

General Psychiatry **What do we know about pseudodementia?**

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ABSTRACT

Depression and dementia can lead to generalised cognitive and memory dysfunction. Thus, differentiating these disorders is important and challenging. Pseudodementia is a term used clinically to describe symptoms that resemble dementia but are caused by other conditions (most frequently depression), rather than being recognised as an official diagnosis. Pseudodementia is characterised by a cognitive impairment that mimics dementia but which does not have its origin in neurological degeneration, deriving instead from functional psychiatric conditions. This condition is more commonly observed in older adults (particularly those over the age of 50 or 60 years), and its risk factors overlap with those for depression. Pseudodementia is essentially characterised by deficits in memory, executive function and speech and, therefore, can easily be confused with dementia, although there are aspects that allow its differentiation. Diagnosing pseudodementia can be difficult, especially as there is significant overlap between its symptoms and those of other conditions. However, it is important to recognise characteristic aspects of this disorder, as its correct identification is essential for proper treatment.

INTRODUCTION

The connection between depression and dementia arises from two key factors: first, cognitive impairments can be observed in depression, and second, dementia may also present with depressive symptoms.

When assessing a patient displaying signs of cognitive decline, various reversible factors must be ruled out before confirming a neurodegenerative condition. Among the well-recognised factors to consider, depression holds significant importance as it can substantially harm cognition, particularly among older individuals. In severe cases, depression may even be mistaken for neurodegenerative conditions. Historically, this phenomenon is known as ‘pseudodementia’.^{1,2}

Depression is a mental disorder that includes a depressed mood that lasts for at least 2 weeks, accompanied by loss of interest or pleasure in nearly all activities, feelings of guilt or suicide, social isolation and sleep and appetite disturbances.^{1,3}

Depressive symptoms are estimated to occur in many older individuals, with 1%–5% of older people living in the community

meeting criteria for major depression.⁴ Thus, geriatric depression is a common problem, with some epidemiological studies suggesting that a significant part of the older population exhibits prominent affective symptoms.⁵

Certain symptoms of depressive disorders tend to be more pronounced among older adults. Post⁶ had already reported in 1962 that one-third of older patients with depression had severe psychomotor retardation or agitation, and a recent meta-analysis confirmed these findings.^{7,8} It also reported that hypochondria and somatic symptoms were more common in older adults, whereas guilt and loss of libido were less common than in younger adults.^{7,8}

The development of depressive disorders in geriatric individuals shares a broad aetiology with similar disorders occurring at younger ages, where biological, psychological and social factors all play significant roles. Studies have shown a genetic contribution comparable to that of depression occurring at a younger age.^{9,10}

Loneliness and the difficulties of old age can be expected to be important contributing factors. However, the main difference in the aetiology of depression in the elderly is related to vascular factors. There is evidence for a multifaceted and bidirectional relationship between geriatric depression and vascular diseases. Studies show that diabetes, stroke and an elevated vascular risk score are all significant risk factors for depression in older adults, but curiously, hypertension, smoking and dyslipidaemia are not.¹¹ Proposed biological mechanisms include the frequently seen white matter changes in the magnetic resonance imaging (MRI) of geriatric patients with depression, which reflect focal areas of ischaemia and infarction, with effects on brain connectivity.¹¹ In addition, inflammation is also considered a contributory factor.¹¹

Furthermore, depression often co-occurs with a wide range of medical conditions, including Alzheimer’s disease (AD), stroke and Parkinson’s disease, making the diagnosis



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of these diseases difficult and exacerbating the functional decline associated with each one.¹²⁻¹⁴

METHODS

Since this work represents a narrative review, the authors carried out a non-systematic review of the existing literature. We conducted a search in the PubMed database using the following Medical Subject Headings words as search terms: “pseudodementia”, “cognition”, “memory” and “depression”. Simultaneously, the authors conducted a manual search in other relevant and reliable sources (books and scientific websites).

