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Behavioural and Psychological Symptoms in Alzheimer's Disease

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Abstract. Alzheimer's disease (AD) is not only characterised by cognitive decline, but the behavioural and psychological symptoms (BPSD) it induces are equally prevalent, with almost all patients experiencing one or two symptoms, including hallucinations, delusions, depression and anxiety. This has had a significant negative impact on patients' quality of life, carer burden, and clinical outcomes. This review synthesises clinical, neuropsychological and neurobiological findings to explore the main BPSD types and pathological features of AD from a neuroscientific perspective. It also summarises their common and symptom-specific mechanisms based on past studies. The results suggest that the mechanisms underlying the pathogenesis of these symptoms usually involve complex brain structural changes and alterations in neurotransmitter systems, at the same time, interact with each other. The nature and pathological features of BPSDs will, in turn, provide meaningful information about the underlying neurodegenerative pathological mechanisms, such as AD. Therefore, its importance as a potential diagnostic and therapeutic target should be given greater attention. In addition to this, based on several non-pharmacological therapies that are quite encouraging, this paper explores the clinical and nursing implications.

Keywords: Alzheimer's disease, clinical implications, behavioural and psychological symptoms, neuropsychology, cognitive neuroscience

1. Introduction

Alzheimer's disease (AD) is a progressive, irreversible neurodegenerative disease that accounts for 60-70% of dementia cases, and with approximately 24 million people worldwide currently living with AD, it has become a major global health problem. In the traditional context, AD is characterised by a gradual decline in cognitive functioning, particularly situational memory, followed by deficits in executive functioning, language and attention. However, studies have shown that these features do not fully reflect the clinical presentation of AD, with at least 90% of patients presenting with a variety of neuropsychiatric or behavioural and psychological symptoms (BPSD) at any stage in the course of the disease [1], mainly involving changes in a range of non-cognitive domains such as mood, behaviour and perception. These include hallucinations, delusions, anxiety, depression, agitation, apathy, and aggressive behaviour. Within this, a meta-analysis of AD showed that the overall prevalence of depression was as high as 42% and anxiety was 39%. The prevalence of delusions and hallucinations was 31% and 16% respectively [2]. The origin of these symptoms

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usually involves an interplay of biological, psychological and social factors, and most people with AD experience at least one psychological or behavioural symptom during the course of their illness.

From a modern clinical perspective, the importance and significance of studying Psychological Symptoms in AD is unquestionable due to its impact on multiple levels. Firstly, for both the patient and their carers (or family, household), BPSD significantly affects the patient's prognosis, reducing quality of life while increasing distress and confusion. Stress for their carers typically increases with the number and severity of BPSD, with just one or two symptoms resulting in carers needing to devote double the time to care. When they are distressed or overburdened, they may engage in inappropriate behaviours or communication with the patient. In addition, many carers may be unable to rest at night to care for a loved one, which can lead to a range of psychological disorders, including depression due to sleep deprivation. In addition to being associated with higher mortality rates for patients, this has become one of the main reasons for higher hospitalisation rates and earlier institutionalisation. At the same time, at the level of the healthcare system, BPSD significantly increases the complexity and economic costs of treatment, such as increased use of medications.

This paper uses a literature review approach, aims to explore behavioural and psychological symptoms in Alzheimer's disease from a cognitive neuroscience perspective, with a focus on four main domains of BPSD: hallucinations, delusions, anxiety, and depression. Specifically, the aim is to answer three key research questions: first, what are the clinical features of BPSDs in AD, and how do they evolve over the course of the disease? Second, why does AD cause these symptoms, and what are the neural mechanisms behind them? Finally, what are the implications for clinical intervention and nursing practice? To address these research questions, this study will begin with an overview of AD-related BPSD, focusing on its core pathology and natural course. In addition, this study will explore the four main symptoms mentioned above in more detail and systematically connect behavioural manifestations and pathological causes, leading to a discussion of the relevant neural mechanisms and implications from a clinical and nursing perspective.

