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Review Article

Behavioral and Psychological Symptoms of Dementia

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Abstract

More than 50% of people with dementia experience behavioral and psychological symptoms of dementia (BPSD). The development of BPSD is associated with a more rapid rate of cognitive decline, greater impairment in activities of daily living, and diminished quality of life. The most prevalent BPSD are apathy, depression, irritability, agitation and anxiety, The first step to better understand the psychiatric manifestations of dementia is to appropriately recognize and describe types of behavioral and psychological symptoms of dementia.

The delusions are typically less complex and organized. Visual hallucinations are particularly common in subjects with dementia with Lewy bodies. Wandering is a frequent behavioral disorder in demented patients. The basis of wandering is multifactorial; biomedical, psychosocial and person-environment factors must be considered. Aggressiveness or aberrant motor behavior has been more frequently reported in men with dementia. Appetite changes are particularly frequent in frontotemporal dementia.

BPSD may respond to those environmental and psychosocial interventions, however, drug therapy is often required for more severe presentations. There are multiple classes of drugs used for BPSD, including antipsychotics, anticonvulsants, antidepressants, anxiolytics, cholinesterase inhibitors and NMDA modulators. It was concluded that there were small but statistically significant benefits using aripiprazole, olanzapine, and risperidone in the treatment of BPSD. The guideline is to minimize antipsychotic use in older people with dementia, to initiate antipsychotics only in patients with severe distress, and to limit the dose and treatment duration.

INTRODUCTION

Dementia, with Alzheimer's disease as the most common cause, is a progressive illness affecting cognitive functions. The cognitive impairment is often accompanied by behavioral and psychological symptoms of dementia (BPSD). The most prevalent BPSD are apathy, depression, irritability, agitation and anxiety, while the rarest are euphoria, hallucinations, and disinhibition. Importantly, 50% of patients have at least four neuropsychiatric symptoms simultaneously [1]. It is estimated that almost all older individuals with dementia will develop BPSD at some point during progression of their illness. The behavioral problems rather than the cognitive problems are important contributing factors for caregiver burden and institutionalization. Psychotic symptoms and disruptive behaviors have been reported to be the most burdensome to caregivers [2]. Thus, in addition to cognitive deterioration, BPSD are a relevant and meaningful clinical target for intervention. Management of BPSD is very complex for families and health care professionals. Use of antipsychotics for the management of BPSD is controversial due to limited efficacy and the risk of serious adverse effects. Additionally, comorbid neuropsychiatric symptoms have been associated with worse cognitive performance and functional disability in MCI subjects [3]. Clinical psychoneuropathologic features and management, which can be burdens for patients and caregivers, were reviewed especially focusing on the most common type of dementia, Alzheimer's disease.

CLINICAL AND PSYCHONEUROPATHOLOGIC FEATURES OF BPSD

Neuropsychiatric symptoms in patients with dementia are heterogeneous and largely unpredictable, affecting the emotional experience, thought content, perception, and motor function. The first step to better understand the psychiatric manifestations of dementia is to appropriately recognize and describe types of behavioral and psychological symptoms of dementia

Neuropsychiatric symptoms of dementia occur in syndromes identified as psychosis, agitation, aggression, depression, anxiety, apathy, disinhibition, motor disturbance, night-time behaviors, and appetite and eating problems (Table 1).

Table 1: Types of behavioral and psychological symptoms of dementia.

Symptoms
Delusions
Hallucinations
Agitation
Aggression (physical or verbal)
Depression or Dysphoria
Anxiety
Apathy or indifference
Disinhibition
Irritability or lability
Motor disturbance
Night-time behaviors (waking and getting up at night)
Appetite or eating change

Although these symptoms are seen almost generally in dementia, regardless of the underlying cause, some types of dementia are associated with certain behaviors. Depression is more common in vascular dementia and hallucinations are seen more often in Lewy body dementia [4].

Patients with fronto temporal dementia often exhibit behavioral changes, such as disinhibition, wandering, social inappropriateness, and apathy.

