

## CASE REPORT

## Cognitive impairment and depressive symptoms coexisting in an elderly man

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**SUMMARY**

We report a case of a man aged 67 years presenting with recent depressive symptoms and paranoid ideations in addition to 1-year cognitive impairment. He has vascular risk factors and family history of memory loss. An episode of depression 2 decades ago resolved spontaneously but was followed by occupational decline. On mental state examination, he denied having depressed mood, hallucinations or delusions, but there were prominent word-finding difficulties and impaired attention and concentration.

**BACKGROUND**

Elderly people with depression are less likely to endorse affective symptoms when compared with their younger counterparts. On the other hand, they are more likely to display cognitive changes and somatic symptoms. As a result, making a clinical diagnosis of dementia in this age group based on the evidence of cognitive decline is also not as simple, as it is often complicated by depressive features besides physical frailty and concurrent medical illnesses. The controversial use of the term pseudodementia actually reflects a diagnostic obstacle faced in dealing with patients with various psychiatric disorders, particularly depression, presenting with evidence of cognitive deterioration.

**CASE PRESENTATION**

A man aged 67 years was brought to our hospital for psychiatric assessment as he had broken a glass door in his house without provocation. He had justified his act as an attempt to protect his family from harm. He appeared very anxious and suspicious at that time but denied experiencing hallucinations preceding this incident. The act itself was not within the control of external agency, and it had not been accompanied by any thought alienation.

Preceding this incident, there were 2 months of intermittent depressive symptoms, including low mood, anhedonia, anergia, poor appetite and occasional negative cognitions including hopelessness. There was no sleep disturbance, suicidal ideation or manic symptoms. Collateral history revealed subjective symptoms of memory loss and difficulty in word-finding over a period of a year. The family had noted that he began having poor concentration and progressively slow responses to questions. Despite these observations by the family, the patient appeared apathetic to his memory loss and his low mood, systematically denying either symptom set existed. There was otherwise no

apraxia or ataxia. His activities of daily living and functional review showed him as an independent man with hypertension and diabetes mellitus which have been adequately controlled.

A look into his psychiatric history, however, revealed that a behavioural change had occurred 20 years ago when he was socially withdrawn, had insomnia, demonstrated irrelevant speech, appeared suspicious and showed disorganised behaviour. He got well after 3 years after traditional herbal treatment however of an unknown nature. There was no family history of psychiatric illness, but both his parents had subjective symptoms of forgetfulness. He had otherwise received 6 years of formal education and worked as a shop manager until about 10 years ago when he resigned and took up a lower-paid job. He smokes around 10 sticks of cigarette/day but has never abused alcohol or illicit substances. He was described as a friendly man who coped with problems well.

Mental state examination revealed an elderly man with good hygiene but poor eye contact and rapport. His speech was of slow rate, tone and volume and there were numerous pauses due to word-finding difficulty. He was having depressed mood and had restricted affect. There were no hallucinations, delusions or suicidal thoughts. He was orientated to time, place and person, but attention and concentration were impaired. His episodic memory was impaired via free recall testing, but semantic memory was intact via categorical fluency testing. A physical examination including a full neurological examination revealed no clear abnormalities.

**INVESTIGATIONS**

Full blood count, renal, thyroid and liver function tests, infectious disease screening and iron studies were normal. Fasting sugars, cholesterol and triglycerides were not deranged. Blood sugar level was within 5.4–8.6 mmol/L. His body mass index (BMI) was normal. Blood pressure monitoring in ward was within normotensive range.

His ECG showed normal sinus rhythm and he was on oral antidiabetic medications only.

CT brain (figure 1) suggested small vessel disease with cerebral atrophy, with no clear infarcts. A subsequent MRI has been arranged to further elucidate any brain pathology.

Psychological investigations included MMSE testing where he scored 22/30, losing points largely in terms of recall, attention and calculation. Montreal Cognitive Assessment was also carried out and he scored 17/30. Frontal lobe testing



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**Figure 1** Non-contrast-enhanced CT brain shows cerebral atrophy with absence of infarcts.

demonstrated severe impairment, scoring only 5/18 with defects, particularly noticeable in similarities testing and lexical fluency. Parietal and temporal lobe testing was normal.

Beck's Anxiety inventory was performed and his score was 6 (normal range). His serial Geriatric Depression Score (GDS) charting score was 12/15 consecutively. Confusion Assessment Method serial scores were 0 throughout admission. However, no formal neuropsychiatric assessment was performed.

