

Anxiety Symptoms in Dementia: A Caregiver's Guide

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Introduction to Anxiety and Dementia

The co-occurrence of generalized anxiety and symptoms of anxiety within the context of progressive neurocognitive disorders, collectively known as dementia, represents a significant clinical challenge in geriatric psychiatry and neurology. While dementia is fundamentally characterized by a decline in cognitive functions, including memory, language, and executive abilities, the presence of Behavioral and Psychological Symptoms of Dementia (BPSD) is nearly universal, and **anxiety** stands out as one of the most prevalent and often underestimated components. Estimates suggest that clinically significant anxiety affects between 20% and 50% of individuals diagnosed with various forms of dementia, including Alzheimer's disease (AD), vascular dementia, and Lewy body dementia. The difficulty lies not only in the high incidence but also in the diagnostic complexity, as the patient's reduced ability to articulate internal emotional states means anxiety often manifests through observable behaviors rather than verbalized worry or fear.

Understanding the relationship between anxiety and neurodegeneration requires acknowledging a complex, potentially bidirectional feedback loop. Anxiety can precede the formal diagnosis of dementia by many years, particularly in individuals with Mild Cognitive Impairment (MCI), acting as a potential risk factor or early prodrome for AD. Conversely, as cognitive function erodes, the resulting confusion, disorientation, loss of autonomy, and difficulty processing new information create an environment ripe for the development of severe anxiety. The individual becomes increasingly aware of their functional limitations, leading to feelings of helplessness and profound insecurity, which directly fuel anxious responses. This interaction necessitates a refined clinical approach that looks beyond purely cognitive assessment to address the affective burden of the disease.

Accurate identification of anxiety in this population is crucial because untreated anxiety significantly diminishes the individual's quality of life, exacerbates cognitive decline, and increases the risk of other BPSD, such as agitation and aggression. Standardized anxiety scales developed for cognitively intact populations are often unreliable in moderate to severe dementia due to reliance on self-report and abstract reasoning. Therefore, clinicians and caregivers must shift their focus toward observing subtle behavioral cues, physiological changes, and contextual triggers that signal underlying distress, recognizing that anxiety in dementia is frequently a response to an overwhelming or unpredictable environment rather than a standalone psychiatric disorder.

The Neurobiological Overlap of Anxiety and Dementia

The neurobiological substrate underlying the frequent co-occurrence of anxiety and dementia involves shared pathology within key brain regions responsible for emotional regulation, memory consolidation, and threat detection. Central to this overlap is the **limbic system**, particularly the **amygdala**, which serves as the brain's primary alarm center, processing fear and generating

anxiety responses. In AD, the amygdala is often one of the earliest structures to accumulate neurofibrillary tangles and amyloid plaques, leading to structural and functional abnormalities. Hyperactivity or dysregulation of the amygdala, often seen in anxiety disorders, is exacerbated by neurodegenerative processes, resulting in an increased sensitivity to perceived threats and a reduced threshold for generating fight-or-flight responses, even in benign situations.

Furthermore, the integrity of the prefrontal cortex (PFC) is vital for modulating and inhibiting limbic responses. As dementia progresses, the PFC, responsible for executive functions and rational appraisal, suffers significant atrophy and functional disconnection from subcortical structures. This loss of inhibitory control means that the anxious signals generated by the amygdala are not effectively dampened, leading to persistent, generalized worry and restlessness. Neurotransmitter systems are also profoundly impacted; deficiencies in the inhibitory neurotransmitter **GABA** (gamma-aminobutyric acid) contribute to neuronal hyperexcitability, mirroring the pathophysiology found in generalized anxiety disorder. Simultaneously, alterations in the serotonergic system (5-HT) originating from the raphe nuclei, and increased activity in the noradrenergic system (norepinephrine), contribute to heightened arousal, vigilance, and somatic symptoms of anxiety.

The concept of chronic stress and the resulting hypothalamic-pituitary-adrenal (HPA) axis dysregulation also links the two conditions. Chronic anxiety maintains elevated levels of **cortisol**, a stress hormone that is neurotoxic, particularly to the hippocampus--a structure critical for memory and contextual fear processing. This chronic stress state accelerates hippocampal volume loss and exacerbates the formation of amyloid pathology, thereby linking psychological distress directly to the physical progression of the neurodegenerative disease. Understanding these shared mechanisms highlights that anxiety is not merely a psychological reaction to dementia but an intrinsic component of the underlying brain pathology, demanding targeted neurobiological interventions alongside psychosocial support.

Behavioral Manifestations of Anxiety in Dementia

In the absence of reliable verbal self-report, anxiety in dementia patients often translates into observable behaviors that can be misinterpreted as general agitation, restlessness, or resistance to care. One of the most common manifestations is **pacing and wandering**, which is frequently driven by a need to fulfill an unmet emotional need, typically security or familiarity. The individual may pace repetitively along a specific path or search incessantly for a person, object, or location that represents safety, signaling a feeling of fundamental unease or fear about their current environment. This searching behavior is a concrete attempt to resolve the abstract feeling of anxiety that they can no longer cognitively process or articulate as "I am worried."

