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Subjective Memory Complaints in Everyday Clinical Practice: A Practical Guide to Evaluation and Treatment

Jozelio Freire de Carvalho
Institute of Health Sciences, Federal University of Bahia, Salvador, Bahia, Brazil

* Corresponding Author: **Jozelio Freire de Carvalho**

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Abstract

Background: Subjective memory complaints are common in outpatient practice, including among individuals without demonstrable cognitive impairment or established neurological disease. These complaints may reflect potentially modifiable conditions, including sleep disturbances, psychological distress, chronic pain, medication effects, and systemic disorders, but may occasionally precede objective cognitive decline. Their evaluation is challenging, particularly when patients request pharmacological or nutraceutical treatment despite reassuring cognitive findings.

Objective: To provide a practical, evidence-informed approach to the evaluation and management of subjective memory complaints, emphasizing clinical assessment, laboratory investigation, non-pharmacological management, and interventions with reported favorable cognitive outcomes.

Methods: This clinically oriented narrative review integrates diagnostic frameworks, clinical practice guidelines, randomized controlled trials, and systematic reviews relevant to subjective cognitive complaints and cognitive enhancement. Findings from individuals with subjective cognitive decline are distinguished from those obtained in cognitively healthy adults, patients with mild cognitive impairment, and individuals with dementia. Favorable therapeutic findings are discussed alongside negative evidence and methodological limitations.

Results: Evaluation should begin with characterization of the complaint, assessment of functional independence, medication review, identification of sleep and psychological disturbances, and appropriate cognitive screening. Laboratory investigations should be individualized according to clinical findings. Non-pharmacological management remains central to treatment. Positive cognitive outcomes have been reported for citicoline, phosphatidylserine-containing formulations, *Bacopa monnieri*, and creatine, although consistency, clinical significance, and applicability to isolated subjective cognitive decline remain uncertain. Evidence for *Ginkgo biloba* is particularly uncertain in individuals with subjective cognitive complaints.

Conclusions: Subjective memory complaints require a structured but proportionate clinical approach. Identifying potentially reversible contributors, recognizing warning signs of objective cognitive impairment, and providing individualized follow-up remain the principal priorities. Although selected pharmacological and nutraceutical interventions have demonstrated favorable results, current evidence does not establish a routine treatment for isolated subjective cognitive decline.

Keywords: Subjective cognitive decline, Memory complaints, Cognitive assessment, Primary care, Cognitive enhancement, Nutraceuticals, Citicoline

1. Introduction

Memory complaints are frequently encountered in everyday medical practice and represent an important source of concern for patients and their families. Individuals commonly report forgetting names, misplacing personal belongings, difficulty retrieving familiar words, or an inability to remember recent conversations. These symptoms may generate considerable anxiety, particularly when patients associate forgetfulness with the possibility of Alzheimer's disease or another neurodegenerative disorder. Nevertheless, subjective memory difficulties frequently occur in individuals who maintain normal occupational

performance, social functioning, and independence in daily activities, highlighting the importance of distinguishing perceived cognitive decline from objectively measurable impairment [1].

Subjective cognitive decline (SCD) has been defined as a self-experienced persistent decline in cognitive capacity compared with a previously normal level of functioning, despite performance within the expected range on standardized cognitive assessments. Although memory is the most frequently reported domain, subjective difficulties may also involve attention, language, executive function, and processing speed. Importantly, SCD does not represent a single disease entity and should not automatically be interpreted as an early manifestation of Alzheimer's disease. Its underlying mechanisms are heterogeneous, and its clinical significance depends on symptom characteristics, associated risk factors, and the presence of additional clinical findings [1].