Non-standardised inclusion and exclusion criteria were used. Search criteria were limited to articles published in English or Portuguese until May 2023. Relevant bibliographical sources were selected only if they focused on information relevant to the review, and which covered the topics the authors intended to address (definition, history, epidemiology, characteristics, causes and risks factors, diagnosis and differential diagnosis, treatment and prognosis).

The included articles (n=35) were online articles, book chapters, meta-analysis, reviews, retrospective studies, prospective cohort studies, cross-sectional studies and original research. These articles were reviewed, and relevant data were extracted to report the key findings.

PSEUDODEMENTIA

The term “pseudodementia” is a popular clinical concept, although unaccepted as an independent nosological category in any classification system.^{7 15-17} Pseudodementia describes various functional psychiatric conditions, with depression being the most prevalent. These conditions can simulate organic dementia but typically show the potential for reversal with appropriate treatment.^{1 2 7 18}

Individuals with moderate-to-severe depressive disorders frequently develop a broad decline in cognitive functioning in association with attention deficit and psychomotor retardation. These patients may also struggle with clear thinking, concentration difficulties, decision-making challenges, notable forgetfulness and problems with reasoning.¹⁹⁻²¹

Both depression and dementia can lead to reduced motivation, impaired concentration and retardation, and, consequently, both can lead to generalised cognitive and memory dysfunctions.

Therefore, differentiating this diagnosis from dementia is important but challenging. The distinction is further complicated by the evidence that depressive pseudodementia is a strong predictor of subsequent dementia.²²

HISTORY

The term “pseudodementia” was initially introduced in 1961 by psychiatrist Leslie Kiloh. Kiloh observed that patients exhibiting cognitive symptoms resembling those

found in individuals with dementia showed a potential for the reversal of deficits. The cognitive decline in these cases was actually due to mental health conditions rather than central nervous system disorders, and Kiloh pointed out the potential reversibility of cognitive impairment in many of these cases, making his article significant at a time when dementia was considered a condition that could not be reversed.^{7 8 18 23 24}

Uncertainties surrounding the classification and attributes of pseudodementia, as well as concerns regarding its misleading name, have prompted suggestions to discontinue its usage. Certain professionals and experts in the field advocate against continuing to employ this term due to the absence of distinct and objective diagnostic criteria, as well as the potential for the prefix ‘pseudo’ to inaccurately imply the cognitive deficits are not real. However, others defend its practicality and usefulness as it has held up well in clinical practice and highlights those patients who may have a treatable condition.⁸

EPIDEMIOLOGY

There are few studies on the prevalence of pseudodementia. Ferran *et al*²⁵ found that pseudodementia accounted for 18% of referrals to a presenile dementia service, but it was not diagnosed by the majority of referrers.¹⁵

Rabins²⁶ studied rates of reversible dementia in a group of patients admitted to a psychiatric hospital and found that the annual incidence among patients with dementia referred to psychiatric services was approximately 41%.

Estimates suggest that between 2% and 32% of older adults with cognitive problems actually have pseudodementia.¹

PRESENTATION/CHARACTERISTICS

Depression in old age is clinically heterogeneous, and the severity of depressive symptoms does not directly relate to the level of cognitive impairment. Despite the heterogeneity, there are some distinctive neurocognitive and behavioural changes that appear to be attributable to geriatric depression and that are characteristic of a large group of patients who do not have neurological disease.²⁷

Pseudodementia typically encompasses memory issues, impairments in executive functioning and speech deficits. Notable cognitive symptoms may include difficulties in remembering words or remembering things in general, reduced attention and concentration, challenges in task completion or decision-making, decreased speech fluency and speed and impaired processing speed. Individuals with pseudodementia commonly experience significant distress due to the cognitive impairments they encounter.^{1 7 28}

Key symptoms of pseudodementia comprise speech impairments, memory lapses or deficits, attention difficulties, emotional regulation challenges and problems with organisation or task planning. Remarkably, these

symptoms are also highly prevalent among individuals with dementia. Consequently, doctors may often lean towards diagnosing and treating individuals exhibiting these symptoms as if they have dementia.^{1 7 16}

In addition, cognitive tasks that require sustained effort and elaborate cognitive processing may be more affected in depression than tasks that can be accomplished more automatically.

