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Pseudodementia twelve years on

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Abstract

This paper reports a longitudinal study of 19 patients diagnosed as having pseudodementia more than a decade earlier. In only one patient was the earlier diagnosis changed to definite dementia and, in this patient, there were strong indicators that such a diagnosis should have been made initially. In a second patient, dementia could not be excluded. The remaining patients did not show evidence of a dementing illness and the courses of the illnesses resembled the primary psychiatric disorders responsible for the pseudodementia. The results validate the clinical utility of the term "pseudodementia".

The term "pseudodementia" has been used to describe a syndrome in which features suggesting dementia are associated with non-organic psychiatric illness.[1 2] The term has no diagnostic specificity[1 3] as it includes a heterogeneous collection of disorders[4] although depressive disorders are most likely to be represented.[2] Yet its descriptive value is recognised widely by clinicians[1–5] who use the term to mean one or more of the following: 1) the impairment in memory, learning, and related cognitive functions is caused by a psychiatric illness; 2) the impairment is likely to be non-progressive and is potentially reversible if the primary illness is treated; 3) either no neuropathological process can be identified, or if such a process exists, it is minor and insufficient to explain the severity of the cognitive deficits.[6]

Opposition to the term has been extensive and has taken various forms. The most common criticism is a conceptual one. Critics[6] recognise that cognitive deficits do occur as a result of depression and some other psychiatric disorders,[7 8] but argue that they represent, in fact, a "true" dementia secondary to the pathophysiology causing the psychiatric disorders.[9] A number of researchers have provided direct or indirect evidence to support the argument that cognitive deficit is an intrinsic aspect of depression[10–13] and schizophrenia[14] and not merely a reflection of poor motivation.[15] These critics also argue that the term "pseudodementia" originated at a time when dementia was described as a progressive, irreversible and untreatable condition.[16] Many early studies of dementia used this definition.[17 18] Contemporary definitions[5 19] of dementia, however, accept the notion of reversibility, thus opening the way for its application to the cognitive deficits of depression. Another recent development is the challenge to the assumption that dementia is always a cortical phenomenon with the suggestion that a "subcortical" dementia may exist[20 21]; many of the features of "pseudodementia" resemble those of subcortical dementia.[6 7 9 22]

Other authors, while not criticising the concept, have argued that the diagnosis of "pseudodementia" is often erroneous and the patients have genuine organic brain disease, perhaps with a superimposed depression that leads to the error.[23–25] In other words, the "pseudodementia" is in fact a "pseudo-pseudodementia".[4] Some of these authors[23 26] demonstrated that even when the depression of depressive pseudodementia had been adequately treated, the dementia did not automatically recede. That depression is common and an underrecognised part of the dementia syndrome has been emphasised by many.[24 27 28] The functional disturbance in so-called pseudodementia, according to these authors, is greater than that expected from the severity of the psychiatric illness alone,[23] and the course of the cognitive change and the severity of the psychiatric disorder have not always shown a consistent relationship.[6] One study[29] suggested that organic lesions were commonly present in patients of pseudodementia. Thus, it is suggested by these critics that the diagnosis of pseudodementia is a trap as it often misses underlying dementia or other organic mental disorder.

Caine, in his influential review of the syndrome,[3] suggested that a longitudinal natural history study of pseudodementia would enhance the understanding of the relationship of behavioural and intellectual abnormalities and indicate the prognostic significance of the diagnosis. If pseudodementia is a condition promoted by an organic diathesis that has escaped detection, long term follow up might delineate such factors. If pseudodementia is mistaken dementia, this would show up at least in the medium term. Alternatively, if it is merely one manifestation of a "functional" psychiatric illness with no organic basis, this would again be borne out by the follow up data.

In this paper, we report a longitudinal study of 19 patients diagnosed as having pseudodementia more than a decade earlier. The definition of pseudodementia used in this study is detailed below, but it essentially refers to a picture of dementia produced by a non organic psychiatric disorder. The term "dementia" in this paper refers to a DSM-IIIR dementia which incorporates definitive or strongly

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presumptive evidence of organic aetiology but does not include irreversibility.