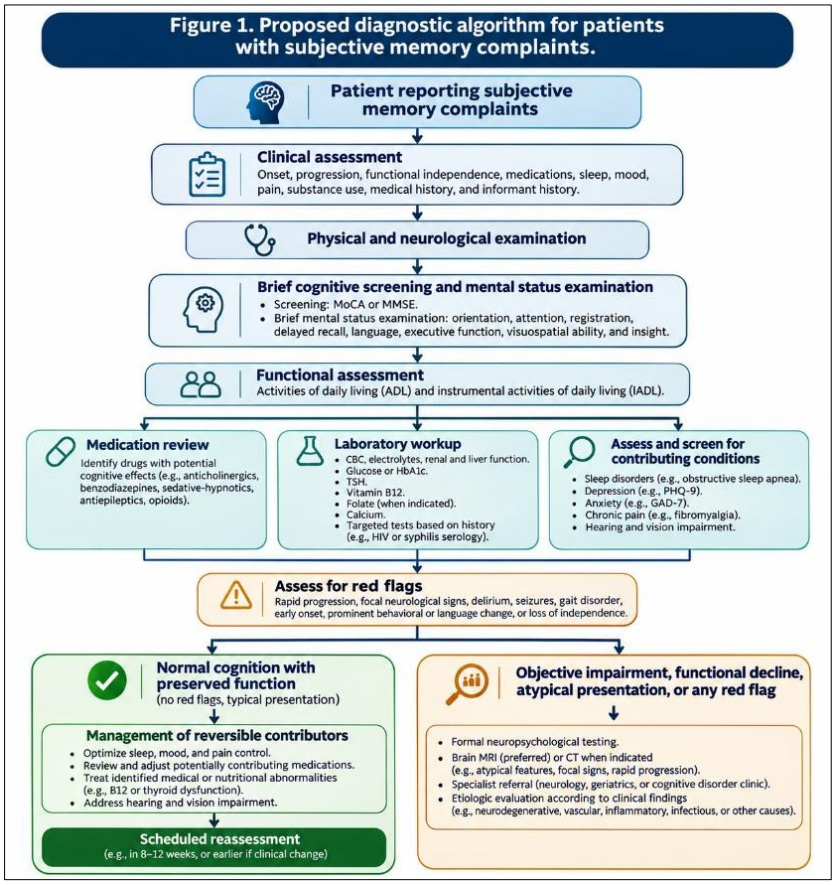

Fig 1

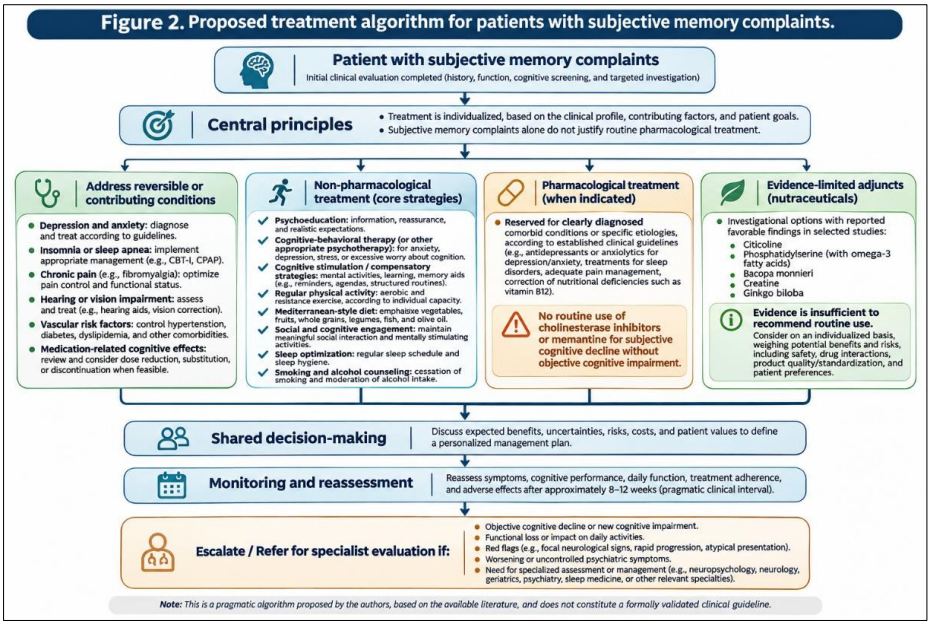

Fig 2

The relationship between subjective cognitive complaints and objective cognitive performance is complex. Patients may describe substantial difficulties in everyday life despite normal neuropsychological findings, while others may demonstrate measurable cognitive abnormalities without expressing significant concern. This discrepancy emphasizes that subjective complaints and standardized cognitive tests assess partially different aspects of cognitive functioning. Consequently, normal screening results should not invalidate the patient's experience, just as persistent forgetfulness should not automatically establish a neurological diagnosis [1, 2].