As pseudodementia is often linked to depression, the person may also experience symptoms that include depressed mood, fatigue or anergy, insomnia or hypersomnia, anhedonia, anorexia or hyperphagia and suicidal thoughts or behaviours.³

Memory deficits

It is now generally accepted that depression presents with a series of deficits in the domains of episodic memory and learning.

Both explicit verbal and visual memory functions exhibit deficits in individuals with both melancholic (endogenous) and non-melancholic (non-endogenous) depression. Nevertheless, these patients demonstrate intact performance on implicit memory tasks, and certain other memory functions appear to remain unaffected.^{28 29}

The neurobiological underpinnings of these memory deficits remain uncertain. Temporal lobe lesions are known to result in episodic memory deficits. Furthermore, reductions in hippocampal volume are found in patients with major depression. These findings may point to a temporal lobe deficit as the reason for cognitive decline.^{28 30}

However, most researchers suggest that such deficits may be primarily due to attention problems and not due to structural problems.²⁹

Executive function deficits

Depressive disorders commonly exhibit executive function deficits with clinically significant impact, potentially serving as a mediator of the functional impairment experienced by patients. Among the executive functions, task switching skills and attentional focus were found to be particularly affected in patients with depression.^{28 29 31}

Older individuals with depression experience executive function deficits, although to a lesser extent compared with patients with AD.²⁹

Another significant deficit is impaired processing speed, which presents clinically as psychomotor retardation.²⁹

On remission of the depressive episode, improvements were observed in the domains of visual learning ability, spatial recognition memory, psychomotor speed and executive function.²⁹

Speech and language function deficits

In measures of naming, repetition, general reading ability, syntax and auditory verbal comprehension, patients with depression exhibited superior performance compared with patients with AD. At the same time, healthy individuals tended to perform better than patients with

depression in general, although these differences were considerably less obvious than those observed between patients with depression and patients with AD.²⁹

It is also important to highlight other points mentioned in the studies, namely²⁹:

- ▶ The assessment of neuropsychological functions in older individuals with depression has revealed evidence of a subtle decline in ‘right hemisphere skills’ (eg, performance intelligence quotient, visual construction ability, non-verbal visual memory).
- ▶ In formal neuropsychological tests, these patients show deficits in tests sensitive to frontal lobe function.

Hence, the cognitive score pattern indicating dysfunction in the right hemisphere and anterior cerebral regions align with findings from certain studies demonstrating reduced regional cerebral blood flow in the right hemisphere and bilateral frontal lobe in individuals with depression.

CAUSES AND RISK FACTORS

Mood-related disorders, including depression, are among the possible causes of pseudodementia. However, they are not the sole contributors. Other mental health conditions such as schizophrenia, other psychoses, dissociative disorder, conversion disorder and mania can also lead to cognitive impairment and manifest in a similar clinical condition. These disorders may cause additional typical symptoms, concomitant with apparent symptoms of dementia.^{1 2 7 18}

The risk factors for pseudodementia are very similar to those for depression. Among them is gender—with women being statistically more likely to develop depression—family history, divorce and lower socioeconomic status.¹⁶

Certain researchers propose that pseudodementia may arise when a mood-related disorder develops in a brain that is already somewhat compromised due to the effects of ageing.¹⁶

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Diagnosing pseudodementia in older adults poses challenges due to several factors.

First, the ageing process itself brings about cognitive and brain function changes, making it challenging to distinguish between age-related changes and initial indications of depression or dementia. Second, diagnosing pseudodementia becomes more complex as symptoms of depression and various progressive neurological conditions frequently overlap in older individuals. Lastly, it is entirely possible for an individual to concurrently experience both genuine dementia and depression, further adding to the complexity of diagnosis.^{12-14 16 18 28 32}

Due to the complexities involved, diagnosing pseudodementia often requires a thorough and comprehensive evaluation. Medical professionals prioritise ruling out dementia and other cognitive issues before exploring

alternative potential causes. This meticulous approach may take some time to ensure an accurate diagnosis.