These symptoms occur across all stages of dementia, although their type and prominence depend on the stage. Anxiety and depression are common in early stage Alzheimer's disease and may worsen with progression. Agitation is common, persistent, and may increase with disease severity. Apathy is commonly reported by family members and caregivers across all stages of dementia and tends to worsen over time, whereas delusions, hallucinations, and aggression are more episodic and more common in moderate to severe stages of the disease.

Psychotic symptoms

Delusions are false beliefs based on incorrect inference about external reality that persist despite the evidence to the contrary and these beliefs are not ordinarily accepted by other members of the person's culture or subculture. Hallucinations are perceptions in the absence of external stimuli.

The delusions are typically less complex and organized than those observed in non-demented psychotic patient and the usual content of delusional thoughts involves suspiciousness, abandonment, and misidentification [5]. Common examples: people are coming into the home and hiding or stealing objects, the place in which one is residing is not one's home, conviction that spouse is an imposter, believes that other persons have acted with any malice.

Visual hallucinations are particularly common in subjects with dementia with Lewy bodies (DLB). They are recurrent, and typically consist of well formed images of animals or persons that the patient describes in detail [6].

An evidence based review of the psychopathology of FTD showed rare occurrence of delusions and hallucinations [7].

Cholinergic deficits have been described for hallucinations and delusions in both AD and DLB [8], thus providing a rationale for the therapeutic use of cholinergic drugs to treat these symptoms.

Alzheimer's disease: Psychosis, occurs in a subset of AD patients during progression of the disease [9,10]. Hallucinations can occur in any modality, but are typically visual [11-13]. They are usually associated with greater cognitive impairment [14-16]. Common delusions are of persecution, infidelity, abandonment, and delusions are rarely observed at terminal stages of dementia [17]. AD with psychosis is often associated with other psychiatric and behavioral disturbances, the most frequent and troublesome of which are agitation and aggression [18,19].

Psychotic symptoms in demented patients usually demonstratepreferential involvement of the frontal lobe and limbic regions [20]. Anatomically, these changes partially coincide with cholinergic and dopaminergic pathways supporting, together with neurochemical and pharmacological evidence, the role of acetylcholine and dopamine imbalance in the pathogenesis of AD psychosis [21].

Neuropathology Early studies on neuropathological correlates of psychosis in AD showed significantly increased densities of neuritic plaques (NP) and neurofibrillary tangles (NFT) in the presubiculum and middle frontal cortex, respectively. This finding was consistent with the increased rate of cognitive decline that accompanies behavioral and psychotic disorders [22]. In a prospective clinicopathologic study of 56 patients with autopsy-confirmed AD, AD patients with auditory hallucinations or delusions had significantly higher neuron numbers in the parahippocampal gyrus and lower cell counts in the dorsal raphe nucleus than patients without these symptoms. Delusional misidentifications (e.g. the Capgras-type and the 'phantom boarder' symptoms) were associated with lower neuron numbers in the hippocampal area CA1 [23]. A recent semiquantitative study of neuropathological changes in postmortem hippocampus reveled that an increased tangle load was associated with increased severity of aggressive behaviors and presence of chronic aggression, suggesting a pathogenic link between tangle load in the hippocampus and aggressive behavior [24]. The hippocampus, along with associated structures in the temporal lobe, is particularly vulnerable to Alzheimer's disease and shows significant pathology even in early stages of disease [25]. In addition to the well-established roles in learning and memory formation, the hippocampus is also known to be involved in aggressive behaviors and trait [26,27]. Further studies are needed to delineate and assess these potential pathogenic mechanisms.

Lewy body disease: Psychotic symptoms are frequent and disabling in patients with LB disease [28]. They include complex hallucinations and delusions of a paranoid type [29].