Several potentially modifiable factors may contribute to subjective memory complaints. Insufficient sleep, anxiety, depression, chronic psychological stress, fatigue, persistent pain, and medication effects may interfere with attention and information processing. Endocrine, metabolic, and nutritional abnormalities may also contribute in selected patients. Conversely, longitudinal studies indicate that some individuals with SCD subsequently develop objective cognitive impairment, particularly when symptoms are progressive or accompanied by functional changes, informant-confirmed decline, or additional risk characteristics [3].

Recent clinical recommendations emphasize the importance of integrating symptom history, functional assessment, cognitive examination, and individualized investigations. The Alzheimer's Association DETeCD-ADRD guideline provides a structured approach to patients with suspected cognitive impairment, while the World Health Organization and the Lancet Commission emphasize the importance of addressing modifiable risk factors and maintaining cognitive health [4–6]. However, the intensity of investigation should be proportionate to the clinical presentation, since individuals with isolated subjective complaints and preserved functioning differ substantially from patients with established cognitive syndromes.

An additional challenge concerns the increasing availability of medications and nutraceutical products marketed for cognitive enhancement. Patients frequently request treatment even when no specific neurological diagnosis has been established. Although several compounds have demonstrated favorable outcomes in individual clinical trials, the supporting evidence is often limited by heterogeneous populations, small sample sizes, short follow-up periods, or improvements restricted to selected neuropsychological outcomes. The present review therefore aims to provide a practical approach to the evaluation and management of subjective memory complaints, with particular attention to identifying reversible contributors and critically examining interventions that have demonstrated positive clinical findings.

2. Clinical Characterization of Subjective Memory Complaints

2.1. Distinguishing Memory Dysfunction from Attentional Difficulties

The first challenge in evaluating a patient reporting memory loss is determining which cognitive process is actually affected. Memory depends on several interconnected mechanisms, including attention, encoding, consolidation, storage, and retrieval of information. A disturbance at any of these stages may be perceived as forgetfulness, although the underlying problem may not represent a primary disorder of memory storage. This distinction has practical implications because attentional difficulties are particularly common in patients experiencing stress, fatigue, insufficient sleep, or chronic pain.

For example, a patient who cannot recall information from a recent conversation may have failed to adequately register the information because attention was divided when it was presented. Similarly, difficulty remembering names or retrieving familiar words may occur in otherwise cognitively healthy individuals, especially during periods of increased mental demand. In contrast, repeated inability to retain recently learned information despite adequate attention, particularly when accompanied by functional changes, deserves more detailed investigation. The clinical interview should therefore explore not only what the patient forgets but also the circumstances in which the difficulty occurs and whether the information can subsequently be recalled with contextual cues [1, 4].

The temporal characteristics of symptoms are equally important. Complaints that fluctuate according to stress, sleep quality, or fatigue may suggest potentially modifiable contributors, whereas progressive difficulties affecting multiple cognitive domains raise greater concern for an evolving cognitive disorder. Nevertheless, no isolated symptom pattern is sufficiently specific to establish a diagnosis, and interpretation should always consider the patient's age, educational background, previous cognitive abilities, and clinical context.

2.2. Functional Assessment and Progression

Assessment of functional independence is one of the most useful components of the initial consultation because it helps distinguish isolated subjective complaints from cognitive changes with broader clinical consequences. Patients should be asked whether they continue to manage finances, medications, transportation, appointments, professional responsibilities, and household activities without assistance. Particular attention should be given to newly developed difficulties in activities that were previously performed independently, rather than longstanding limitations unrelated to cognition.

