Review: Delusions in Dementia

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Introduction

The definition of a delusion is 'a false, unshakeable idea or belief ... held with extraordinary conviction and subjective certainty' [1]. Delusions are a common symptom of a range of psychiatric illnesses including schizophrenia, depression, bipolar disorder and dementia. The inclusion of dementia in this list is sometimes surprising to both doctors and carers, who restrict the symptoms of dementia to the cognitive domain; yet Alzheimer's first description was of a 51-year-old woman with focal cognitive deficits, delusions of jealousy and auditory hallucinations [2]. Considerable research has focused on the neuropathological findings, neuropsychological deficits and genetics of these diseases. It is only more recently that we have begun to untangle the non-cognitive or psychiatric features of the dementias.

More recently several factors have resulted in growing interest in this aspect of dementia. First has been the emergence of old age psychiatry as a discipline in its own right. In the UK every district now has psychiatrists with special responsibility for the care of older people with psychiatric illness, and clear guidelines exist for sharing the care of demented patients between them and geriatricians. The second factor has been social policy change. The focus of care for demented patients has shifted from hospital-based institutions to the community, with demented persons remaining in their own home supported by their families and community-based care. The presence of non-cognitive symptoms is very much less acceptable to family carers than memory or other deficits, and may significantly increase their burden. Finally, careful psychiatric assessment of patients with dementia may provide fourfold benefits: explanation and reassurance for the care-giver; the introduction of appropriate treatment for the patient; psychiatric symptoms may help to identify subtypes of dementia and estimate prognosis; and, scientifically, we may increase our understanding of the underlying functional, neurochemical and neuroanatomical substrates of psychotic symptoms, with potential application to other psychiatric disorders.

Epidemiology

Delusional symptoms in dementia are common, with a prevalence of between 10% and 70% [3] and an annual incidence rate of between 1% and 5% [4]. The prevalence varies with the methodology of the study and the population being studied, with the highest rates being found when structured assessment instruments are used. Using data from ten studies involving 2787 patients with Alzheimer’s disease defined by NINCDS/ADRDA [5] criteria, Allen and Burns [3] have calculated a weighted mean prevalence of delusions of 29.6%. Some studies have shown that delusions are more common in patients with moderate cognitive impairment [6], while other studies have found no association with cognitive impairment [7]. It is possible that as the dementia progresses, either increasing language impairment prevents the person expressing their delusions or that the cognitive impairments may restrict the development of complex delusional ideas. There are limited data on length and resolution of symptoms; however a few small studies have demonstrated a resolution rate of 2% per year, with duration of symptoms between 2 and 4 years [4].

Categories of delusions

An understanding of the types and frequencies of the delusional ideas experienced by patients with dementia is useful both in recognizing symptoms reported by care-givers, and as an aide-mémoire during history-taking and mental state examination. The Table gives a summary and brief description of the delusions most commonly found in dementia, with the approximate percentage of patients who experience each type of delusional idea. No formal classification of these symptoms exists, with a wide selection of categories being used by different studies. Classification is likely to be an important step in advancing research into the psychiatry of dementia.

Aetiological considerations

Some insights into the demented person’s delusional system are possible; in the earliest stages of Alzheimer’s disease memory is predominantly affected. Delusional explanations may then replace memory for lost items (delusions of theft), or activities of the care-giver or others (delusions of jealousy and persecutory delusions); impaired abilities to recognize faces (prosopagnosia) may lead to the failure to recognize the patient’s own reflection, with the delusional belief that he or she is seeing a stranger (the picture sign), or mis-recognition of the care-giver may give rise to the delusional idea that the care-giver has been replaced by an imposter (Capgras delusion).

Both the patient and the care-giver are affected by the presence of delusions. For the patient, the presence of delusions may result in increased aggression, agitation,
wandering, insomnia, urinary incontinence and distress [8]. In the long term, psychotic symptoms are associated with increased rates of hospitalization and are linked to entry into residential care [9]. For the caregiver, these symptoms are frequently frightening and outside their previous experience. Delusions are also commonly directed at the caregiver (jealousy, persecution), which together with the behavioural consequences increase the burden of care and diminish any positive experience of caring.

Few studies have examined the relationships between delusions and neuroimaging, neuropsychological and histopathological changes. From CT studies, an association has been noted in patients with delusional misidentification of right frontal lobe degeneration with relative preservation of the left frontal lobe [10]. Functional neuroimaging techniques such as PET and SPECT [11] have also demonstrated differences such as reduced bilateral temporal lobe blood flow [12] between deluded and non-deluded patients. Refining these techniques in groups of patients with specific symptoms may permit greater understanding of the anatomical substrates of particular phenomenological symptoms.

Similarly, few studies have examined the association of delusions in dementia and neuropsychological change. When comparing deluded and non-deluded patients, one study found that the neuropsychological differences between the two groups were not outstanding [6]. Other studies have suggested that delusions may have a particular association with frontal/temporal [13] or temporal/parietal [14] neuropsychological impairments. In a neuropathological study, Forstl et al. [15] found an association with delusions in patients with less severe cell loss in the parahippocampal gyrus and with lower cell counts in the dorsal raphe nucleus. Delusions and delusional misidentification were also common in five patients with basal ganglia mineralization.

Most studies have concentrated on Alzheimer’s disease, vascular dementia and, more recently, senile dementia of the Lewy body type (SDLT). The current consensus is that the frequency of delusions is slightly greater in Alzheimer’s disease than vascular dementia [3], while SDLT patients suffer delusions much more frequently, with prevalences of up to 57% [16]. Very little information is available for the unusual dementias and focal degenerations, but one study suggests that psychotic symptoms are very rare in Pick’s disease [17].