Neuropathology Clinicopathologic studies have consistently reported an higher frequency and earlier onset of visual hallucinations in subjects with Lewy pathology than in demented subjects with AD [30,31]. In a community-based clinicopathologic study of 148 demented subjects, subjects with visual hallucinations had significantly more frequent Lewy-related pathology than those without visual hallucinations. In addition, a higher frequency of visual hallucinations was observed in subjects with neocortical LBs than in subjects with

limbic-, amygdala- or brainstem-predominant LB pathology [32].

Among 129 cases of pathologically proven Parkinson's disease (PD), patients with visual hallucinations had significantly higher LB scores (7.7) than those without visual hallucinations (6.6; P = 0.02) [33]. Other studies showed an association between neuronal loss and the severity of $^{\alpha}$Syn deposition in the intralaminal nuclei of the thalamus [34]. A recent study of 162 autopsy-confirmed PD cases showed a relationship between cerebral amyloid angiopathy (CAA) in the occipital cortex and visual hallucinations during life that was not seen in non-PD cases, confirming the suggestion that pathology within the primary visual system may play a role in the pathogenesis of visual hallucinations [35].

Affective symptoms

As the symptoms of depression are frequently masked by dementia, the patient rarely is able to express the typical pathological feelings of sadness, preoccupation with depressing topics, hopeless and loss of self esteem. Both depression and elated mood are commonly associated with irritability, which can be aggravated by hunger, sleepiness, and pain. Affective lability is characterized by rapid emotional shifts, within seconds or minutes.

Several lines of evidence suggest that depression shares pathophysiological routes with dementia. It has been hypothesized that chronic depression may accelerate neurodegenerative changes of AD as a result of the neurotoxic effects of elevated cortisol levels in the hippocampus [36]. A disturbed serotoninergic system has been associated with depressive symptoms in AD as several areas of the brain exhibit decreased serotonin concentration, with a significant reduction in 5-HT1 and 5-HT2 receptors throughout the cerebral cortex [29]. Similarly, loss of noradrenergic cells in consequence of degeneration of the locus coeruleus is also more frequently seen in dementia subjects with depressive symptoms subjects than without depressive symptoms [37]. Changes of GABAergic plasma levels observed in final stages of AD have also been associated with depression, apathy, and aggressive behaviors [38].

Neuropathology Depression is little known about its neuropathologic correlates. Patients with depression had more neurofibrillary tangles and neuritic plaquesin the hippocampus than patients without depression. AD patients with depression had more advanced stage of neurofibrillary tangles than patients without depression [39].

Apathy

Apathy has been defined as a disorder of motivation with additional loss or diminished goal-directed behaviors, cognitive activities and emotions [40]. Apathy may be mistaken for depression because both symptoms can manifest themselves as diminished interest, slowing and lack of energy [41].

Studies of the neuropathological correlates of apathy (as assessed with the NPI) in 29 autopsied subjects with definite AD found that chronic apathy was significantly associated with NFT in the anterior cingulate cortex [42]. In a study of 31 autopsy patients with a diagnosis of definite AD, a significant association between apathy and the amount of NFT in the left anterior cingulate gyrus was reported [43].

Wandering

Wandering is a frequent behavioral disorder in demented patients and one of the most exhausting for caregivers. Wandering refers to seemingly aimless ambulation, often with observable patterns such as lapping, pacing, or random ambulation. It has been associated with negative consequences such as higher morbidity and mortality [44]. Peak incidence of ambulation in the nursing home occurs between 5 p.m. and 7p.m [45].

The basis of wandering is multifactorial; biomedical, psychosocial and person-environment factors must be considered. According Lee et al., [46] negative emotional expression and higher cognitive status were negatively related to wandering rates after controlling for other predictors. They noted that one possible explanation for these results is that persons with dementia who are sad or angry may respond by sitting alone or staying in their rooms for periods, rather than walking around.

