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### **Research Article**

# Age-Related Hearing Loss an Under-Recognised Cause of Memory Difficulties Mimicking Early Alzheimer's Disease

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#### Abstract

A 76 year-old male with a diagnosis of early Alzheimer's disease volunteered for a research study. Testing revealed a severe progressive hearing loss significantly affecting quality of life and leading to low mood and memory complaints. Formal testing revealed no objective evidence of cognitive decline or specific memory impairments. Volumetric analysis of structural MRI showed no abnormalities suggestive of Alzheimer's disease. Evidence from this case suggests that the high prevalence of un- or undertreated hearing loss can complicate the dementia assessment procedure by mimicking symptoms of cognitive impairment and therefore should be excluded as part of the diagnostic work-up.

Keywords: Hearing Loss; Dementia; Alzheimer's Disease; Cognition; Diagnosis

# Introduction

Early diagnosis of Alzheimer's Disease (AD) is often associated with better prognosis due to access to treatment, better forward planning and postponement of institutionalisation. An inaccurate diagnosis of dementia may be harmful because of adverse events and negative effect on quality of life. It is, therefore, essential to identify all features leading to inappropriate diagnosis. We describe a case of severe hearing loss (HL) mimicking the clinical features of early AD.

# **Case Presentation**

#### **Clinical summary**

A 76-year-old male, LW, with a diagnosis of early AD presented as a research volunteer. His clinical notes reported significant memory decline over 12 months, recent lack of understanding of finances and leaving the cooker alight. He was still active, showing no problems with driving, personal care, or independently going out and finding the way home. At time of diagnosis, his MMSE score was 30/30 and he had insight into his memory difficulties. Slight atrophy in temporal lobes was reported on MRI. LW was diagnosed with early AD and started on Donepezil that was subsequently stopped due to side effects.

Two years later, LW described his current mood as bad and memory as average, with periods of absentmindedness and lack of motivation. LW used a hearing aid because of severe HL. His hearing was deteriorating and affecting his enjoyment of music, an important part of his life. Both patient and spouse described the HL to be more debilitating than the consequences of AD.

## Neuropsychological & Neuroradiological findings

Neuropsychological testing and in-depth scanning was initiated, including extraction of bilateral hippocampi volumes.

The neuropsychological battery assessed various aspects of cognition (Table 1). Diminished performance was reported only for the Visuoconstructive Apraxia test, suggesting impairment in ability

 Table 1: Scores on the neuropsychological tests compared with controls.

 Controls Mean

Test	LW Score	Controls Mean Score	(S.D)
Mini Mental State Examination	27	28.42	1.43
Raven's Progressive Matrices (PM47)	34	31.08	3.76
Digit Cancellation	51	51.18	5.67
Confrontational Naming	20	19.5	0.7
Verbal Paired Associates	17	13.57	4.32
The Pyramids & Palm Trees Test	52	50.92	1.15
Rey Complex Figure Test:			
Copying	35	33.37	3.47
Delayed (10 minutes)	15	13.84	6.99
Category Fluency	46	53.25	12.55
Letter Fluency	35	41.44	15.57
Digit Span (Forward)	9	6.76	1.44
Digit Span (Backward)	6	5.36	1.34
The Stroop Test:			
Time Interference Effect	17.5	24.12	11.51
Error Interference Effect	0	0.63	3.86
Visuoconstructive Apraxia Test	10*	13.48	0.65
Token Test	32.5	33.87	1.58
WAIS- Similarities	26	20.42	8.24
Logical Memory:			
Immediate	14	13.89	2.69
Delayed (10 minutes)	20	18.78	1.93

\*Score at least 2 S.D lower than those achieved by the controls (n=52), mean age=77.2 (5.4), mean years of education=14 (3.78), M=24.

to organise and manipulate spatial information. Memory, attention, reasoning and executive abilities were all within normal limits when compared to reference controls.

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	LW	Mean ± SD	Range		
Hippocampal volume (ml)	2.62	2.57 ± 0.249	2.14 to 3.03 3.58 x10 <sup>-3</sup> to 4.75		
Hippocampal fraction	4.24 x10 <sup>-3</sup>	4.16 x10 <sup>-3</sup> ± 0.37 x10 <sup>-3</sup>	x10 <sup>-3</sup>		

#### Table 2: Hippocampal volumes for LW and controls



**Figure 1**: Coronal section of LW's brain showing the hippocampi. **Disclaimers**: The authors have no conflict of interest to disclose. Informed consent was obtained from this patient. Ethical approval was granted by NRES Committee North East- Newcastle and North Tyneside (Ref: 170445, 15/ NE/0152). This study was funded by an award by Deafness Support Network to JL and AV. This is a summary of independent research carried out at the NIHR Sheffield Biomedical Research Centre (Translational Neuroscience). The views expressed are those of the authors and not necessarily those of the NHS, the NIHR or the Department of Health. Author JL is currently supported by the NIHR Manchester Biomedical Research Centre.

No significant difference from controls was found in the wholebrain analysis of LW's brain, nor in hippocampal atrophy (Figure 1). No significant difference was found in hippocampal volume (p=.854) or hippo campal fraction (p=.843) between LW and controls (Table 2).

# **Discussion**

LW presented with subjective memory problems and severe, debilitating HL erroneously interpreted as early manifestation of AD. The neuropsychological profile was not consistent with AD, the clinical hallmark's being deficits in episodic memory and semantic disturbance [1] and there was no evidence of progression of symptoms.

The initial report and subsequent two-year follow-up by LW's original psychiatrist had no mention of HL, and its impact on symptoms had not been considered in his diagnosis which was reached based on incomplete history, limited cognitive testing and no objective documentation of memory impairment. This study

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highlights the strong connection between HL and psychological symptoms in line with published evidence [2]. We suggest the psychological associations of LW's HL contributed to subjective memory complaints, whereby HL masqueraded as a functional memory disorder.

A diagnosis of AD has consequences on personal identity, feelings of loss, uncertainty and frustration [3]. There are implications for the individual that go beyond the clinical sphere. LW reported feeling nervous about telling friends and family to the point of isolating himself, anxiousness around driving and cooking, fear for his future and the impending decline of abilities. Possible factors that could affect individual cognitive function in ageing, such HL, should be discounted before making a diagnosis of AD, especially when there is limited evidence of objective cognitive impairment or progression of symptoms.

# Conclusion

To summarise, HL is common in the elderly, and symptoms may overlap with those of cognitive decline due to neurodegeneration. Clinicians should assess patients hearing status and associating features, such as depression and low self-esteem before making a diagnosis of dementia. There is a risk that a) the recent inclusion of HL as an independent risk factor for dementia [4] and b) the impact of HL on cognitive testing [5] might lead to greater number of inaccurate diagnoses. In LW, HL masqueraded as a functional memory disorder and failure to make a correct diagnosis led to inappropriate treatment and psychological distress for both LW and his family.

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