GUIDELINES FOR THE PSYCHOLOGICAL AND BEHAVIORAL MANAGEMENT OF DEMENTIA SYMPTOMS

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Abstract

Most dementia patients have BPSD. Dementia affects more than cognition and function. BPSD comprises emotional, psychotic, and behavioral symptoms.

Environment, psychology, and biology cause BPSD. Several variables typically cause BPSD. So, the patient, family, and care team must undergo an etiopathogenetic evaluation. Therapeutic decision trees altered by individual and environmental risk profiles provide personalized treatment programs. Therapy is challenging. Clinical empiricism bridges controlled research gaps.

BPSD therapy requires psychosocial methods. Drugs frequently follow non-pharmacological techniques. Reviewing the treatment plan and prescriptions periodically may detect relapse and remove inappropriate drugs. BPSD may persist despite proper treatment. Swiss interprofessional BPSD treatment guidelines inform this narrative review. Recommendations came from a thorough literature study. Medline, Embase, ISI, and Cochrane-Database yielded results. Evidence categories were WFBS. Swiss clinicians were assessed.

Keywords: *attachment, environmental factors, etiopathogenetic, individualized treatment, personality*

Introduction

Behavioural and psychological symptoms of dementia include a variety of symptoms and indicators of altered perception, mental content, mood, or conduct. Most dementia sufferers acquire BPSD. BPSD is serious. They induce dementia. BPSD sufferers and caretakers. They also increase the risk of falls and fractures that need emergency department visits and hospitalization. Finally, BPSD increases treatment and care costs. BPSD sufferers might be challenging to treat. Several mediators produce BPSD. Psychological (life history, personality), social (support network, housing arrangements), and biological (brain changes, comorbidities, medication) factors may interact. Etiopathogenetic analysis should guide treatment. Symptomatic treatments are only moderately effective and have minimal proof. Psychosocial, non-pharmacological therapy should be the cornerstone, with mental drugs used only when required. Using treatment algorithms, clinical experience, and expert knowledge, a personalized therapeutic plan may be created.

Clinical presentation of BPSD

BPSD causes apathy, depression, anxiety, delusions, hallucinations, sexual or social disinhibition, sleep–wake cycle abnormalities, aggressiveness, agitation, and other inappropriate behaviors. 14 BPSD should be assessed using the Neuropsychiatric Inventory (NPI) and BEHAVE-AD. BPSD clusters include affective, psychotic, hyperactive, and apathetic. In a population-based study, NPI-measured BPSD was predicted throughout dementia with an 80% cumulative incidence from cognitive symptoms. Most BPSD patients had apathy, unhappiness, anxiety, and agitation. A recent comprehensive study demonstrated wide variation in prevalence, incidence, and longitudinal course. Wandering is a persistent BPSD symptom. BPSD's "natural course" is uncertain.

Depression

80% of dementia patients over 5 years had depressive symptoms. 10%–20% experienced severe depression. Depression raises the risk of dementia-related severe depression. Depression causes sleeplessness, circadian rhythm problems, and anxiety. Serotonin, dopamine, epi-nephrine, frontal atrophy, and amygdala activation may produce depression.

Hallucinations

Eye disease-related Charles Bonnet syndrome should also be evaluated. Visual hallucinations in Charles Bonnet syndrome are short-lived and well-tolerated by patients. They may not need treatment other than that for the underlying eye illness. Carbamazepine may help. Auditory hallucinations are psychotic and need therapy.

Agitation

Agitation is the hardest BPSD because it might interfere with inpatient treatment. BPSD agitation treatment trials prevail. Agitation is an undefined spectrum of aberrant hyperactive motor actions including wandering and leaving home and physically or verbally hostile behaviors like refusing treatment. A broad consensus definition for cognitive disorder agitation was recently suggested. This new descriptor emphasizes agitation's emotional distress and inability beyond behavior. Sundowning may agitate.