Information from a family member or another reliable informant may provide additional insight, especially when the patient and relatives disagree regarding the severity or progression of symptoms. A patient who reports occasional forgetfulness but remains fully independent and performs normally on cognitive screening may require a different approach from an individual whose relatives describe progressive repetition of questions, difficulty organizing daily activities, or declining occupational performance. Functional deterioration should not automatically be equated with dementia, but it warrants a more comprehensive assessment [3, 4].

2.3. Psychological Factors and Emotional Distress

Psychological distress represents an important contributor to subjective memory complaints and should be evaluated routinely. Anxiety, depressive symptoms, chronic stress, and emotional exhaustion may impair concentration, reduce cognitive efficiency, and increase awareness of minor memory failures. Patients may consequently interpret ordinary lapses in attention or retrieval as evidence of significant cognitive deterioration, particularly when they are concerned about developing dementia.

The relationship between psychological symptoms and perceived cognitive dysfunction is often bidirectional. Emotional distress may increase cognitive complaints, while persistent concern about memory may further aggravate anxiety and attentional difficulties. Instruments such as the Patient Health Questionnaire-9 and Generalized Anxiety Disorder-7 can support clinical assessment, although their results should complement rather than replace a detailed interview. Importantly, the presence of anxiety or depression does not exclude a coexisting cognitive disorder, and progressive symptoms should not be dismissed solely on psychological grounds [1, 3].

2.4. Sleep Disturbances

Sleep quality is closely related to attention, learning, and memory consolidation, making sleep assessment an essential component of the clinical evaluation. Insomnia, insufficient sleep duration, irregular sleep schedules, and obstructive sleep apnea may contribute to daytime cognitive difficulties that patients describe as memory loss. These symptoms may become particularly noticeable during tasks requiring sustained attention, rapid information processing, or simultaneous management of several activities.

A practical sleep history should include usual sleep duration, difficulty initiating or maintaining sleep, excessive daytime sleepiness, snoring, witnessed breathing interruptions, and use of medications that affect sleep architecture or daytime alertness. When obstructive sleep apnea is suspected, further evaluation should follow established clinical recommendations. Addressing a clinically significant sleep disorder may improve daytime functioning and perceived cognitive performance, although improvement should not be assumed to occur uniformly in every patient [5, 6].

2.5. Chronic Pain and Fibromyalgia

Chronic pain deserves particular attention because cognitive complaints frequently accompany conditions characterized by persistent pain, fatigue, and sleep disturbances. Fibromyalgia provides a clinically relevant example, as patients commonly report difficulties with concentration, working memory, word retrieval, and mental processing speed. These symptoms, often described as *fibro fog*, may be sufficiently distressing to become a major reason for consultation, even when conventional neurological examinations are unremarkable.

A systematic review and meta-analysis by Nicholls and Kelly identified poorer verbal working memory performance among individuals with fibromyalgia compared with healthy controls, with a moderate effect size (Hedges' g = −0.66; 95% CI −0.94 to −0.38). In contrast, the pooled analysis did not demonstrate a statistically significant overall difference in visual working memory. These findings suggest that cognitive abnormalities in fibromyalgia may be domain-specific rather than reflecting uniform impairment across all memory processes [7].

The mechanisms underlying these complaints are likely multifactorial. Persistent pain may compete for attentional resources, while nonrestorative sleep, fatigue, emotional distress, and centrally acting medications may further reduce cognitive efficiency. In this context, the clinician should evaluate cognitive symptoms as part of the broader clinical syndrome rather than assuming the presence of an independent neurological disorder. Management should prioritize optimization of pain control, sleep, physical functioning, and psychological well-being, while recognizing that direct evidence supporting specific memory-enhancing medications in fibromyalgia remains limited.