Another hallmark behavioral symptom is **repetitive questioning**, often regarding time, location, or the imminent arrival of a loved one. This behavior is a coping mechanism intended to re-establish

cognitive anchors in a confusing reality. Although frustrating for caregivers, the repetitive nature stems from the immediate dissipation of the reassurance provided, due to short-term memory impairment. The question is asked not for new information, but for the momentary comfort provided by the interaction and the confirmation of safety. Furthermore, anxiety can manifest as **clinging behavior** or shadowing, where the patient follows the primary caregiver closely, expressing profound distress when separation occurs, often interpreted as separation anxiety stemming from a deep fear of abandonment in a world they perceive as increasingly hostile or unpredictable.

In more pronounced cases, anxiety can trigger **catastrophic reactions**--intense emotional outbursts disproportionate to the stimuli, such as screaming, hitting, or extreme withdrawal, often precipitated by minor environmental changes, transitions, or perceived demands. These reactions are instantaneous expressions of overwhelming fear and inability to cope. Clinicians must also observe less dramatic, but equally significant, indicators of anxiety that relate to routine activities. These may include a refusal to bathe or change clothing, not because of obstinance, but due to anxiety related to the vulnerability, temperature changes, or sensory overload associated with the task. Key behavioral indicators frequently observed include:

Restlessness: Fidgeting, inability to sit still during mealtimes or activities.

Excessive Checking: Repeatedly checking doors, windows, or pockets for items.

Hoarding or Hiding: Accumulating objects (often meaningless) driven by a need for control and security.

Refusal of Care: Resistance to personal care tasks due to fear of the unknown or invasion of personal space.

Physiological and Somatic Indicators

When the ability to verbally communicate internal distress is compromised by dementia, anxiety often finds expression through the autonomic nervous system, manifesting as a range of physiological and somatic symptoms that can easily be misattributed to primary medical conditions or the normal aging process. The activation of the sympathetic nervous system triggers classic fight-or-flight responses, including noticeable increases in **heart rate (tachycardia)** and **respiratory rate** (rapid, shallow breathing). These symptoms, if chronic, can place undue strain on the cardiovascular system and are often documented as unexplained physical discomfort or distress rather than recognized as psychological anxiety. Careful monitoring of vital signs during stressful events, such as transitions or unfamiliar visitors, can help link these physical changes directly to emotional triggers.

Gastrointestinal disturbances are also highly common somatic expressions of anxiety. Patients may experience unexplained nausea, loss of appetite, or significant changes in bowel or bladder habits, such as frequent urination or functional constipation. These physical complaints, while

potentially having medical origins, should be investigated as potential manifestations of chronic stress and anxiety, particularly when they coincide with periods of high environmental stress or changes in routine. Furthermore, increased **muscle tension** is a pervasive sign, often evidenced by a rigid posture, clenched fists, or continuous fidgeting, which reflects an inability to relax and a state of hypervigilance. The patient may complain of generalized aches and pains (arthralgia or myalgia) that do not respond well to standard analgesics, suggesting a somatization of emotional distress.

Sleep architecture is profoundly affected by anxiety in dementia. Patients frequently suffer from **initial insomnia**, finding it difficult to fall asleep due to racing thoughts or generalized worry, or they may experience frequent nighttime awakenings accompanied by disorientation and panic. This nocturnal anxiety is often intensified during the "sundowning" phenomenon, where cognitive symptoms and BPSD worsen in the late afternoon and evening hours. The resulting chronic sleep deprivation not only reduces the patient's immediate functional capacity but also exacerbates the neurocognitive decline and lowers the threshold for anxious reactions during waking hours, creating a vicious cycle that demands integrated therapeutic strategies focusing on both sleep hygiene and anxiety management.

Differential Diagnosis Challenges

Differentiating anxiety symptoms from other common BPSD and co-occurring psychiatric conditions poses a significant diagnostic hurdle in dementia care, given the overlapping symptom profiles and the patient's reduced capacity for nuanced self-reporting. A primary challenge involves distinguishing anxiety from **major depressive disorder (MDD)**, which frequently co-exists with dementia. While both conditions involve significant emotional distress, depression is often characterized by pervasive sadness, anhedonia (loss of pleasure), and psychomotor retardation (slowness), whereas anxiety typically involves restlessness, hyperarousal, and future-oriented worry. However, many dementia patients exhibit a mixed presentation, known as anxious depression, where restlessness and tearfulness coexist with apathy and withdrawal, complicating the precise determination of the primary affective disturbance.

The differentiation between anxiety and **agitation** is also clinically critical. Agitation is generally defined as excessive motor activity associated with verbal or physical aggression, whereas anxiety is characterized by fear, tension, and worry. While severe anxiety can certainly lead to agitated behaviors (e.g., pacing rapidly while crying), the underlying emotional driver is different. If the behavior is clearly a response to a perceived threat or fear (e.g., fear of being left alone), it is primarily anxiety-driven. If the behavior is non-goal-directed, aggressive, or purely reactive without an identifiable fear trigger, it leans toward agitation. Clinicians must strive to identify the antecedent to the behavior to correctly categorize the symptom and select the appropriate intervention.