Delusions

Delusions in dementia may not constitute psychotic symptoms and should not prevent attempts to comprehend them. They may sometimes match reality or be neither incorrigible nor absolute.

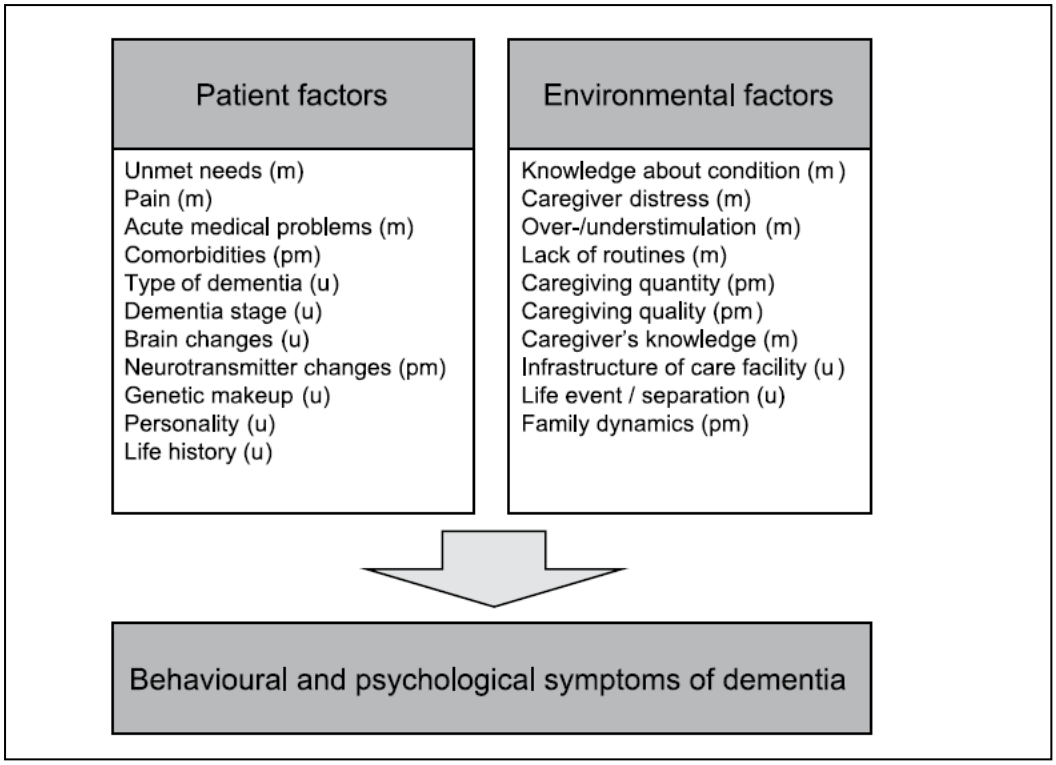

Figure 1 Simplified etiopathogenetic model of BPSD. m, modifiable; pm, potentially modifiable; u, modifiable.

Theft, lost items, danger, abandonment, and the idea that one's house is not home are common dementia delusions. Only half of illusions create behavioral problems. 28 Misidentification syndromes, when dementia patients regularly misidentify individuals,

places, objects, or events, are prevalent delusions. Sensory deficiency typically causes dementia delusions like Charles Bonnet syndrome in visual impairment.

Apathy

Loss of enthusiasm and interest in regular tasks is apathy. 30 In extreme cases, patients may spend most of the day in bed or a chair, unable to begin any purposeful activity. BPSD's most common manifestation, apathy, has a dismal prognosis and high mortality. Apathy seldom requires hospitalization since it is less disruptive to caregivers than other BPSD.

Sleep problems and disturbances of circadian rhythms

Comorbidities or dementias may induce sleep disturbances. Sleeplessness plagues caregivers. While effective medicines are needed, treatment evidence is sparse. REM sleep behavior disorder (RBD) with daily attention fluctuations, greater daytime naps, and longer night sleeps may indicate dementia with Lewy bodies (DLB). Parkinson's and multiple system atrophy have RBD. RBD is rare in frontotemporal dementia (FTD), however circadian rhythmicity disorders may cause evening activity.