2.6. Medication-Associated Cognitive Symptoms

A systematic review of current medications is indispensable because several commonly prescribed agents may contribute to cognitive symptoms through sedation, impaired attention, anticholinergic activity, or other central nervous system effects. Benzodiazepines, sedative-hypnotics, medications with substantial anticholinergic properties, selected antihistamines, certain antiepileptic drugs, and other centrally acting treatments deserve particular consideration. The possibility of additive effects is especially relevant in patients receiving multiple medications.

Clinical assessment should establish whether cognitive symptoms began or worsened after treatment initiation, dose escalation, or the introduction of additional medications. When a potentially contributing drug is identified, the clinician should evaluate the indication, expected benefit, available alternatives, and risks associated with modification. Supervised dose reduction or substitution may be appropriate in selected circumstances, but abrupt discontinuation should be avoided when withdrawal or recurrence of the underlying condition may occur [4, 6].

3. Practical Diagnostic Evaluation

3.1. Cognitive Screening

Brief cognitive screening provides an objective reference point and may help identify patients requiring more extensive investigation. Instruments such as the Montreal Cognitive Assessment (MoCA) and Mini-Mental State Examination can be used according to the clinical setting, language, educational background, and availability of validated versions. However, their diagnostic performance varies across populations, and results should not be interpreted independently of the clinical history or functional assessment.

In patients with isolated subjective complaints, normal screening performance may provide reassurance, particularly when functional independence is preserved and no concerning neurological findings are present. Nevertheless, brief instruments may fail to detect subtle cognitive abnormalities, especially in individuals with high premorbid cognitive abilities. Conversely, low educational attainment, language differences, sensory impairment, and emotional distress may influence test performance. Persistent or progressive complaints, especially when supported by an informant, may justify formal neuropsychological assessment even when initial screening is within the expected range [4].

3.2. Physical and Neurological Examination

The physical examination should be directed toward identifying systemic or neurological conditions that may contribute to cognitive symptoms. Blood pressure measurement, general medical assessment, and evaluation of hearing and vision are particularly relevant because vascular risk factors and sensory impairment may influence cognitive functioning. A focused neurological examination should assess cranial nerves, motor function, coordination, gait, and the presence of focal abnormalities or movement disorders.

In most patients with isolated subjective memory complaints, the neurological examination is expected to be normal.

Nevertheless, abnormalities such as focal weakness, gait disturbance, parkinsonian features, or other unexplained neurological signs should prompt reconsideration of the initial diagnostic hypothesis. The examination also provides an opportunity to identify clinical findings suggesting endocrine, nutritional, metabolic, or systemic disease [4, 6].

3.3. Laboratory Investigations

Laboratory testing should be guided by the clinical presentation rather than performed indiscriminately. The principal objective is to identify potentially reversible systemic abnormalities that may contribute to impaired attention, fatigue, or cognitive symptoms. A pragmatic initial assessment may include complete blood count, thyroid-stimulating hormone, vitamin B12, glucose and glycated hemoglobin, renal function, electrolytes, and calcium. Liver function tests may also be appropriate according to the patient's clinical characteristics and medical history.

Additional investigations should be individualized. Folate measurement may be relevant in patients with nutritional risk factors, whereas ferritin may be useful when iron deficiency is suspected. Vitamin D testing should be guided by established indications rather than requested solely because of subjective forgetfulness. Infectious disease testing, inflammatory markers, and other specialized investigations should be considered when supported by clinical findings, epidemiological factors, or relevant comorbidities.

A distinction should be maintained between laboratory evaluation for isolated subjective complaints and the broader diagnostic investigation recommended when objective cognitive impairment is suspected. The Alzheimer's Association guideline recommends a structured investigation in patients with suspected cognitive-behavioral syndromes, but this should not be interpreted as requiring every possible laboratory test in every patient with subjective forgetfulness. The absence of abnormalities on routine laboratory testing does not establish a neurological diagnosis, but it may help exclude selected contributing conditions and support a proportionate management strategy [4].