Distinguishing between depression and other conditions in older adults is supported by careful screening of both depressive symptoms and cognitive status, which may be complemented by a neuropsychological assessment that provides information regarding the nature of cognitive impairments and current patient status for future comparisons (baseline). The information is useful in diagnosis and management.^{1 17}

A diagnosis of depression rather than dementia is first considered when there are symptoms such as delirium of ruin, high levels of anxiety, significant hypochondriacal worries and a previous depressive episode. However, individuals with depression may express concerns regarding memory problems and experience distress related to this. When assessed using neuropsychological tests, they typically demonstrate satisfactory performance in memory-related tasks. On the other hand, individuals with dementia often deny having memory problems or minimise their importance, and show impairment in neuropsychological tests.^{1 18 23 28}

Obtaining an accurate diagnosis is crucial for individuals experiencing cognitive problems as certain causes are reversible. Cardoso *et al*²⁸ warn that several situations can lead to cognitive deficits, making it essential to collect a complete clinical history and to assess the baseline functional status of the patient. This review also emphasises that a neurological examination should always be performed, as well as excluding the main organic causes of cognitive impairment.²⁸

For instance, comorbidities such as vascular, infectious, traumatic, autoimmune, idiopathic and even malnutrition-related conditions can present symptoms that resemble dementia. Prime examples are individuals with chronic vitamin B₁₂ deficiency or hypothyroidism that may exhibit dementia-like symptoms. Thus, underlying conditions or deficiencies should be routinely excluded through an analytical study.^{2 28}

Imaging tests also aid the diagnosis of pseudodementia, as they provide clues to the underlying causes or show the effects of degeneration on the brain in the case of dementia.^{2 28 32} MRI shows changes such as atrophy of the medial temporal lobe in people with AD, whereas positron emission tomography and single-photon emission computed tomography (CT) images of the brain show reduced blood flow in some areas.^{23 32 33}

Lastly, certain forms of dementia, such as AD and Parkinson's disease, share mood-related symptoms that resemble those seen in depression.²⁹

The Geriatric Depression Scale (GDS) is frequently employed as a tool to assist in distinguishing between pseudodementia and other types of dementia. The results from the GDS are combined with additional patient information regarding their history and current functioning, aiding in the diagnostic process. People with pseudodementia are likely to score high on the GDS—this reflects that the patient is depressed.^{1 16 18 32 34}

The short version of this scale (**box 1**) when applied in a consultation context can be useful, more practical and less exhaustive.

The factors that can help us to differentiate between pseudodementia and dementia are presented in **table 1**.

Pseudo-pseudodementia

With regard to the differential diagnosis, it is important to be aware of another entity: pseudo-pseudodementia.

A notable limitation in much of the existing research on pseudodementia is the lack of thorough screening for organic dementia, making it challenging to exclude individuals with pre-existing neurodegenerative diseases. This brings attention to another potential scenario where neurodegenerative diseases are misdiagnosed as psychiatric disorders, a possibility acknowledged by Kiloh.⁸ This scenario can be seen as the reverse of pseudodementia, where organic dementia disguises itself as a purely psychiatric condition. Due to the resemblance to pseudodementia, this condition has occasionally been referred to as 'pseudo-pseudodementia'.⁷

While it is important to note that the term does not aim to supplant existing diagnoses, its purpose is to draw attention to a clinical scenario in which psychiatric symptoms appear consistent with a primary psychiatric disorder, potentially leading to misinterpretation and obscuring the true underlying diagnosis of a neurodegenerative disease.

This phenomenon appears to be relatively common, as evidenced by a study that reported approximately 28% of patients with neurodegenerative diseases receiving an initial psychiatric diagnosis during the period between

Box 1 Short version of the Geriatric Depression Scale³⁸

The patient must choose the answer that best applies to the last week.