Etiopathogenesis of BPSD

BPSD usually has a complicated etiopathogenesis. For didactic simplicity and practicability, biological, psychological, and social/environmental elements might be classified into causal and contributory components (Figure 1). This provides a paradigm for BPSD etiopatho-genetic therapy.

Biological perspective

Dementia, brain lesions. BPSD causes dementia-related brain lesions and neurotransmission abnormalities. Comorbidity, therapy, and genetics may lessen its impact. Psychotic symptoms resulted from hippocampus, parahippocampal, and brain stem nuclei cell loss in Alzheimer's disease (AD). Vascular factors may induce mixed AD-vascular dementia. FTD involves behavior and aphasia (bv-FTD or behavioural variant of FTD). Disinhibiting including sexual misbehavior and impulsivity—starts early. Sporadic and familial FTD. AD-like neurotransmitter and corticolimbic alterations induce FTD behavior issues. Early dementia needs FTD differential diagnosis. BPSD in FTD may be harder to treat than in other dementias. Neuroanatomical studies correlate BPSD to dementia-related brain abnormalities. Apathy was linked to anterior cingulate cortex and fronto-subcortical hypoperfusion, demonstrating a dissociation between the prefrontal cortex and the mediodorsal and front thalamic nuclei. Frontal or temporal lobe hypoperfusion induced aggressiveness and psychosis. White matter alterations, atrophy, and vascular damage cause BPSD. Neurotransmission/neuromodulation changes. BPSD impairs neurotransmission. AD frontal and temporal cortices cholinergic alterations may cause abnormal motor activity and aggression. Cholinergic temporal cortex deficiencies may cause DLB visual hallucinations. Temporal brain dopamine deficiency induced AD aggressiveness. Aggression follows LOC norepinephrine neuron loss. Dopaminergic deficiency induced AD apathy. Serotonin increased anger, melancholy, anxiety, agitation, and restlessness.

Glutamatergic pathways control synaptic plasticity, neuron survival, and hippocampus neurogenesis. AD's glutamate depletion may produce psychosis. In severe AD, low frontal and temporal GABA and high plasma GABA induced depression and apathy. Neurotransmitter-affecting medicines may induce or ameliorate BPSD. BPSD affects the hypothalamic–pituitary–adrenal axis, homocystein metabolism, circadian rhythms, and sleep.

BPSD research imply hereditary vulnerability. ApoE4 carriers were more delusional and violent. Neurotransmitter polymorphisms may induce BPSD.

Health concerns. BPSD treatment needs physical issues. Pain, infections, electrolyte imbalances, metabolic disorders, urine retention, constipation, and cerumen induce somatic BPSD. A comprehensive medical evaluation is necessary since any of these might cause BPSD. Pain causes BPSD. Dementia patients must identify and treat pain.

Psychological and environmental perspective

Stress and susceptibility cause BPSD, therefore several psychological and systemic causes may apply (personality, environmental ele- ments both physical and emotional that contribute to the occurrence of BPSD). Character qualities. Dementia changes personality. AD raises neuroticism, lowers extraversion, openness, and conscientiousness, and preserves agreeability. Personality affects dementia clinically, despite appearances. Personality affects BPSD. Premorbid neuroticism may cause depression, cognitive deterioration, and AD. Aggressive dementia patients had higher BPSD. Correlations are rare. Most research involves retrospective personality exams, which might bias results. Depression and BPSD risk dementia.

Life events. Stress, hippocampus hypotrophy, and insecure relationship may raise dementia BPSD risk. Dementia caused senior nursing home patients to develop attachment behaviors. Avoidant dementia patients were unhappier. Environmental dangers. Environments may impact BPSD. Hence, unobtrusive safety features, variety of places in tranquil surroundings, single rooms, modest facility size, and stimulation levels optimized for patient capabilities are linked to nursing home residents' well-being and decreased BPSD. Conflict and caregiver stress might worsen BPSD.