Wick and Zanni reported that nursing home residents who wander have double the risk of fracture compared with residents who do not wander [44]. Wandering behavior has been shown to be a key determinant of patient's death [47]. Wandering behavior seems innocent enough, but it is exhausting for the family and clinicians as well as for health-care policy makers, because accidents, getting lost, malnutrition, weight loss, fatigue, sleep disturbance, social isolation, earlier institutionalization, and increased the risk of fall are associated with wandering.

Wandering correlates with the severity of cognitive impairment [48], problems in recent and remote memory, orientation to time and place, and the ability to respond appropriately to a given conversation topic. According to Hope et al., [49] wandering behavior occurs in patients who have scored 13 or less on the Mini-Mental State Examination and it lasts for a period of several years [50]. Patients with AD are more likely to be wanderers than those with vascular dementia [51].

Psychopathology Wandering and delusions were the only clinical predictors of aggressive behavior in a case mix of Alzheimer's patients [52]. Anxious states may justify wandering behavior : an anxious resident may move about in an effort to relieve anxiety, discomfort, or unsettled state, but such motivation may be unknown to observers, so the movement may be perceived as aimless. Moderate to severe depression was found more frequently in demented wanderers [53].

Management The first step in any systematic investigation of wandering involves reliable and valid measurement of the phenomenon. Accurate diagnosis of underlying psychiatric and physical conditions that may be triggering wandering is essential. Traditional management comprised physical barriers and physical restraints, but a new method in the management of wandering has evolved with a move towards prompting safe walking rather than preventing wandering. According to Schonfeld et al., [54] use of interventions such environmental alterations and social therapeutic activities may help create a safe and pleasant environment for staff and residents. A walking

program for physically active persons with severe dementia reduced interpersonal tension on the dementia unit of a nursing home [55].

Several medications are used on residents, who exhibit wandering, but strong evidence for their effectiveness is lacking and they can produce side-effects. According to Meguro et al., [56] risperidone has some benefit over placebo in reducing wandering, but it is increases fall risk.

Other symptoms

Agitation has been defined as 'inappropriate verbal, vocal, or motor activity that is not judged by an outside observer to result directly from the needs or confusion of the agitated individual. Aggressiveness or aberrant motor behavior has been more frequently reported in men with dementia where female gender has been associated with depressive/anxious symptoms and verbally agitated help-seeking behavior [57].

Sleep pattern changes included hypersomnia, insomnia, fragmented sleep, and rapid eye movement sleep behavior disorder. Patients with dementia often show daytime napping and night-time awakening associated with poor quality of sleep [58]. Several factors such as pain, need to urinate during the night, diuretics, as well as coffee and bronchodilators, may contribute to this problem.

Appetite changes, e.g., preference for sweets, are particularly frequent in fronto temporal dementia. Most

dementia patients lose weight which can be due to hyper metabolism and inflammatory processes, in relation with hormonal disturbances [59,60].

ASSESSMENT

The assessment of neuropsychiatric symptoms requires a thorough examination to collect specific and detailed information about the past history, patient's subjective experiences, and objective behavior. Information from a family member or caregiver is essential to obtain characterization of neuropsychiatric disturbances.

The first behavior rating scale for AD was the BEHAVE-AD [61], evaluating the presence and severity of 25 behavioral symptoms in 7 symptomatic categories (paranoid and delusional ideation, hallucination, activity disturbances, aggressiveness, sleep disturbances, affective symptoms, and anxieties and phobias), and providing a global rating of caregiver burden. Currently, one of the most extensively used instruments to assess BPSD is the Neuropsychiatric Inventory (NPI) whose validity and reliability has been well established in several languages [62]. It consists of a semi structured interview retrospectively assessing 12symptoms based on the caregiver information: delusions, hallucinations, agitation, depression, anxiety, apathy, irritability, euphoria, disinhibition, aberrant motor behavior, night time behavior disturbances, and eating behavior abnormalities.

MANAGEMENT

Nonpharmacological treatment options

Possible nonpharmacological options traditionally suggested by the guidelines include aromatherapy, multisensory stimulation, therapeutic use of music, animal-assisted therapy, and massage, but often come with the recommendation that more research is needed. Benefits from psycho-educational interventions for caregivers were documented to be long-lasting, especially when delivered individually [63].