1. Are you satisfied with your life? **YES/NO**
2. Have you abandoned many of your activities and interests? **YES/NO**
3. Do you feel that your life is empty? **YES/NO**
4. Do you often get bored? **YES/NO**
5. Are you in a good mood most of the time? **YES/NO**
6. Are you afraid that something bad will happen to you? **YES/NO**
7. Do you feel happy most of the time? **YES/NO**
8. Do you often feel hopeless? **YES/NO**
9. Do you prefer to stay at home instead of going out and doing new things? **YES/NO**
10. Do you feel that you have more memory problems than most? **YES/NO**
11. Do you think it is wonderful to be alive now? **YES/NO**
12. Do you feel very useless the way you are now? **YES/NO**
13. Do you feel full of energy? **YES/NO**
14. Do you feel that your situation is hopeless/insoluble? **YES/NO**
15. Do you think most people are better than you? **YES/NO**

Answers in bold indicate signs of depression. One point is given for each answer in bold.

A score >5 points is suggestive of depression.

A score ≥10 points is almost always indicative of depression.

A score >5 points should justify a follow-up assessment.

symptom onset and the ultimate diagnosis. Individuals with the behavioural variant of frontotemporal dementia seem to be particularly susceptible, with 51% of them receiving a preceding psychiatric diagnosis, in contrast to 23% of those diagnosed with Alzheimer's dementia. For both groups, the most common psychiatric diagnosis given was depression, followed, in the case of frontotemporal dementia, by bipolar disorder and schizophrenia.⁷

The frequency of these errors highlights the challenges involved in recognising the correct diagnosis.

An article published in 2020 proposes that pseudopseudodementia serves as a valuable concept that acknowledges the intricacies beyond the conventional dementia-pseudodementia dichotomy. It highlights the difficulties in distinguishing between pseudodementia and pseudo-pseudodementia and determining whether psychiatric symptoms mimic or result from neurodegeneration.⁷

Comorbid dementia and depression

The incidence of depression may be 30% in vascular dementia and in AD, and over 40% in Parkinson's disease dementia and Huntington's disease dementia.²⁷ A study published in 2023 shows that depression is a comorbidity often associated with dementia and also that depression increases the risk of dementia.¹³ People with any type of dementia have a high incidence of major depression, particularly if they have vascular dementia or Parkinson's disease dementia—these patients often have more insight and awareness of their condition than people with AD.^{11 13 27–29} However, there is some disagreement on this point. For example, the conclusions of a study from 2022 cast doubt on the frequency of depressive pseudodementia disorder in older populations with documented depression and memory impairments.²⁴

Due to the similarities in symptoms between dementia and depression, it can be challenging to ascertain whether an individual living with dementia is also experiencing depression. Some revealing signs include loss of interest and pleasure in activities the person usually enjoys, social isolation, lack of energy, negativity, hopelessness or even nihilistic feelings of worthlessness or sadness, ideas of guilt and self-harm and increased confusion.^{34 35}

Furthermore, when people with dementia develop depression, their overall cognitive ability may decline significantly, with worsening of memory and concentration symptoms.^{12 34 35}

TREATMENT

The treatment of pseudodementia can be a time-consuming and individualised process, as various individuals may respond differently to various treatment options.

Once other potential diagnoses have been ruled out and pseudodementia is suspected, the recommended treatment will primarily concentrate on addressing the underlying cause. This often entails treating the underlying depression that triggered the symptoms. Treatment

Table 1 Features that help distinguish between dementia and pseudodementia^{2 18 23 28 32 33 39}