2. Overview of Behavioural and Psychological Symptoms of Dementia (BPSD)

2.1. Definition of BPSD

AD is currently the most widely distributed neurodegenerative disease in the world and is typically characterised by memory loss and other cognitive impairments. BPSD of dementia, on the other hand, refer to a heterogeneous group of non-cognitive impairments that affect perception, thought content, mood and behaviour in people with dementia. A growing body of research has recognised BPSD as a core feature of AD, rather than merely a supplementary problem such as a response to cognitive decline or environmental stressors, and its clinical significance is on a par with that of cognitive impairment [3]. More importantly, BPSD tends to co-occur with and interact with cognitive deficits, further impairing the patient's daily functioning and complicating the clinical management of the disease.

Altomari et al. have framed it in terms of four distinct sub-syndromes: hyperactivity (including agitation, euphoria, disinhibition, irritability, and aberrant motor behaviours), psychotic symptoms (including sleep and nocturnal behavioural disturbances, delusions, and hallucinations), affective disorders (including anxiety, depression) and apathy symptoms (not limited to apathy per se, but also eating disorders). In clinical practice and research settings over the past 25 years, all examinations for the twelve BPSDs, as well as the BPSD subsyndromes, have been assessed using the Neuropsychiatric Inventory (NPI).

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2.2. Prevalence and course of BPSD in AD

The above symptoms are usually closely related to the underlying disease process. One study classified patients into three phases based on the natural course of the disease: pre-onset (T0 phase), post-onset to 5 years (T0 phase or disease manifestation), and post-onset to 5 years (T1 phase or disease progression) [1]. Assessment of the NPI has shown that, in terms of the order of neuropsychiatric symptom onset, depressive and anxiety symptoms appeared significantly earlier than the other BPSDs, and previous studies have explored the temporal relationship between anxiety-depressive symptoms and memory loss, whose early relevance in predicting AD is thought to exist [4]. This means that they can serve as an important 'early warning signal' in the detection of dementia. And as the severity of the disease increased (within T1), both hallucinations and delusions were present in significant proportions. Concurrently, symptoms may fluctuate, worsen, or alternate in their presentation.

In addition, the prevalence, incidence, and severity of BPSD vary depending on the type of patient. Specifically, there are differences in the presentation of BPSD in patients with early-onset Alzheimer's disease (EOAD, age of onset <65 years) and late-onset Alzheimer's disease (LOAD, age of onset >65 years). Statistical results point to a significantly higher overall prevalence of BPSD in patients with EOAD. However, the majority of BPSD also appeared significantly later in EOAD patients compared to LOAD [1].

2.3. General neuropathological basis

At the morphological level of neuropathology, AD is defined as a clinicopathological entity. Characterised by the accumulation of extracellular parenchymal β-amyloid (amyloid plaques) and intraneuronal hyperphosphorylated tau protein neuroprogenitor fibre tangles, accompanied and accompanied by vascular amyloid deposition, early synaptic loss, neuronal loss and brain atrophy [5].

These pathological burdens are not limited to the medial temporal lobe region but are widely distributed throughout the neocortex, limbic system, and subcortical structures, including the frontal and temporal cortex, the amygdala, and other areas associated with emotion, processing, and executive control. One study noted that AD patients are prone to emotional processing and perceptual deficits due to atrophy of the amygdala, anterior temporal cortex and orbitofrontal cortex [6]. For example, comprehension of negative emotions (including sadness, anger, and fear) is more severely impaired than comprehension of happy emotions. A study of patients with frontotemporal dementia also showed that this group consistently tends to risky or impulsive decision-making, a lack of sensitivity to future consequences, and an emotionally unresponsive response to unethical behaviours, and that such decision-making deficits are often caused by disruptions in broader neural networks [7]. These findings suggest that when brain networks involved in emotional processing and decision control are impaired, BPSD may follow, providing important clues to understanding BPSD in AD.