Subjects and method

Between May 1973 and December 1977, the Neuropsychiatric Institute, Sydney, conducted a multidisciplinary study of patients who presented with a provisional diagnosis of dementia. The patients were referred, in the main, by psychiatrists and neurologists and were assessed as inpatients. Their clinical details were recorded in a standardised interview and a number of laboratory investigations were carried out as previously detailed.[30] The Wechsler Adult Intelligence Scale, the Wechsler Memory Scale Form 1, the Graham Kendall Memory for Designs Test, the Benton Visual Retention Test, and a formalised test of parietal lobe functioning were administered. Of the 200 patients assessed in this study, 21 (10·5%) were diagnosed as having pseudodementia, that is, they had significant cognitive deficits which were considered secondary to a non-organic psychiatric illness, usually depression or psychosis. The earlier paper[30] reported 20 patients, but recent re-examination revealed 21 patients. The diagnosis was suspected by Smith and Kiloh[30] if the patients had one or more of the following: 1) depression or psychosis was a prominent feature of the presentation, and/or 2) there was a history of previous psychiatric illness of comparable form followed by complete recovery, and/or 3) the illness was of acute onset and short duration; and there was an absence of abnormality on electroencephalogram (EEG), air encephalogram (AEG) (before the availability of CT) or CT scan or the abnormality, if present, was minor and judged to be not aetiologically significant.

The diagnosis of pseudodementia was confirmed retrospectively for the purposes of this follow up if these patients fulfilled all of the following criteria: 1) evidence of clinically significant disturbance in short-term and long-term memory; 2) evidence of clinically significant intellectual impairment in the form of poor abstraction, poor judgment, impaired reasoning or logical processes, disturbance in other higher cortical functions or significant personality change; 3) occurrence of the memory and intellectual impairment in clear consciousness; 4) a relatively recent onset, a few months to a year previously. If the history was longer, there had been a recent exacerbation leading to the index presentation; 5) strong cross-sectional and/or historical evidence of a "functional" psychiatric illness, such as an affective or schizophrenic disorder, that could possibly account for the current problem; 6) the absence of evidence from the history, physical examination, or laboratory tests of a specific organic factor (or factors) of severity judged to be sufficient to cause the disturbance; and 7) demonstrated or strongly presumed reversibility of the memory and intellectual deficits with appropriate treatment of the psychiatric illness.

Two of the 21 patients in the study did not, on restrospective analysis, have significant memory or intellectual dysfunction (criteria 1 and 2) to justify the diagnosis of pseudodementia. One was diagnosed as having a bipolar disorder, currently depressed and the other as having paraphrenia. These patients are excluded from the follow up study.

Initial patient characteristics

The mean age of the patients was 53 (range 26–64) years with 17 (89·5%) in the age range 45–64 years. Fifteen (80%) were female. Eleven patients (58%) received a diagnosis of depression, three being in the depressive phase of a bipolar disorder. Five (26%) patients had a schizophrenic illness, two (10·5%) had a manic episode, and one (5·3%) had a schizophreniform disorder. The details of the disorders are presented in the table.

Sixteen patients had a long history of psychiatric illness, the remaining three having presented in their first episodes. Two patients (cases 5 and 6) had a history of possible pseudodementia in a previous episode of the illness documented in their records. The index admission at which the diagnosis of pseudodementia was made had a mean duration of six months. Only one patient (case 11) had significant brain disease in the form of a previous cerebrovascular accident and residual left hemiparesis. Two patients had hypothyroidism (cases 2, 4) but were adequately treated at the time of presentation. In terms of family history, one patient's (case 14) father had developed a dementing illness in the fourth decade, although this history was not available until one year later. One patient's (case 1) mother had Parkinson's disease without a history of dementia. Five patients had histories of affective illness in a first-degree relative. Eight (42%) had an abnormal EEG, generally in the form of intermittent non-specific slowing without diagnostic significance, except for case 11 (with past CVA) and case 19 (significance unknown).

All patients were judged to be cognitively impaired on neuropsychological evaluation so that an organic process could not be ruled out on the basis of the results. The patients demonstrated impairment in memory and general intellectual functioning. Three patients (cases 9, 10 and 14) had features that suggested aphasia, apraxia or agnosia. Case 9 had dressing apraxia, dyscalculia, finger agnosia and disorientation in space. Case 10 made a few mistakes on finger recognition and naming tasks and her copying of geometric figures was poor. Case 14 had minor expressive dysphasia and constructional apraxia. Two other patients (cases 5 and 7) had difficulty copying geometric figures.

