

# Pseudo-dementia: An Artefact or a Grey Area of Geropsychiatry?

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## Abstract

*Pseudodementia is a phenotype approximated by a wide variety of underlying disorders. The history of disturbance in pseudodementia is often short and abrupt onset, while dementia is more often insidious. Clinically, people with pseudodementia differ from those with true dementia when their memory is tested. On other terms the relationship between dementia and depression has been so intricate and complex that sometimes it gets difficult to distinguish between the two clinically. Depression is an early symptom of dementia' was proposed and it underscored the*

*importance of differentiating pseudodementia from 'organic' dementia. Though rare, pseudodementia has proved to the clinical science the importance of treating early depression. It has bridged the gap between the 'organic' and 'functional' cerebral diseases. So understanding and treating pseudodementia would help us to prevent and rather cure various chronic cerebral diseases in the near future.*

## Key words:

*Pseudodementia, Depression, clinical difference, cerebral disease*

## Introduction

53 years has passed since Leslie Kiloh's paper titled, 'Pseudo-dementia' was published in *Australian psychiatry* (Kiloh LG, 1961). It was a decisive moment, since for the very first time in the history it was shown that an 'organic' brain ailment could be reversible if properly managed. It was not for the first time that the term pseudodementia was used. In earlier 19th century, Albert Mariet research on 'Melancholy Dementia' also suggested that 'sadness' was the primary defect in emergence of cognitive defects, such as dementia and hallucination (BERRIOS GE 1985). However Kiloh's paper provided an impulse to psychiatrists to focus on

potential reversibility of cognitive disorders, especially dementia. Pseudodementia, as Leslie suggested, is defined as an *intellectual impairment in patients with a primary psychiatric disorder, in which the features of intellectual abnormality resemble, those of a neuropathologically induced cognitive deficit. This neuropsychological impairment is reversible, and there is no apparent primary neuropathological process that leads to the genesis of this disturbance* (Caine ED 1981). Eighteen years later Well.C diagnosed 10 patients with pseudodementia (Wells C 1979) and in 1981, Caine

proposed the first ever-diagnostic criteria for it (Caine ED 1981).

A theory, 'Depression is an early symptom of dementia' was proposed and it underscored the importance of differentiating pseudodementia from 'organic' dementia, prevalence of which was estimated to be around 10-20% (Garcia CA et al 1981, Jeste, DV et al 1990). However over-time it was soon realized that so proved reversible dementia prevalence was much lower than it was thought of. In 1988, two meta-analyses suggested that the prevalence of reversible dementia was significantly lower than had been estimated previously. It was predicted that further work would indicate an even lower rate. In October 2003 Clarfield AM published a paper showing meta-analysis study of 39 articles, representing 7042 patients of whom 5620 (87.2%) had dementia. It revealed that only 0.6% of dementia cases actually reversed (0.29% partially, 0.31% fully) (Clarfield AM, 2003). Better and longer follow-up in studies of patients presenting with depression and cognitive impairment was thought to be the primary reason for this dramatic shift in the prevalence of reversible dementias. These studies had a significant clinical and economical implication on the future workup of dementia. Since now doctors were insignificant about a disease, which is so rare, undermining its huge significance.

#### **Differentiating dementia from depression:**

The relationship between dementia and depression has been so intricate and complex that sometimes it gets difficult to distinguish between the two clinically. Depression and dementia, among the most common conditions in clinical practice, can coexist or sometimes can succeed each other, and often confuse clinicians (Kobayashi T et al 2011). The clinical term "pseudodementia" has remained a permanent nosological entity in the

literature for over 100 years and recognition of the fact that clinical symptoms associated with reversible neuropsychiatric conditions can mimic irreversible disorders was known as early as the middle of the 19th century. But today due to recent advancement in our methodology to identify brain anomaly, the diagnosis of pseudodementia has been made easy. A study done in 1989 reports use of EEG in distinguishing depressive pseudodementia and dementia with secondary depression (Brenner RP et al 1989). In 2000 a study showed sleep polygraphy to be a better diagnostic tool in distinguishing dementia and depression in pseudodementia patients (Kohl FS et al 2000). However these tests have their own limitations making it difficult to clinically differentiate between dementia and depression till today. Being one of the most common psychiatric ailments, it's an ignominy that medical science does not have a reliable method for its clinical diagnosis.

#### **Is depression the only risk factor?**

As already discussed, depression is the foremost cause for causing dementia in the individuals having pseudodementia, but on accounts of its rarity it was always fascinating as to what other factors were mandatory for causing this ailment. Brain single photon emission computed tomography findings in depressive pseudodementia patients revealed some facts regarding the pathogenesis of this disease (Cho MJ et al, 2002). This study, conducted in 2002, compared the cerebral blood flow in patients with 'depressive pseudodementia', patients with 'depression free of cognitive impairment', patients with 'dementia associated with Alzheimer's disease' and healthy patients, which served as control. The depressive pseudodementia group of patients showed a decreased blood flow in temporal parietal region of the brain, similar to that of Alzheimer's disease and different from that of depression group. It indicated though vaguely, that both

Alzheimer's and depression have somewhat similar effects on brain leading to dementia. Similarly an interesting case study, done in 2006, linked pseudodementia with autism (Pollard AJ et al 2004). A female child aged 6 years with autism was diagnosed with pseudodementia and she responded well to antidepressant treatment. Though unusual this study made the picture of pathogenesis of pseudodementia much more complicated.

In 2004 it was found that the syndrome was found more common in women belonging to higher socio-economic background with past psychiatric history (majorly accompanied by depressive symptoms). This descriptive case study, done by Hepple J (2004), suggested that pseudodementia in these people was caused by a cataclysmic reaction to cumulative loss in later life that have predisposing borderline and narcissistic personality traits and the treatment of which using psychotherapeutic approaches may limit the progression of the syndrome if it is recognised at an early stage.

Recently on 1st June 2014 an article published on pseudodementia finally shed some light on the genetic basis of the disease (Bieniek KF 2014). A consecutive series of 31 cases from the brain bank for neurodegenerative disorders at Mayo Clinic were screened to assess the incidence of the expanded C9ORF72 repeat in cases of depressive pseudodementia. The presence of the hexanucleotide repeat was established using immunohistochemistry with a highly disease-specific antibody (C9RANT), and

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was further validated in carriers using repeat-primed polymerase chain reaction and Southern blotting. This article increased the horizon of clinico-pathological presentations of C9ORF72 expanded hexanucleotide repeat by including psychiatric disorders such as pseudodementia.

Though remaining clinically silent for over a century, pseudodementia is finally getting the clinical importance it earlier deserved. Much has been found and much still has to be discovered about its pathogenesis.

## Conclusion

In summary, depressive pseudo-dementia evolved from a concern about the improper labelling of elderly patients with depression as having irreversible dementia. Recent data has shown that this condition is extremely rare, but treating it remains still important. While it may not cure the cognitive disorder or reverse the dementia, it will likely improve the patient's quality of life. Though rare, pseudodementia has proved to the clinical science the importance of treating early depression. It has bridged the gap between the 'organic' and 'functional' cerebral diseases. So understanding and treating pseudodementia would help us to prevent and rather cure various chronic cerebral diseases in the near future, making the concept of pseudodementia to be useful in spite of its limitations.

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