Treatment

The treatment of BPSD is often highly challeng- ing due to the complex etiopathogenesis of the symptoms and signs and the multi-morbidity of patients. BPSD management requires both a patient-centred and caregiver-centred focus and interventions to provide comfort to patients and alleviate caregiver burden are indispensable. Treating concomitant somatic diseases can reduce BPSD.6 Effective pain management is part of a successful BPSD treatment.81 Most expert recommendations and guidelines prefer non-pharmacological interventions as the first- line approach.

Although the evidence for most non-pharmacological strategies is weak, their efficacy is supported by long-standing clini- cal experience. Pharmacotherapy for BPSD is frequently provided, but it carries the risk of serious side-effects. Therefore, non-pharmaco- logical therapies are considered the first choice and should also be continued when pharmaco-therapy is necessary. In order to measure treat- ment effects, frequency and severity of BPSD should be quantified at baseline, possibly using a validated scale or questionnaire, such as the NPI16 or BEHAVE-AD. Moreover, several algorithms have been published to guide the diagnostic and therapeutic process for BPSD. We suggest using the simplified BPSD-DATE algorithm (describe and measure, analyse, treat, evaluate; see Figure 2).

Non-pharmacological approaches

Study designs for non-pharmacological therapies differ greatly, making their findings difficult to generalize. BPSD pharmacotherapy has little proof.

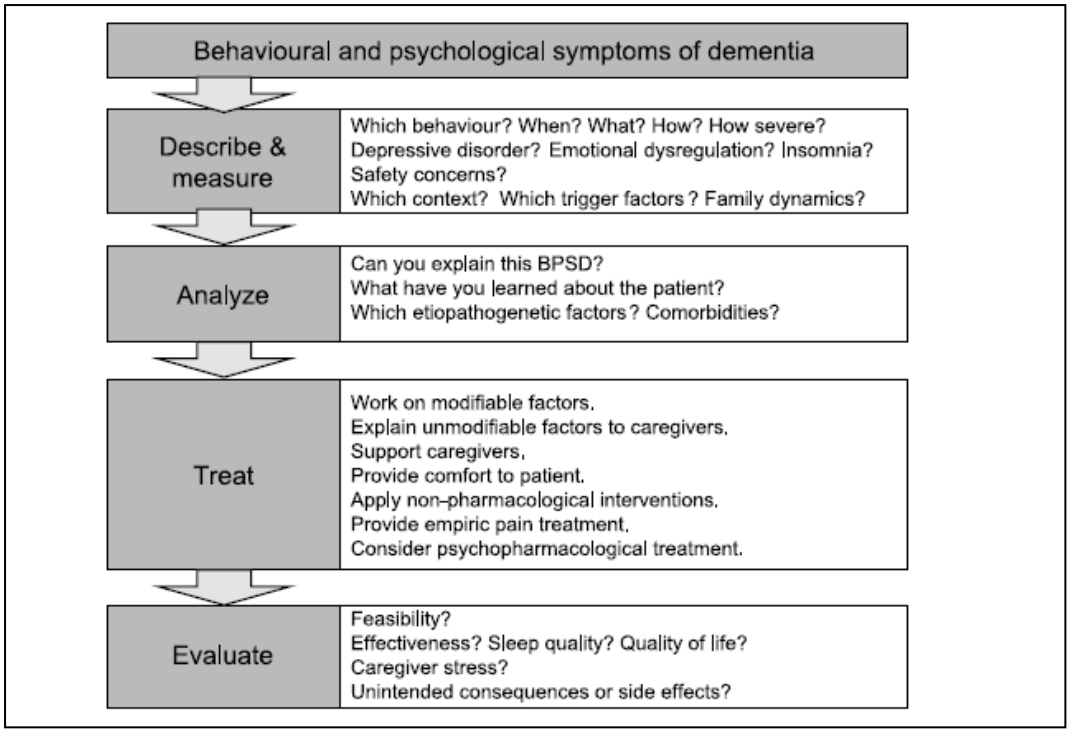

Figure 2 BPSD-DATE interventional algorithm.