Using information from psychiatric assessments to aid diagnosis has only recently been explored. The diagnostic criteria commonly used for Alzheimer’s disease (NINCDS/ADRDA [5] and DSM-IV [18]) and vascular dementia (NINDS/AIREN [19]) make no reference to psychotic symptoms. However, for SDLT, the presence of hallucinations and delusions is an important part of the operational criteria [16].

Better classification of psychotic symptoms, applied to pure populations of patients with genetic or pathologically defined illnesses, may eventually demonstrate a greater role for using mental state information in improving the diagnostic accuracy of clinical criteria. Future studies will need better design if they are to begin to explore this question. Most previous studies of psychiatric symptoms in dementia have been limited: in

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### Table. Common delusions in dementia

<table>
<thead>
<tr>
<th>Type of delusion</th>
<th>Examples</th>
<th>Approximate prevalence (%)</th>
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<tbody>
<tr>
<td>Theft</td>
<td>Complain of people breaking into the house to steal or hide things</td>
<td>22</td>
</tr>
<tr>
<td>Phantom boarder</td>
<td>Other people living in the house: examples include the patient laying extra places at the table, or making extra cups of tea</td>
<td>20</td>
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<tr>
<td>Persecution and endangerment</td>
<td>Beliefs that people are ‘out to get me’, or that food is being poisoned</td>
<td>17</td>
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<tr>
<td>Spouse infidelity</td>
<td>‘You are seeing someone else behind my back’</td>
<td>5</td>
</tr>
<tr>
<td>One’s house is not one’s home</td>
<td>The person may ask ‘When are we going home?’ when they are in their own home, or, if more strongly held, may leave their home in search of ‘home’, which may sometimes be a previous address</td>
<td>5</td>
</tr>
<tr>
<td>Delusions of infestation</td>
<td>The person believes that they or their home is infested by small organisms, e.g. spiders, worms, lice or ants</td>
<td>5</td>
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<tr>
<td>Picture sign (A form of delusional misidentification)</td>
<td>Believing their mirror image is someone else either in the room or in a picture</td>
<td>5</td>
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<tr>
<td>Delusions relating to the television</td>
<td>The patient loses contact with the boundary between reality and TV, believing that events in the TV programme are happening to the patient in real life</td>
<td>5</td>
</tr>
<tr>
<td>Abandonment</td>
<td>‘You want to put me in a nursing home’</td>
<td>4</td>
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<tr>
<td>Delusional mis-identification</td>
<td>‘You are not my husband, what have you done with him?’—the belief that a familiar person has been replaced by an impostor who looks like the person</td>
<td>3</td>
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<tr>
<td>(Capgras and Fregoli syndromes)</td>
<td></td>
<td></td>
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<tr>
<td>Delusions of love</td>
<td>The belief that a prominent, famous or otherwise unreachable person is secretly in love with them</td>
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particular, earlier studies which have not considered SDLT will have been biased by the inclusion of this group of patients. There has also been a tendency to group symptoms by type and to report only the prevalence of these groups of symptoms.

Prognosis
Several studies have examined the outcome for patients with dementia and psychotic symptoms. The evidence suggests that such patients have a more rapid rate of cognitive decline, but paradoxically do not have increased mortality [3].

The aetiological data suggest an association between delusions and temporal lobe damage with frontal or parietal lobe involvement. The more rapid decline to severe cognitive impairment (temporal lobe), combined with the increased behavioural symptoms (frontal lobe), functional impairment (parietal lobe) and car-givers' stress may account for the linkage of psychotic symptoms with entry into residential care [20].

Management
The treatment of delusions in dementia is an important but remarkably neglected area. The vast number of clinical trials of treatments of the cognitive deficits of dementia contrasts with the paucity of trials of treatment for non-cognitive, and particularly delusional symptoms. There are no formal guidelines for treatment and therefore we are left with the use of general principles. Alternatives to drug therapy should be considered first; these may often be successful. The key is to educate car-givers to understand the symptoms and to remain calm; calm carers reduce the emotional response in patients, who may then have their attention diverted away from the delusional ideas. Inevitably, this will not be effective for every patient and neuroleptic medication may be required. The most important recommendation here is to use low doses of high potency neuroleptics such as haloperidol or fluphenazine. Unfortunately, many patients with dementia are prescribed low potency neuroleptics, such as thioridazine, which result in sedation, postural hypotension, and increased cognitive impairment from the anticholinergic and antihistaminic side-effects. The high potency neuroleptics do have side-effects (including extra-pyramidal symptoms and tardive dyskinesia), but at low doses these are minimized. Particular difficulties concern patients with SDLT who may suffer exaggerated adverse reactions to neuroleptics. In any dementia patient, neuroleptics should be introduced cautiously, starting at the lowest dose and titrating dose against response. Particularly severe reactions have occurred when SDLT patients are inadvertently given depot injections of neuroleptics. Risperidone is one of a new class of benzisoxazole neuroleptics that does not seem to have the haematological side-effects of the other atypical neuroleptics such as clozapine and remoxipride. Risperidone has a high potency and relatively few anticholinergic or extra-pyramidal side-effects, and is therefore suited to treating the psychotic demented patient. Clinical trials are in progress, and a few case reports have suggested its efficacy in dementia [21].

Our knowledge of the dementias has grown rapidly, particularly in the past 25 years. These advances began in the laboratory, and have been driven by interest in the genetics and neurobiology of these diseases. Changes in health and social policy are altering the patterns of care provision for dementia patients and their carers, increasing the demand for better clinical care. Delusions and other non-cognitive symptoms of dementia are very common, yet we are still only beginning to understand their associations, classifications and treatment.

References
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