3. Hallucinations in AD

3.1. Clinical presentation of hallucinations

Hallucinations, a psychotic symptom in AD, are perceptual experiences that occur in the absence of an external stimulus, and are also recognised as a core clinical diagnostic feature of dementia with

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Lewy bodies. Hallucinations in patients with AD can originate from any sensory pathway and manifest primarily as visual and auditory hallucinations. Visual hallucinations are about twice as common as auditory hallucinations in several studies of people with dementia [2]. However, there have also been cases regarding olfactory, somatic and tactile hallucinations [8]. At the same time, the occurrence of hallucinations usually implies more severe cognitive impairment and faster deterioration of the disease, and is also associated with increased mortality. Typically, patients think they see people who are not there, or mistake shadows, reflections, or patterns for human figures.

3.2. How AD leads to hallucinations

Several cognitive mechanisms have been suggested to explain the hallucinations in patients with Alzheimer's disease. Firstly, inhibitory dysfunction results in a reduced ability to process perceptions, and patients experience severe confusion between sensory information and memory information. Normally, inhibitory control processes reduce the competition between appropriate and inappropriate information and suppress distracting memories. However, Hemsley's model suggests that memory loss in AD patients interacts with inhibitory processing and that patients may even use stored memories to “fill in” missing perceptual details [9]. This results in an influx of redundant or irrelevant information from memory into consciousness. Thus, the patient's hallucinations may be related to these intrusive thoughts or memory representations that are difficult to suppress. Second, a significant correlation has been suggested between hallucinations and social isolation and between hallucinations and loneliness in patients with AD [8], who use hallucinations to compensate for the lack of sensation, contact, or interaction with people, and to free themselves from the boredom and emptiness of isolation brought about by the disease, which is more pronounced in patients in institutions or nursing homes.

3.3. Neural mechanisms underlying hallucinations

The current body of research addressing this issue of AD is small. From a neurobiological perspective, a previous study based on data from the NPI scale suggested that the onset of hallucinations may be associated with a thinning of the lateral parietal cortex and reduced function [10]. However, this was questioned because the specific sensory modalities of the hallucinations were not assessed. Another study mentioned that AD patients with visual hallucinations usually have smaller occipital lobe volumes and enhanced periventricular high signal in the occipital lobe. This suggests that pathological changes in sensory processing pathways play a role in AD hallucinations [8]. Second, inadequate perfusion of the right parietal and frontotemporal lobes in AD patients may also be important for hallucinations. In addition, there are still studies that use the right anterior-posterior network and the anterior insula as core regions, aiming to argue that hallucinations are related to atrophy in this brain region [11]. And in the absence of cortical acetylcholine, currently irrelevant intrinsic and sensory information also spontaneously enters the conscious level, activating a paradoxical effect similar to induced dreaming as a potential cause of hallucinations.

3.4. Clinical and nursing implications

On a behavioural level, hallucinations can lead to several negative consequences, including, but not limited to, verbal arguments, aggressive behaviour [8]. Moreover, care strategies for people with AD generally include environmental interventions such as keeping away from high-decibel noise distractions, keeping away from objects such as mirrors that may cause reflections or have complex

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shapes, and optimising ambient light. Acceptance and commitment therapy may also be an option, avoiding direct denial of the patient's experience or critique of the hallucinatory beliefs, and reassuring language should be used to treat it as a mindfulness, which is attributed to reducing the patient's agitation and aggressive behaviour. Since there are no typical anti-AD medications licensed by the U.S. Food and Drug Administration (FDA), antipsychotic medications are used when intervening in the symptoms of AD patients. Caution should be exercised, and the benefits must be weighed against the risks of adverse effects and mortality; the tolerability of AD patients should always be closely monitored.