if a common active but non-specific element like good human connection makes various non-pharmacological therapies effective. Home-based behavioral management strategies, caregiver-based therapies or staff training in communication skills, person-centered care, dementia care mapping against agitation and music therapy against agitation and anxiety have the best scientific evidence.

Psychosocial interventions

Patient and caregiver psychoeducation reduces BPSD. Multi-component single and group session programs that focus on stressful events, provide disease information and assistance, and allow for sharing of daily problem-solving experiences are effective. Caregivers find behavioural management training more burdensome. Coping strategy-based family carer therapy and tailored activities for dementia patients and caregivers improved quality of life at home. 88Social counseling may organize help and support patients and carers throughout psychoeducational treatments. Psychosocial therapies reduce caregiver sadness and postpone institutionalization.

Nursing care

The need-driven dementia-compromised behavior model (NDB) may explain BPSD as a disordered need expression. This paradigm allows nurse behavioural analysis to identify patients' urgent requirements and their causes. Pain, hunger, and thirst may be quickly addressed to treat BPSD. Nonetheless, the patient's personality, biography, comorbidities, and lack of resources might worsen the sickness. The NDB-based Serial Trial Intervention employs systematic serial assessments and sequential treatment trials to identify and solve unmet requirements that may cause BPSD. It lowers BPSD and psychotropic medication usage. Nurses may soothe patients with vocalization and sexual disinhibition interventions.

Physical activity

Older folks benefit from regular exercise. In dementia patients, frequent physical exercise improves physical, cognitive, functional, and behavioral outcomes and reduces BPSD. Training programs usually include walking or isotonic activities.

Sensory stimulation and music therapy

Music therapy and multisensorial stimulation like snoezelen reduce agitation and disruptive behavior during and after sessions. Long-term consequences are unproven. Biographical music and sensory stimulation work better.

Reality orientation and cognitive stimulation therapy

These therapies enhance BPSD by improving everyday orientation to people, time, and environment. Reality orientation treatment improves mood and reduces BPSD better with other methods. Cognitive stimulation treatment tackles present functional issues utilizing information processing. BPSD is affected immediately, however data are inconsistent.

Validation therapy

This patient-centered technique encourages and validates emotions to heal unresolved issues. Positively affirming sentiments may decrease irritation.

Reminiscence therapy

Reminiscence therapy employs commonplace items to jog memory and help individuals appreciate their experiences. This treatment may lift your spirits.

Psychotherapeutic interventions

Psychological therapy for mild-to-moderate dementia have been studied. Cognitive-behavioural techniques have the most effectiveness proof. Caregiver involvement improves psychotherapeutic therapies that address everyday issues. Psychoeducation and family counseling boost efficacy. Behavioral management reduces dementia-related despair, anxiety, aggressiveness, and agitation. That lasts months. Individual counseling may help caregivers who suffer depressed.

Psychopharmacotherapy

Psychopharmacotherapy in BPSD must be carefully considered since dementia patients are more susceptible to pharmacological side effects. Polypharmacy and multi-morbidity complicate medication. Most medicines are off-label for BPSD. Psychopharmacotherapy should follow a thorough clinical and laboratory check, including a drug history and ECG. As BPSD improves, psychotropic drug usage should be reduced. Elderly people require lower psychiatric medication dosages since their metabolism is changed.