Furthermore, anxiety must be differentiated from **delirium** and **psychotic features**. Delirium, an acute confusional state, often involves rapid fluctuations in consciousness, disorganized thought, and severe perceptual disturbances, which are distinct from the more consistent, chronic tension found in generalized anxiety. Distinguishing anxiety from psychosis (e.g., paranoia) is subtler. A patient experiencing anxiety might fear that their family has forgotten them (a realistic worry exacerbated by memory loss), whereas a patient experiencing a paranoid delusion might believe staff members are actively plotting to poison their food (a fixed, false belief). While intense fear is present in both, the cognitive content and reality testing involved help delineate the underlying pathology, guiding whether anxiolytics or antipsychotics are the more appropriate treatment route.

Impact on Quality of Life and Caregiving

The presence of chronic, untreated anxiety profoundly erodes the quality of life (QoL) for the individual living with dementia. Anxiety prevents the patient from engaging fully in therapeutic activities, social interactions, and daily routines, leading to functional decline and social isolation. When every new experience or change in routine is met with intense fear or catastrophic reaction, the patient naturally retreats, preferring the perceived safety of isolation, which, paradoxically, accelerates cognitive and physical deterioration due to lack of stimulation. This emotional distress is highly correlated with increased disability, reduced mobility, and a higher reliance on institutional care, as the emotional burden of constant worry and tension outweighs the patient's remaining cognitive reserves.

The consequences of patient anxiety extend dramatically to the **informal caregiving system**, typically family members or spouses. Anxiety-driven behaviors, such as incessant questioning, clinging, and resistance to care, are emotionally exhausting and highly stressful for caregivers. Unlike physical symptoms, emotional distress is continuous and unpredictable, leading to high levels of caregiver burden, depression, and burnout. Studies consistently show a strong correlation between the severity of the patient's BPSD, particularly anxiety and agitation, and the caregiver's decision to pursue long-term care placement. The inability to soothe or reassure a perpetually worried loved one is often cited as the most difficult aspect of dementia care, contributing significantly to caregiver morbidity and reduced self-efficacy.

From a healthcare systems perspective, anxiety symptoms in dementia are associated with substantial economic costs. Patients exhibiting high levels of anxiety are more likely to require frequent emergency room visits, utilize psychotropic medications (often inappropriately prescribed), and necessitate higher staffing ratios in institutional settings. The pervasive nature of the symptoms requires intensive, individualized care plans, often involving consultation with specialists in geriatric psychiatry or behavioral medicine. Effective management of anxiety in the early and middle stages of dementia is therefore critical not only for ethical reasons but also as a preventative measure to reduce the downstream costs associated with crisis management and

premature institutionalization.

Pharmacological and Non-Pharmacological Management Strategies

The management of anxiety in dementia requires an integrated, person-centered approach that prioritizes non-pharmacological interventions before resorting to psychotropic medication. The foundation of non-pharmacological management lies in **environmental modification** and **routine standardization**. Reducing sensory overload (e.g., minimizing noise, harsh lighting, and clutter) helps to reduce triggers for anxious reactions. Establishing highly predictable daily routines provides cognitive anchors, reducing the uncertainty and confusion that fuel anxiety. Techniques such as **validation therapy**, which acknowledges and validates the patient's feelings without necessarily correcting their perception of reality, can be highly effective in reducing immediate distress and catastrophic reactions.

Specific structured non-drug interventions include tailored adaptations of psychological therapies. While traditional Cognitive Behavioral Therapy (CBT) is generally unsuitable for moderate to severe dementia due to cognitive impairment, modified approaches focusing on emotional and behavioral regulation can be beneficial for those in the mild stage. These adaptations often involve simplifying instructions, focusing on relaxation techniques (e.g., deep breathing, guided imagery), and using sensory stimulation like **music therapy**, pet therapy, or aromatherapy to promote calmness and reduce physiological arousal. The goal is not to restructure distorted thoughts, but to provide immediate, accessible coping tools and sensory input that override the anxious state.

When non-pharmacological strategies prove insufficient for severe or persistent anxiety, pharmacological interventions may be necessary, but they must be approached with extreme caution. The use of **Benzodiazepines** (e.g., lorazepam) is generally discouraged for chronic anxiety in dementia due to the significant risks of sedation, increased fall risk, cognitive impairment, and potential paradoxical agitation. Instead, **Selective Serotonin Reuptake Inhibitors (SSRIs)**, such as citalopram or sertraline, are typically considered the first-line psychotropic treatment for generalized anxiety in this population, given their favorable safety profile relative to older anxiolytics and their efficacy in treating co-morbid depression. Treatment must always begin at the lowest possible dose, with careful monitoring for adverse effects and a clear plan for discontinuing the medication if benefits are not observed within a defined period.