4. Delusions in AD

4.1. Clinical characteristics of delusions

Delusions are false beliefs that the patient holds steadfastly despite evidence to the contrary. This indicates a clear abnormality in the patient's ability to make realistic judgments. Unlike those observed in some primary psychotic disorders, the delusions of AD patients are usually simpler, repetitive, and closely related to everyday experiences. The themes of most delusions include stolen delusions, i.e., the belief that others are stealing from them. Persecutory delusions, which are beliefs that others are trying to harm them, exaggerated delusions, somatic delusions, religious delusions, jealousy delusions, and infidelity delusions. A two-factor hypothesis of delusions states that delusions are usually formed by one, a neuropsychological dysfunction that triggers the generation of a false belief, and two, a disorder that is supposed to negate or modify that belief. The former is considered to be disease-specific, as in AD, and encompasses paranoid properties. The latter is usually more prevalent [12].

4.2. Cognitive and emotional factors in delusion formation

As mentioned above, maintaining delusional thinking requires a dual attitude towards the information received. Error monitoring deficits and cognitive biases combine to form this attitude, i.e., the formation and maintenance of delusions. Firstly, memory deficits, particularly situational memory, cause patients to misinterpret everyday events [12]. Thereafter, as the patient's tolerance for uncertainty decreases, psychological self-protective mechanisms will become involved, allowing the individual to maintain a sense of coherence or self-possession despite cognitive decline. However, this is a rather rigid interpretation of the events. For example, when patients are unable to recall their actions, they may attribute misplacing items to theft.

4.3. Neural mechanisms of delusions in AD

In accordance with multiple past studies that included 253 AD patients who had not used antipsychotics and were taking antipsychotics [12]. Although patients with different types of delusions differ slightly in the areas of the brain damaged. However, if the multiple delusions are considered as the same entity, structurally, one of the main reasons for the occurrence of delusions is related to the reduction of grey matter in the right frontoparietal lobe, the left frontal lobe, the right hippocampus and the left nucleus of the pallidum. At the same time, these studies particularly mention the presence of atrophy of the left lateral and medial orbitofrontal lobes and the superior temporal gyrus in female AD patients. This implies that patients have a reduced capacity for judgment and belief revision. In addition, structural changes were found in the limbic system (especially the amygdala and thalamus). Comparison and integration of multiple neuropsychiatric

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disorders that contribute to delusions, including schizophrenia, Parkinson's and bipolar disorders, revealed that grey matter reductions in these areas are more common in patients with AD. The limbic system plays a central role in emotional processing, salience attribution, and the integration of memories with emotional meaning. Damage can directly lead to emotional overreaction and heightened perception of threat. There is still previous research suggesting that delusions are related to the functional brain connectivity, i.e., the default mode network, of AD patients in the resting state. But in any case, delusions occur based on network disruption in multiple brain regions.

4.4. Clinical and care implications

The patient tends to adopt actual behaviour based on delusional content. Translating these false beliefs into harsh accusations, avoidance, or confrontation dramatically increases stress in the caregiving process and significantly damages interpersonal relationships. It leads to a crisis of trust, conflict and stress for carers. An effective caregiving strategy is therefore one that emphasises emotional buy-in. Also, in terms of clinical perspectives, biomarker identification of delusions is crucial, attributed to its accuracy. Moreover, neuromodulatory therapies based on networks affecting brain circuits are considered a potentially promising therapeutic avenue.

5. Anxiety and depression in AD

5.1. Clinical presentation of anxiety and depression in AD

Neuropsychiatric symptoms such as anxiety and depression are also prevalent among the psychological responses manifested in the clinical course of the disease in patients with Alzheimer's disease (AD). Anxiety, which is typically characterised by nervousness, irritability, frequent wandering, and reduced participation in once-loved social activities, is considered to be linked to marked impairment in daily functioning [13]. In contrast, depression, in general, includes reduced goal-directed behaviour, guilt, despair and sadness, in addition to a socially withdrawn state of mind that shares the same symptoms as anxiety. Both symptoms are widespread in the early and middle stages of AD co-morbidity and often overlap in patients with mild AD. In other words, it can be argued that the presence of depression and anxiety in the early or even before the AD is a dangerous early warning for the onset of dementia.