For cognitive stimulation/training, there was moderate quality of evidence, with a mild to moderate effect on specific outcomes, such as cognitive function, activities of daily living (ADL), behavior, and mood. For reminiscence therapy, the studies included in the systematic review showed positive results on cognition, behavior, and ADL. For aromatherapy, reviews found some positive albeit insignificant effects for reduction of agitation in dementia. There was poor supporting evidence and conflicting results for massage and music therapy, resulting in insufficient evidence to make a recommendation. For light therapy, there was no evidence of effectiveness with regard to cognition, sleep, function, or behavior associated with dementia. Exercise programs for people with dementia were found to have a positive effect on functional ability, physical functioning, and mood in the studies [64]. Regarding depression, recent studies support the effectiveness of home-based exercise programs for people with dementia and their caregivers to reduce depressive symptoms [65]. Additionally psychological care can be appropriate management for depression and dementia. Recently animal-assisted activities were suggested to be associated with a decrease in anxiety and sadness and an increase in positive emotions and motor activity [66].

Pharmacological treatment

More recently, there have been updates regarding the off-label use of atypical antipsychotics for elderly patients with dementia. It was concluded that there were small but statistically significant benefits using aripiprazole, olanzapine, and risperidone in the treatment of BPSD. Adverse events were common, and included death, stroke, and extrapyramidal and urinary symptoms. Although the use of antipsychotics in BPSD is off-label, antipsychotics are still the best pharmacological short-term treatment option for severe persistent symptoms of dementia-related aggression/agitation.

The advice in the guidelines [67] is to minimize antipsychotic use in older people with dementia, to initiate antipsychotics only in patients with severe distress after a risk-benefit analysis, and to limit the dose and treatment duration, with attempts at discontinuation (6–12 weeks).Titrating dosages might decrease the possible adverse events [68]. The effectiveness and tolerability of treatment have to be verified at least every 2 months [68].

We have to also take into account the drug-drug interactions involving antipsychotics, as well as the interactions with food and concomitant diseases. In fact, the interactions may potentially lead to the increase in antipsychotic plasma levels, thus possibly increasing adverse effects [58]. Grapefruit juice (at least 250 mL/d) can inhibit CYP3A4, the enzyme involved in quetiapine, clozapine, and ziprasidone metabolism. Alcohol potentiates both antipsychotic-induced sedation and hypotension. Antipsychotic drugs may increase the sedative effects of benzodiazepines,

hypnotics, anesthetics, and antihistaminic agents. A number of diseases might interfere with antipsychotics, for example, congestive heart failure, liver and kidney diseases, fever, anemia, change in antipsychotic pharmacokinetics, leading to the possible increase in their plasma levels and adverse effects [68,69].

Antidepressants can be an effective and well-tolerated alternative to antipsychotics in vulnerable elderly individuals for treatment of BPSD [70]. This class of drugs has been used primarily for depression, with efficacy especially for the selective serotonin reuptake inhibitors (SSRIs) [71].

Donepezil, galantamine, or rivastigmine have all shown a modest effect on the broad spectrum of neuropsychiatric symptoms in AD [72]. They should be initiated prior to the use of other psychotropic agents since ChEIs reduce behavioral changes and improve or delay cognitive and functional decline [71]. The behavioral symptoms most likely to improve with ChEIs treatment appear to be apathy, depression, and aberrant motor behavior [73]. Memantine, and NMDA receptor antagonist, can also have beneficial effects on behavior, as well as on cognition and function. The use of memantine appears to improve specific behaviors, such as agitation and irritability, which differ from those affected by ChEIs (mood symptoms, apathy, and aberrant motor behavior) [67]. Combination therapy may have advantages in patients with multiple BPSD [71]. Benzodiazepines may be used at short-term for acute agitation associated with anxiety [67].

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