Pseudodementia	Dementia
Abrupt onset, usually within days or weeks	Insidious, subtle onset
Rapid symptom progression Symptoms of short duration and more severe in the morning	Progression of symptoms is slow, gradual Long-term symptomatology with nocturnal worsening
Family aware of the onset of symptoms	Family initially unaware of disabilities/deficits
Personal history of depressive or manic episodes often	Rarely with personal history of mood disorders
Family history of mood disorders	Family history of dementia often
Depressed mood; little or no reaction to sad or funny situations; behaviour and affects are inconsistent with the degree of cognitive impairment	Superficial or labile mood; normal or exaggerated response to sad or funny situations; affects are consistent with the degree of cognitive impairment
Poor collaboration; little effort to perform well; often tends to give up; often responds with "I don't know"; apathetic, emphasises failure	Good collaboration; frustrated/reacts catastrophically to error/inability to do well; emphasises performing trivial activities
Highlights memory loss; disturbed by memory lapses but performing well on cognitive tests; greater impairment of personality characteristics (eg, self-confidence, motivation, interests and attention)	Denies, hides and minimises difficulties/deficits; poor performance on cognitive tests; greater impairment in cognitive characteristics (recent memory and temporal orientation)
MMSE with variable results at different times	MMSE with stable result (low score)
Predominance of psychological symptoms: sadness, anxiety, somatic symptoms	Predominance of neurological symptoms: dysphasia, dyspraxia, agnosia, incontinence
CT: usually little evidence of atrophy	CT: atrophy and enlargement of the ventricles
EEG: normal	EEG: pronounced slow activity
SPECT: normal blood circulation pattern	SPECT: parietotemporal and frontal abnormalities often
Good prognosis	Poor prognosis
Usually responds to treatment with antidepressants and/or electroconvulsive therapy	Incurable; treatment consists of controlling symptoms

CT, computed tomography; EEG, electroencephalogram; MMSE, Mini Mental State Examination; SPECT, single-photon emission CT.

for depression can vary by individual, but typically should involve a combination of psychotherapy and antidepressant medication.^{1 2}

Providing appropriate medical treatment for depression can potentially aid in differentiating between pseudodementia and dementia. Effective treatment with antidepressants may help alleviate the cognitive dysfunction associated with depression, leading to an improvement in cognitive symptoms. In contrast, the cognitive dysfunction associated with dementia typically persists and progresses over time. By observing the response to antidepressant treatment, a distinction can be made between the reversible cognitive impairment of pseudodementia and the progressive cognitive decline of dementia.⁷

Pharmacological intervention

When it comes to antidepressants, vortioxetine is a drug approved by the Food and Drug Administration in 2013

for major depressive disorder that is similar to a selective serotonin reuptake inhibitor, and it has emerged as a promising potential treatment for the cognitive symptoms of depression. Preclinical animal studies and extensive randomised controlled trials have demonstrated that vortioxetine can notably enhance cognition in patients with depression who have achieved remission, independent of its antidepressant properties. Clinical trials have revealed significant enhancements in working memory, processing speed, executive function and attention among individuals treated with this medication.³⁵ MRI studies have shown that vortioxetine modulates a brain circuit involved in the working memory. They have also shown that it stimulates the growth of dendritic spines and synaptic connections *in vitro* and stimulates gene expression of factors involved in neuroplasticity. These findings suggest a possible biological mechanism underlying vortioxetine's procognitive effect in patients with depression and healthy controls.^{21 23 36}

Non-pharmacological interventions

It should be noted that, in cases where antidepressant therapy is not well tolerated or is ineffective, electroconvulsive therapy can be considered.²³

In addition to medical treatments, other mental health therapies play a vital role in depression treatment. Psychotherapeutic approaches, such as cognitive-behavioural therapy or interpersonal therapy, are valuable in addressing the symptoms and some aetiological causes of depression. These therapies can be conducted on an individual basis or in group settings and offer strategies for managing and reducing depressive symptoms.¹

While depression can be effectively treated, symptoms, including cognitive impairment, typically do not subside immediately. Both medication and psychotherapy techniques may take several weeks before a noticeable reduction in symptoms occurs. Additionally, individuals with depression may experience relapses, highlighting the importance of ongoing treatment and support.¹

Lastly, even with adequate treatment, not all cognitive deficits associated with depression necessarily resolve, especially in the geriatric population. In older patients, it may even be due to the co-occurrence of another pathological process such as AD or cerebrovascular disease.

