CT scan of the brain was not performed. All routine blood tests, including serum levels of vitamin B12 and folate and tests for thyroid function, were normal. The MMSE was performed to determine cognitive function and the patient’s score was 0/30. The patient did not cooperate during the examinations: he was looking around as if he was lost and was unable to say a word. The impression given was that his output did not correspond to real cognitive decline; neither, however, was he considered to have pseudodementia due to depression. During that particular hospitalization, the patient’s symptomatology was substantially different from that that had led to the diagnosis of masked depression.

We decided to increase the dose of mirtazapine to 60 mg/day, even though the clinical picture did not correspond to depression, in order to maximize antidepressant treatment and to exclude any possibility of pseudodementia due to unrecognized depression. Mirtazapine had been well tolerated by the patient and had been very effective in the past. No substantial improvement was observed, apart from a small improvement in the patient’s sleep. His behavior continued to be disorganized and communication with him was impossible. After 35 days of hospitalization, the patient was discharged without any changes in the clinical picture at the time of his admission. Because the patient and his wife lived far away from our unit and the mental health services in this area were not developed, we were not able to arrange any psychosocial intervention. In fact, there were difficulties even with the follow-up procedure.

Six months after his hospitalization, the patient was examined again as an outpatient. He came after pressure from his wife because he did not want to leave his house. His behavior had not changed from the time of his hospitalization. He continued looking around as is he was lost and spoke only a little. The descriptions given by his wife suggested that all this time his behavior was characterized by regression. He was eating only under pressure and occasionally he was unable to take care of his personal hygiene. He was displaying psychomotor excitement, but could sleep during the night. He was unable to get out of the house and was usually withdrawn with disorganized behavior and weak communication, mostly negative (e.g. he often displayed motiveless resistance to his wife’s instructions and attempts to help him with self-care). The patient was completely dependent on his wife, although at certain times he was aggressive towards her. The symptomatology was like that observed during his hospitalization, despite the continuous administration of mirtazapine, 60 mg/day. After a further year, the patient was re-examined as an outpatient. His mental state was completely different. He was now orientated in time and space and displayed no aggressive behavior. He was able to care for himself and his wife was happy with his improvement.

**DISCUSSION**

The patient in the case described above displayed certain features of cognitive impairment. During the interview, he appeared unable to understand the questions and kept looking around with a look of query and distress. Moreover, the patient appeared to be spatially disoriented and was found occasionally wandering within the hospital. He was unable
to care for himself. Generally, he was absolutely dependent on his wife, who was with him during the entire period of hospitalization, because he needed her prompting even to eat. From time to time he was agitated and aggressive towards her. Despite this regressive behavior, the impression of the personnel of the unit and the clinicians was that he could accomplish much better that what he was showing. The patient’s symptomatology was of acute onset and rapid progression. All blood tests were normal, as were the physical and neurological examinations; a CT scan of the brain, taken 6 months ago, had excluded any brain lesions. The symptoms did not improve despite treatment with antidepressant medication, which was administrated on the basis of his past history, and his behavior remained unchanged during the period of hospitalization.

According to the principles stated by Wells,15 we considered this patient to be a case of pseudodementia and not a demented case. The patient’s family had been aware of the dysfunction and its severity, the onset of the symptomatology could be dated precisely and the duration of the symptoms was short before medical help had been sought. Moreover, the progression of the symptoms was rapid after an acute onset. Furthermore, there had been a history of previous psychiatric disorder (masked depression in this case), which is common in patients with pseudodementia. The patient had been uncooperative and did not make any effort to perform in the MMSE. Finally, the result was considered not to be a suitable evaluation for real cognitive decline, but rather an indication of poor co-operation of the part of the patient. The reason for this may have been a common psychotic state, probably due to a psychotic disorder such as late catatonia. The term ‘catatonia’ refers to a syndrome characterized by mutism, negativism, rigidity, posturing, stereotypy and staring.34 Catatonia has been associated with several neurological and general medical conditions, mood disorders and schizophrenia.35 Catatonic states have also been reported in cases of dementia with Lewy bodies.36 It has been suggested that catatonia in the elderly can mimic dementia in certain cases.37 Furthermore, delay in identification of the syndrome in the elderly has been associated with adverse outcome.38

It could be argued that certain features of the present case may be applied to the diagnosis of late catatonia. For example, the patient occasionally displayed negative behavior and aggressiveness. Moreover, onset had been acute, thus resembling the onset of the catatonia subtype called periodic catatonia.39 However, the patient did not fulfill the diagnostic criteria for catatonia proposed by Taylor and Fink.35 He did not display immobility, mutism or stupor, nor any of the other core symptoms of catatonia, such as stereotypy, echophenomena, catalepsy, automatic obedience, posturing and ambitendency. Furthermore, the symptomatology did not run the typical bipolar course with hyperkinetic and akinetic episodes, which characterizes periodic catatonia.39 Thus, the clinical picture was considered to be insufficient for the diagnosis of catatonia. In addition, neurological and physical examinations, as well as laboratory tests, did not reveal any general medical or neurological condition that could account for the syndrome of catatonia. Moreover, the patient did not present any psychotic symptoms and there was no history of psychotic disorder.