Follow up procedure

An attempt was made to trace all patients in 1987 by contacting the recorded addresses, the respective family doctors and the referring hospitals. For the eight (42%) subjects who had died in this interval, information was obtained from medical records, death certificates, reports of necropsy examination if conduc-

Table Clinical features of pseudodementia patients and their status at follow up

Case No	Age	Sex	Diagnosis	Total duration of psych illness (years)	Duration of index episode (months)	Family history (1° relative)	Abnormal investigations (neurological)	Duration of follow up (years)	Status in 1987	Final diagnosis	Course of illness
1	45	F	Bipolar depressed	11	12	Parkinson's disease	—	12	Alive	Bipolar disorder, no dementia	Multiple episodes with full remission
2	49	F	Bipolar depressed	29	6	—	EEG	12	Alive	Bipolar disorder	Multiple episodes with full remission
3	63	F	Bipolar depressed	30	6	Affective illness	—	12	Alive*	Bipolar disorder, no dementia	Multiple episodes with one further admission (1986)
4	56	F	Bipolar manic	26	4	Affective illness	AEG	12	Alive	Bipolar disorder, no dementia	Multiple episodes with good recovery
5	50	M	Bipolar manic	30	12	—	—	11	Dead (1984)	Bipolar disorder, no dementia	Further manic episodes. Died of lithium toxicity. NE = no cerebral atrophy
6	57	F	Major depression	10	6	—	—	2	Dead (1978)	Major depression, no dementia	Committed suicide; NE = no cerebral degeneration
7	61	F	Major depression	10	4	—	EEG	6	Dead (1980)	Major depression, no dementia	Depressive episodes. Aspirated during ECT and died. No NE
8	54	F	Major depression	1	1	Affective illness	—	6	Dead (1980)	Major depression, no dementia	Intraoperative death. NE = No cerebral degeneration
9	63	M	Major depression	25	5	—	EEG	2	Dead (1978)	Major depression, possible dementia	One further depressive episode. Possible underlying dementia (? aerteriosclerotic). Died of pneumonia
10	62	F	Major depression**	36	12	Affective illness alcoholism	EEG	11	Dead (1985)	Major depression, no dementia	Died of Ca breast with metastases
11	63	F	Major depression	15	1·5	—	EEG	12	Dead (1987)	Bipolar disorder, no dementia	Three more episodes. Died following second stroke
12	57	F	Major depression	31	10	—	AEG	12	Alive*	Major depression, no dementia	One further episode of depression. Currently no treatment
13	45	F	Major depression	1	12	—	—	12	Alive	Major depression, no dementia	Variably but constantly depressed till 1983 and then improved
14	26	M	Schiz, chronic; acute exac***	4	5	Dementia	EEG	6	Dead (1981)	Huntington's chorea	Progressive deterioration; diagnosis became clear at one year
15	58	F	Schizophrenia, with depression	27	7	—	EEG	12	Alive	Schizophrenia, residual, no dementia	Performance poor but no deterioration
16	50	F	Schizo, chronic, acute exac***	31	5	—	AEG	12	Alive	Schizophrenia chronic, no dementia	Cognitive impairment persistent but non-progressive
17	64	F	Schizo, chronic, acute exac***	27	1·5	—	X-ray skull	14	Alive	Schizophrenia residual, no dementia	Mild to moderate impairment, non-progressive
18	57	M	Schizophreniform disorder	0·1	1	Affective illness	—	14	Alive	Schizophrenia chronic, no dementia	Mild cognitive deficit, non-progressive
19	59	F	Schizophrenia with depression	12	1·5	—	EEG	13	Alive	Schizophrenia residual, no dementia	Improved and remained relatively well

*Refused detailed assessment; **Possible underlying dementia; ***Chronic with acute exacerbation; NE = Necropsy examination.

ted (3 cases) and interviews with relatives. Nine (47%) subjects still alive were admitted for two days for a full medical and psychiatric examination, investigations for dementia including EEG and CT scan of the head, and a detailed neuropsychological assessment which included subtests of the Wechsler Adult Intelligence Scale, Wechsler Memory Scale Form I and tests for frontal lobe functioning, memory and visuo-spatial abilities. The clinical psychologist was blind to the patient's medical and psychiatric history. Information was also obtained from a close relative and the patient's family doctor or psychiatrist about the patient's functioning since the index admission and current status. The remaining two (10·5%) patients still alive refused personal interview with the investigators, and, for these subjects, information was obtained from their doctors and close informants and the discharge summaries from hospitals they had been admitted to since the index admission. A life chart for the course of illness was constructed for each patient and a consensus reached on the final diagnosis between the investigators and the patient's primary physicians.