### DIFFERENTIAL DIAGNOSIS

Alzheimer dementia would be the provisional diagnosis in this case. It is the most prevalent degenerative dementia and there have been progressive subjective symptoms of memory loss in the past year with associated mood symptoms. The family history of memory and cognitive symptoms that did not improve with antidepressant treatment further bolsters this diagnosis.

Major vascular neurocognitive disorder is a major differential diagnosis in this case. The patient has certainly been experiencing cognitive disruption in terms of learning and memory and also language disruption. Vascular-type aetiology is contributed by the fact that his cognitive decline is compounded by vascular risk factors. Nevertheless, the prevalence of Alzheimer's means it is still more likely.

Another strong contending differential is frontotemporal dementia behavioural type. The brain CT scan suggests a predominantly frontotemporal pattern of atrophy. Moreover, neuropsychological testing demonstrates predominantly attentional and executive function impairment (frontal lobe testing 5/18). The history of a psychiatric episode 20 years ago and his subsequent functional decline also lend weight to this differential.

Major depressive disorder with mood-congruent psychotic symptoms is another possibility because there were depressive symptoms interspersed during the period of memory loss. This may have also explained his episode 20 years ago which remitted spontaneously. However, the depressive symptoms do not appear to be the primary presentation back then.

On the other hand, there was a need to rule out delirium, but factors weighing against this diagnosis included the long duration of illness and absence of fluctuating consciousness.

The possibility of recurrent brief psychotic disorder was also considered, but that diagnosis would not fully account for the distinct mood and memory symptoms.

### TREATMENT

He was treated with sertraline 50 mg ON for the depressive features. At the same time, oral risperidone 0.5 mg ON was started on a suspicion of psychosis, but later tapered off as there were no further psychotic symptoms in ward since admission. Psychoeducation was given, but no psychotherapy was offered as the symptoms were felt to be largely organic in nature. As vascular dementia was suspected, no anticholinesterases were initiated.

### OUTCOME AND FOLLOW-UP

Following the initiation of medications, his low mood gradually improved over a month. This was evidenced by GDS scores falling to 6. However, when this occurred, it seemed that his poor memory did not, as his MMSE remained static at 22/30. The diagnosis hence leaned more towards the direction of neurocognitive disorder. The word-finding difficulties and problems with attention and concentration were still clearly present, despite adequate duration of treatment with antidepressants.

### DISCUSSION

The conjunction of cognitive impairment and depression as a presenting symptom can be a particularly tricky clinical scenario, as depressive disorder and major neurocognitive illness can share both sets of symptoms.<sup>1</sup> Reversible cognitive disturbances are most certainly a prominent part of a depressive symptom, especially in the elderly population.<sup>2</sup> However, at the same time, observations have noted that many patients with reversible dementia do not achieve complete recovery, even though their intellectual functions have recovered following the remission of depression.<sup>3</sup>

Thus, the focus should be on establishing clear temporality to see which set of symptoms are secondary to the other; to establish whether the cognitive impairment is irreversible following the amelioration of depressive symptoms; and to establish the primacy and prominence or otherwise of depressive symptoms. In patients with depression with cognitive impairment, cognitive assessments usually demonstrate improvements quantitatively post-treatment.<sup>4 5</sup> Patients with depression with reversible dementia are also more subjectively concerned about their memory loss<sup>6</sup> and have a more severe depressive picture with prominent hopelessness and helplessness compared with the cognitively unimpaired elderly who are depressed.<sup>7 8</sup>

The patient illustrated had been in remission for many years prior to this presentation, and had clearly experienced cognitive decline prior to developing his recent-onset depressive symptoms. In addition, his cognitive decline appeared to be constant, despite a month of antidepressant treatment and subsequent remission of depressive symptoms. Therefore, it is more likely that he had a combination of reversible and irreversible dementia. The former would have been more pseudodementia secondary to a depressive disorder which would have been reversed with antidepressants, thus non-blinding the underlying long-term baseline cognitive impairment.

## Learning points

- ▶ Vascular dementia can present in the absence of, or in the presence of, well-controlled, pre-existing vascular risk factors.
- ▶ A dementia picture can easily be obscured by the features of depression.
- ▶ Cognitive deterioration is a usual feature of depression in the elderly.

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**Patient consent** Obtained.

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