Antidementia drugs

Cholinesterase inhibitors and memantine may help treat BPSD. Cholinesterase inhibitors may enhance emotional features in mild-to-moderate dementia. Memantine and cholinesterase inhibitors may treat BPSD. In mild to severe dementia, donepezil may reduce apathy, sadness, tension, and irritability. Rivastigmine and galantamine have comparable results. Yet, donepezil seems ineffective in treating AD agitation. Cholines-terase inhibitors reduce unpleasant symptoms. In moderate to severe AD, memantine may improve agitation, delusions, hallucinations, and aggressiveness. Recent agitation experiments failed to show a benefit, challenging these conclusions. Lastly, antidepressants may lessen BPSD. Ginkgo biloba extract EGb 761® may also prevent dementia in clinically relevant BPSD patients.

Antidepressants

Antidepressants may enhance cognition, affective symptoms, and other BPSD symptoms

including agitation and aggression. Depression and anxiety are the most frequent BPSD. Anticholinergic side effects disqualify tricyclic antidepressants. SSRIs are well-tolerated and effective. Citalopram, an SSRI, treats dementia agitation as well as atypical antipsychotics. QT-prolongation and hyponatraemia are serious side effects of SSRIs.

Antipsychotics

Initially, only risperidone is authorized for dementia treatment in certain countries. Before prescribing an antipsychotic for BPSD, doctors should check their country's laws. BPSD patients are administered atypical antipsychotics such risperidone and aripiprazole too frequently. They treat psychotic symptoms, aggressiveness, and agitation well. Haloperidol may be used to treat dementia delirium, but not for other purposes. Haloperidol's adverse effects limit its usage to delirium. Atypical antipsychotics cause anticholinergic effects, orthostatic hypotension, seizures, meta-bolic syndrome, weight gain, extrapyramidal symptoms, drowsiness, and QT-prolongation. Antipsychotics in dementia carry a black box warning due to increased mortality and cerebrovascular events. Antipsychotics may help treat certain BPSD, but they should be used sparingly. Risk-benefit assessments are needed during therapy. Quetiapine is extensively used for BPSD despite contradictory data. Despite mixed data, quetiapine may be useful for BPSD, especially in Parkinsonian patients, due to its favorable side-effect profile, notably extrapyramidal symptoms.

Mood stabilizers

Carbamazepine helps dementia agitation, but mood stabilizers have serious adverse effects. Hence, avoid valproic acid. Gabapentine and lamotrigine have minimal clinical experience treating BPSD.

Benzodiazepines BPSD benzodiazepine effectiveness is unproven. In the elderly, benzos cause sleepiness, dizziness, falls, poor cognition, respiratory depression, dependence, and paradoxical disinhibition. They are only indicated for acute crisis management if other methods fail. They should not be recommended as hypnotics and used sparingly.

Other substances Zopiclone, zolpidem, and zaleplone have adverse effects like benzodiazepines. They treat dementia sleep disturbances for a short duration at low dosages. Trazodone may prolong sleep. Melatonin and receptor agonists may cure circadian sleep disorders.

Biological therapies

Light treatment in the morning and light therapy with melatonin at night may help treat sleep or circadian rhythm disorders, "sundowning," and daytime drowsiness, however sleep deprivation is not indicated in BPSD. Insomnia may cause BPSD and irritability.

Individuals may benefit from electroconvulsive treatment. Repeated transcranial magnetic stimulation may help cure BPSD, although research is yet underway.

Conclusion

When dementia progresses, BPSD nearly always occur. BPSD symptoms and indicators vary, but all may cause severe suffering in patients and carers. Even in one patient, BPSD has various biological, psychological, and social/environmental causes and susceptibility factors. A thorough history and clinical assessment with the patient and family or care team are needed. A therapeutic decision tree should include the patient's individual and environmental risk profile to create an individualized treatment strategy. Psychosocial therapies matter. Combining non-pharmacological methods precedes medicinal therapy. Hence, BPSD patients

need an interventional strategy (Figure 2). To identify relapse and stop unsuitable drugs, the treatment plan and prescriptions must be reviewed regularly. Even with effective management, BPSD may persist and challenge all parties.

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