OUTCOMES/PROGNOSIS

Reversible cognitive impairment in moderate-to-severe depression in older adults is considered a strong predictor of dementia according to most authors, although some consider that depression with reversible cognitive impairment may be a prodromal phase of dementia rather than a risk factor.^{13 14 19 20 31} In these cases, the type of depression appears to be qualitatively different: more resistant to treatment and with a more chronic course.¹⁵

Evidence indicates that the most likely types of dementia to develop are AD and vascular dementia.¹⁵

In one study, 44 older patients with depressive pseudodementia were followed for up to 18 years at 6-month intervals. Eighty-nine per cent of the patients originally recruited into the study developed dementia. The main limitation of this study was the lack of a control group.¹⁵

In another study of 5-year to 7-year follow-up conducted by Saez Fonseca *et al*, the conversion rate to dementia over follow-up in a group of older adults with depression with impaired cognitive function was found to be 71.4%, compared with only 18.2% conversion in the cognitively intact group. The group of older patients with depression with impaired cognitive function had a higher risk of dementia and institutionalisation than the group of older patients with depression without impaired cognitive function. Impaired cognitive function was one of the strongest predictors of dementia, with an absolute risk increase of 53.2%.

In another study conducted by Xu *et al*, the incidence rate of dementia in people with depression was far more than those who were free of depression, and high-risk lifestyle factors (like physical inactivity) were associated with higher risk of transition from depression to dementia, highlighting the great significance of integrating comprehensive behavioural interventions, particularly for regular physical activity, for prevention of both depression and postdepression dementia.³⁷

A review carried out by Brodaty and Connors shows that although pseudodementia presenting later in life is associated with an increased likelihood of subsequently developing organic dementia, pseudodementia presenting earlier in life does not show this effect.⁷

Processing speed abnormalities and memory function impairments are cognitive characteristics primarily observed during acute depressive episodes. However, executive deficits may persist in certain individuals with a history of depression and can serve as predictors of treatment response. Recent research indicates that more severe executive deficits at the onset of depression correlate with a poorer prognosis for recovery. In contrast, deficits in visual memory, visual spatial ability and nonverbal intelligence may be depressive cognitive features that remain after recovery from depression and possibly signal an underlying chronic abnormality in the right hemisphere.¹⁵

CONCLUSION

Pseudodementia describes a collection of symptoms commonly observed in older adults that resemble dementia but do not involve neurodegeneration. Pseudodementia is not an official diagnosis but rather a descriptive term for a specific group of patients. In the older population, pseudodementia can have a detrimental impact on cognition and functionality, and it may also serve as a predictor for the subsequent development of dementia.

Pseudodementia shares similar risk factors with depression, including female gender, family history, divorce and low socioeconomic status. While depression is the primary cause of pseudodementia, other mental health conditions such as schizophrenia, psychoses, dissociative disorder, conversion

disorder and mania can also lead to cognitive impairment and produce similar clinical symptoms.

Dementia and depression can coexist, and pseudodementia and dementia can be difficult to distinguish. The main characteristics that allow the differentiation of these conditions must be addressed in the interview (onset and course of symptoms, family history, personal history, etc). Treating pseudodementia involves treating the underlying condition.

To the best of our knowledge, there is no other review on pseudodementia as complete as ours, nor as up-to-date.

The lack of combined information regarding the different dimensions of pseudodementia may itself add to the challenge of managing this condition. Our review compiles a wide range of features of pseudodementia—epidemiology, history, presentation/characteristics, causes, risk factors, diagnostic approach, differential diagnosis, treatment and prognosis. Our work stands out for addressing novel findings and for being extensive. Thus, it will provide valuable help in clinical practice, given the significant prevalence of pseudodementia and the associated challenges of diagnosis and management.

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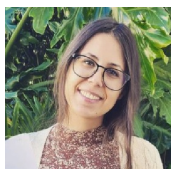
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