Results

These are presented in the table. The mortality rate of our patients was 42%. Of those alive at follow up, six (54·5%) were living at home. Excluding case 14, all patients diagnosed as having schizophrenia were alive at follow up.

Of the 13 patients with affective illness, six (46%) were alive after 12 years, the others having died after a mean 7·1 years at an average age of 65·7 years.

Deceased patients
Eight (42%) patients died from the following causes: suicide (1), possible lithium intoxication (1), Huntington's chorea (1), anaesthetic complications (2), cerebrovascular accident (1), pneumonia (1) and breast cancer (1). Review of medical histories, discharge summaries from hospitals and death certificates upheld the primary psychiatric diagnoses in six cases. These showed cognitive recovery with treatment of the primary illness and no evidence of cognitive decline before death. Necropsy examination was conducted in three cases and did not show evidence of cerebral atrophy. One patient (case 14), who earlier received a diagnosis of schizophrenia was rediagnosed a year later as having Huntington's disease as his cognition deteriorated and choreiform movements became more prominent. In the eighth patient (case 9), who died two years after the initial assessment, dementia could not be ruled out. His depression improved with treatment with electroconvulsive therapy (ECT) but mild memory and intellectual impairment persisted and he had apraxia and agnosia. He had a recurrence of depressive illness two years later and died from pneumonia, but a necropsy examination was not conducted. He had clinical evidence of atherosclerosis and a history of myocardial infarction, and it is possible that his recurrent depressive illness overlay a mild dementia resulting in the initial presentation being considered pseudodementia. Both patients 14 and 9 had shown focal cortical signs (apraxia, aphasia or agnosia) at the time of the initial assessment. However, case 10, who had also had such signs, did not show historical evidence of dementia when she died 11 years later although a formal examination of cortical functions was not conducted on her. Cases 5 and 7, who had demonstrated constructional apraxia at the time of the initial assessment, also did not develop dementia.

Living patients
None of the 11 patients alive 12–14 years after the index admission showed evidence at follow up of a dementing illness on clinical or neuropsychological assessment and their investigations such as EEG and CT (excluding the two patients who refused admission) were within normal limits.

Four patients (cases 15 to 18) showed cognitive impairment on neuropsychological assessment but this was of a lesser degree than that recorded at the initial assessment. These four patients had a final diagnosis of chronic schizophrenia, with three living in nursing homes and the fourth alone at home with intensive community support. The fifth patient (case 19) with schizophrenia who refused interview lived at home alone and, according to the report of her family physician, functioned independently.

The patients with affective illness showed little evidence of cognitive impairment and were all independent in activities of daily living. Four lived at home (two alone and two with family) and two in nursing homes. For the patients with affective illness who had subsequent episodes of illness, the available documents and accounts did not suggest that cognitive impairment recurred in these episodes.

Combined group
All patients with a diagnosis of affective illness showed stability of that diagnosis. One patient (case 11) developed a hypomanic episode for the first time in the follow up period. In two patients (cases 9 and 10), an underlying dementing process was suspected at initial assessment and the depressive illness may have led to an exacerbation of the cognitive deficit. In one of these (case 9), dementia could not be ruled out when he died two years later but an 11 year follow up of the other did not suggest a progressive decline.

The patients with a diagnosis of schizophrenia (cases 14–19) also showed stability of diagnosis except for case 14 mentioned above who was finally diagnosed as having Huntington's disease. A review of this patient's records suggested that his presentation was indeed atypical. He was by far the youngest (26 years), had evidence of marked neuropsychological impairment (in particular aphasia, apraxia and agnosia), showed transient choreiform movements and his father had developed dementia in the fourth decade, this history was unavailable at the time of first assessment. Diagnosis was initially difficult because of the presence of a marked disorder of thought form and delusions.