The early stages of AD are characterised by a poor state of mind, where the patient perceives cognitive decline, reduced ability to understand the environment or fear of future uncertainty. and react emotionally accordingly, while anxiety is the resulting compensatory behaviour. Thus, the literature confirms that at this stage, anxiety does not originate from the brain injury or primary, but is more a psychological response to the loss of independence, the loss of a sense of control over one's own body and the ability to take care of oneself, caused by the difficulty of adapting to the disease [13]. In this scenario, the patient will have repetitive questions in their behaviour and be highly sensitive to unfamiliar environments, as well as develop some degree of separation anxiety from their caregivers.

Research suggests that there are differences in the occurrence of anxiety and depression as the disease progresses. Anxiety in early AD may reflect fear and uncertainty, whereas depression may emerge as cognitive decline becomes more pronounced and perceived as irreversible. Anxiety may also translate into depression. Several mechanisms have been proposed to explain this pattern. First, the persistent experience of failure triggered by daily routines can bring about feelings of helplessness. And as cognitive decline becomes more pronounced in AD patients, anxiety triggered

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by uncertainty may evolve into depression characterised by negativity. At the same time, social detachment (often caused by communication difficulties or declining self-confidence) can further exacerbate this emotional downturn. In addition, at the neurobiological level, the progressive dysfunction of reward circuits with the disease may also reduce the ability to feel pleasure or gain motivation.

Indeed, the association between depression and dementia has sparked an ongoing debate about its underlying causes and causality [14]. Recent research evidence suggests that depression is a certain susceptibility risk factor for dementia typified by AD, capable of acting as a prodromal symptom. Moreover, depressive symptoms in AD patients are usually different in nature from depression in individuals with normal cognitive functioning. It is intended that the former do not necessarily have intense sadness, but more apathy and loss of interest. In contrast, patients may show less self-blame as well as more restricted emotional expression.

5.2. Shared and distinct neural mechanisms of anxiety and depression in AD

Anxiety and depressive symptoms in AD patients share some common underlying neural mechanisms. Firstly, they may arise from neurodegenerative lesions of neural regions and circuits responsible for mood, which can lead to effects on the hypothalamic-pituitary-adrenal axis (HPA axis), which, according to the 'glucocorticoid cascade hypothesis', can be triggered by excessive glucocorticoid-induced neurotoxicity, triggering over-activation of the HPA axis in a state of impaired negative feedback, which will trigger anxiety and depressive symptoms, which in turn exacerbate cognitive decline, constituting a vicious circle [13,14]. Furthermore, neurotransmitter imbalances play a role, with alterations in norepinephrine and serotonin involved in the pathogenesis of depression and anxiety in AD. Of these, the former has potent anti-inflammatory, neuroprotective and neurotrophic effects. Additionally, they are believed to have effects on amyloid beta protein (Aβ) plaque deposition and production, respectively. Higher brain Aβ load is associated with stress responses to depressive and anxiety symptoms in AD.

Nonetheless, there are differences between the two. Hyperperfusion and atrophy of some of the brain regions associated with the reward system and motivation functions, as well as alterations in receptors such as NMDA, are strongly associated with depressive symptoms [13]. In contrast, the onset of anxiety seems to be more closely related to overactivity of the threat processing system.