With regard to the diagnostic probability of depression as an underlying disorder, we have to note that there were no signs or complaints of depressed mood. Furthermore, the attitude of the patient during the MMSE does not support the diagnosis of depression (depressed patients usually answer ‘I do not know’ and they have difficulties concentrating). Depressed patients are likely to complain about their cognitive dysfunction and disability and display marked variability in performance of tasks of similar difficulty.15 Moreover, the poor response of this patient to treatment with an increased dose of an antidepressant of proven efficacy and the significant and persistent regression strongly support a diagnosis of
conversion pseudodementia. In accordance with this diagnosis is the existence of the permanent stressful factor of the serious mental illness of his daughter. Furthermore, as Hepple pointed out, the existence of depressive symptomatology in the past does not exclude a diagnosis of conversion pseudodementia. However, it is worth noting that depression may precede dementia, or become coexistent during the initial stage or the subsequent course of dementia. We have shown that this patient was not depressed or demented and our diagnosis as a case of conversion pseudodementia was confirmed by the subsequent clinical course.

The subsequent course, with marked improvement of cognitive function, excludes any possibility of a misdiagnosis of a progressive dementing disorder. However, it could correspond to the natural course of a depressive episode. As we have pointed out, this was not the case for our patient.

Interestingly, the time from the onset of symptomatology to improvement was just over 1 year. In other reported cases of conversion pseudodementia, improvement was either rapid, within a few weeks, or absent even after many years. From this aspect, ours is a unique case of conversion pseudodementia.

**PROPOSALS**

The incidence of conversion pseudodementia in the elderly is likely to be significant and there are likely to be more unrecognized cases than suggested by the few reports in the literature. It is possible that the syndrome is underdiagnosed. It is also likely that cases of patients with the symptomatology of pseudodementia who are diagnosed and treated as depressed with no response to antidepressant medication are, in fact, cases of conversion pseudodementia. Except in cases of abrupt resolution of cognitive impairment, the diagnosis is often confirmed with the passage of time and this may lead to inappropriate therapeutic interventions. For example, it can result in an inappropriate hospitalization or institutionalization. Moreover, elderly patients may be exposed to the undesirable side-effects of antidepressant medication or ECT. It is known that various drugs can worsen cognitive function, thus provoking diagnostic confusion in certain cases. Finally, precious time for the performance of a suitable therapeutic intervention, such as psychotherapy, can be lost, resulting in the consolidation of the symptomatology, which then becomes more difficult to treat.

It appears that certain criteria for the diagnosis of conversion pseudodementia should be drawn up so that clinicians can consider it as a diagnostic probability in cases of unexplained and persistent non-organic dementia symptomatology. This would allow epidemiological studies to be undertaken to determine the precise incidence of the syndrome and the environmental and individual factors involved in its pathogenesis, as well as an investigation of prognostic factors for long-term outcome. Thus, a better understanding of the syndrome and the application of appropriate treatment will be achieved. In any case, extensive clinical and laboratory investigations should be performed to exclude organic dementia and certain reversible causes of cognitive decline. A detailed evaluation of the patient's history should not be overlooked, paying particular attention to life events. Careful evaluation of the patient's personality may also prove helpful for an accurate diagnosis. Malfunctioning personality traits are frequent in both old and young people and may lead to clinical manifestations that may be misdiagnosed as dementia or as several types of pseudodementia. In diagnostically difficult cases with a background of depressive symptomatology, the trial of a safe and well-tolerated antidepressant is indicated. If there is a resolution of cognitive impairment, this may confirm the diagnosis of pseudodementia due to depression. Clinicians should be very careful in the administration of drug combinations and in the performance of ECT. The pointless exposure of patients to potential side-effects of a treatment should be avoided.

**REFERENCES**

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41 Lantz MS, Buchalter EN. Pseudodementia: Cognitive decline caused by untreated depression may be reversed with treatment. *Geriatrics* 2001; 56: 42–43.