Discussion

The two major indicators of outcome in our patients are the mortality rate and the cognitive status of the survivors. The mortality rate should be considered in the light of reported mortality rates for patients with affective illness, schizophrenia and dementia. Roth,[31] in his study of all mental disorders in the elderly (aged more than 60 years), reported that 14·8% were dead in six months. Hastings,[32] in a much younger (mean aged 39·6 years) group of manic-depressives, reported a mortality of 22·4% in a six to 12 year follow up. Huston and Locher[33] reported a mortality rate of 36% in a 6·5 year follow up of 93 depressives with a mean age of 52 years in the pre-drug era. In two recent long-term prospective studies of depressive illness,[34 35] 23% and 29% were dead 18 and 16 years after initial contact respectively, but these were younger than our patients. The most appropriate comparison to our data is with that of the Huston and Locher[33] cohort, and the mortality trend in that study tended to approach our own. One (8%) of our patients with affective illness died of suicide which is similar to rates reported in the literature.[34 35] Outcome in primary degenerative dementia is poor, with most patients dying within five to seven years of diagnosis[4] although longer

survivals have been shown.[36] Roth[31] found that 60% were dead after six months and 80% within two years. In a study by Heston *et al*[36] dementia patients under 49 survived for a mean of seven years and those aged 55 to 74 for 8·5 years. Considering these reported studies, our patients had a mortality pattern more like that of depressives than those with dementia. Our schizophrenic patients were all alive at follow up with the exception of one whose diagnosis was changed to Huntington's disease. These findings suggest that the initial diagnosis of "pseudodementia" was correct in most instances.

The absence of dementia in the survivors again validated the initial assessment. Although cognitive impairment persisted in four patients with schizophrenia, they did not fulfil the DSM-IIIR criteria for dementia. Such deficits are not unusual in chronic schizophrenia,[37–39] and do not warrant a diagnosis of dementia. The cognitive impairment of our schizophrenic patients was not progressive and, in fact, reversed to some degree. From clinical experience, these patients are not usually referred for assessment for possible dementia unless there has been a recent change in their condition, as was the case in our patients. The reason for the recent deterioration was judged to be an acute exacerbation of the schizophrenic illness in three cases and a superimposed depression in two.

Heaton *et al*,[38] in their review of the neuropsychological studies of schizophrenia from 1965 to 1978, concluded that whereas nonpsychotic mentally ill patients could be reliably differentiated from those with known organic disease, chronic schizophrenics were discriminated from controls with brain damage at an essentially chance rate of 54 per cent. The cognitive deficit in our schizophrenic patients persisted without much improvement or deterioration over the follow up period. The patients, however, maintained independence in their activities of daily living and did not show progressive loss of functioning. If this cognitive deficit is called schizophrenic dementia,[39] it is a dementia which is not generally progressive. Reported studies of the course of cognitive deficit in schizophrenia[40 41] support the finding that general intellectual decline does not occur in these patients although some patients do show deterioration in certain specific tasks.[39] As schizophrenia is still generally regarded as a non-organic psychiatric disorder, cognitive impairment caused by this illness cannot arguably be considered true dementia and this is the position we take.

Re-examination of the clinical picture at initial presentation is revealing. The features most important for the diagnosis were a history of psychiatric disorder of some standing and the presence of significant psychopathology cross-sectionally. The same observation has been made previously.[2 3] In all patients, neuropsychological assessments showed significant deficits so that organic deterioration was considered a strong possibility. In no case could the clinical psychologist state convincingly that the deficit was due solely to psychiatric illness. The presence of focal cortical signs such as apraxia, aphasia or agnosia had some predictive value for dementia. Abnormalities on EEG, AEG or CT scan were minor and did not contribute to the diagnosis in our patients except to possibly rule out identifiable causes of dementia. Those who have previously examined the ability of these investigations[2 18 29 42] to differentiate between depression and dementia have commented on the considerable overlap of abnormalities so that confident separation of the two syndromes was often not possible. Neuroendocrinological studies in recent years, particularly the dexamethasone suppression test, have also not been able to distinguish between the two with any certainty.[43 44]

In summary, this study shows that the diagnosis of pseudodementia predicts a course of illness which seems to reflect the primary psychiatric illness responsible for the picture of cognitive impairment. The suggestion that such patients usually have an underlying dementia with a superimposed non-organic psychiatric illness is not sustained. The study also indicates that the presence of focal cortical signs increases the possibility of a true dementia but does not affirm it. Constructional difficulties observed in our patients did not seem to have diagnostic significance. In the final analysis, a diagnosis of a non-organic psychiatric illness, supported if possible by past history and the rigorous exclusion of organic factors, allows a process of "pseudodementia" to be affirmed which has predictive validity.

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