5.3. Clinical and nursing implications for affective symptoms

As described in the vicious circle above. Therefore, recognising symptoms of depression and anxiety at an early stage, including subclinical features, and following up with targeted treatment can help to control the progression of AD. Although antidepressants can promote neurogenesis and effectively suppress psychological symptoms while attenuating tau protein pathology to limit the onset of dementia [14]. However, non-pharmacological therapies are currently recommended as the first-line treatment option. Pharmacological treatments are used when non-pharmacological treatments are ineffective, due to the serious side effects associated with their long-term use. The medical community is currently working on the development of newer therapies that are more tolerable, such as non-invasive neurostimulation or art therapy [2]. In addition, stabilising the external environment while maintaining a routine can help to reduce anxiety. Similarly, interventions should focus on emotional validation rather than behavioural modification. Raising wider awareness of depressive symptoms and recognising patients' distress in the caregiving process is crucial. Attention should also be given to supporting carers (family members) to avoid overstressing them.

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6. Implications for clinical practice and nursing care

In a factor analysis of patients with AD, all symptom clusters of BPSD were found to have a direct and profound impact on the burden of care in the family's community healthcare. In particular, a study noted that psychosis was the most influential symptom cluster based on a classification of three symptom clusters of BPSD [15], including hallucinations, delusions, anxiety and depression, which are the main ones examined in this paper. The condition of people with AD is chronic and long-term, and over time, the higher the burden on the carer, the higher the probability of developing neuropsychiatric symptoms. Caregivers of people with AD are often considered invisible patients for these reasons. Therefore, the care process for AD patients with BPSD should be more scientifically based to reduce the burden and enhance overall well-being for both the caregiver (within the family) and the patient.

Firstly, the key basis that should be recognised is that BPSD in people with AD should not be interpreted as intentional behaviour. They are inherent to the underlying or ongoing disease process. Categorising these behaviours as purely intentional or even a personality trait of the patient may lead to inappropriate responses and behaviours, thereby increasing conflict. In this context, non-pharmacological interventions such as environmental modifications are the treatment of choice, as discussed above [2]. In addition, research has pointed out that communication strategies are crucial and emotionally oriented coping strategies can reduce burnout and psychological burden for people with AD and even caregivers [15]. Caregivers should focus on the intrinsic emotional needs of the patient. In addition, BPSD manifests itself differently in different disease stages. Doctors and social workers should provide personalised and individualised care for this group based on a professional medical model in addition to a person-centred approach, which also relies on the prevalence of the social welfare system. In addition to the crucial role caregivers play in patients' daily lives and the need for appropriate education and training, healthcare professionals have a responsibility to provide support through ongoing assessment, guidance, and interdisciplinary collaboration. Finally, it is equally crucial to reduce the external stigma attached to symptoms.

7. Conclusion

In summary, AD, a classic neurodegenerative disorder, is closely associated with a wide range of BPSD. This paper focuses on a review of recent research around hallucinations, delusions, depression and anxiety. These symptoms are not incidental features of the disorder, but rather core manifestations of the disease. Of these, depression and anxiety are more common in the preclinical and/or early stages of dementia, with hallucinations and delusions tending to appear later in comparison. There are different but overlapping neurological mechanisms for the different symptoms based on neurodegeneration of the regions and circuits responsible for emotions, such as impaired frontotemporal lobe function.

However, there are still limitations to the existing research in this area. The neural mechanisms underlying the neuropsychiatric symptoms of AD have not been fully elucidated. Instead, in addition to cross-sectional clinical observations, further research is needed, especially studies with a longitudinal design that encompasses all stages of the disease process. Additionally, psychosocial factors should be incorporated into a comprehensive neuropsychological assessment.

In addition, future studies could prospectively correlate the appearance of different symptoms of BPSD in AD with more refined neuroimaging methods. This will further capture the dynamic changes in brain networks during the disease process and refine the neurobiological and neuroanatomical mechanisms. As more and more emerging therapies become available, it is equally

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important to develop more person-centred and humane models of care. At the same time, as some of the symptoms in turn lead to neurodegeneration, BPSD should be considered an important target for intervention at an earlier stage.

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