3.4. Neuroimaging and Specialist Referral

Routine neuroimaging is not necessarily indicated for every patient with isolated subjective memory complaints, preserved functional independence, normal neurological examination, and reassuring cognitive screening. In such cases, the diagnostic yield of imaging may be limited, and incidental findings may generate additional investigations without improving clinical management. Decisions regarding imaging should therefore consider the overall probability of structural or neurological disease.

Brain magnetic resonance imaging becomes more relevant when symptoms are progressive, objective cognitive impairment is demonstrated, focal neurological abnormalities are present, or the clinical presentation is atypical. Patients with rapidly evolving symptoms or acute neurological changes require more urgent assessment. Referral for neurological or neuropsychological evaluation should similarly be guided by clinical progression, functional consequences, examination findings, and uncertainty regarding the diagnosis [4].

4. Non-Pharmacological Management

Non-pharmacological interventions constitute the foundation of management because they address common contributors to cognitive complaints while offering benefits that extend beyond memory performance. Rather than treating subjective forgetfulness as an isolated symptom, clinicians should evaluate the patient's overall health, sleep quality, psychological well-being, physical activity, and daily routines. This approach is particularly appropriate when cognitive testing is reassuring and potentially modifiable factors have been identified.

Regular physical activity may improve cardiovascular health, mood, sleep, and selected aspects of cognitive performance. An umbrella review and meta-meta-analysis by Singh and colleagues reported improvements in general cognition, memory, and executive function associated with exercise across different populations. Although these findings support physical activity as part of comprehensive health management, improvements observed in heterogeneous study populations should not be interpreted as proof that exercise consistently resolves isolated subjective memory complaints [8].

Sleep optimization is equally important, particularly when symptoms fluctuate with fatigue or insufficient rest. Management should address insomnia, irregular sleep patterns, and suspected obstructive sleep apnea using interventions appropriate to the underlying condition. Similarly, psychological treatment may be beneficial when anxiety, depression, or persistent stress contributes to cognitive difficulties. In patients with chronic pain, especially fibromyalgia, improving pain-related functioning and sleep may be more clinically meaningful than prescribing an isolated cognitive-enhancing product.

Cognitive stimulation and structured cognitive training may improve performance in selected tasks, but transfer to everyday functioning is variable. Clinicians should distinguish improvement on trained exercises from broader enhancement of memory in daily life. Practical strategies such as consistent routines, organization of appointments, reduction of distractions, and use of external memory aids may nevertheless help patients manage symptoms and maintain confidence in everyday activities. These interventions should be integrated into a broader strategy that also addresses cardiovascular and metabolic health, hearing impairment, and other potentially modifiable contributors [5, 6].

5. Pharmacological and Nutraceutical Interventions

5.1. Interpretation of Therapeutic Evidence

The increasing use of products marketed for cognitive enhancement has created considerable interest in pharmacological and nutraceutical interventions for subjective memory complaints. However, the available literature presents several methodological challenges. Studies frequently include participants with age-associated memory impairment, cognitively healthy older adults, individuals with mild cognitive impairment, and patients with established neurological disorders. These populations differ substantially from patients presenting with isolated subjective cognitive decline, limiting the generalizability of therapeutic findings.

Another important limitation concerns the interpretation of cognitive outcomes. Improvements in individual neuropsychological tests may indicate a measurable effect on a specific cognitive domain without demonstrating meaningful improvement in everyday memory complaints. Furthermore, favorable findings derived from secondary outcomes, small samples, or exploratory analyses require

cautious interpretation. Accordingly, this review presents interventions with reported positive findings while also considering negative studies, conflicting evidence, formulation differences, and potential safety concerns.

5.2. Citicoline

Citicoline, or cytidine diphosphate-choline, is involved in phospholipid metabolism and has been investigated for potential effects on neuronal membrane function and cognitive performance. Interest in its use for memory complaints is supported by clinical studies involving older adults with age-associated cognitive symptoms. However, its proposed biological mechanisms do not independently establish therapeutic efficacy, and clinical outcomes remain the principal basis for evaluating its usefulness.

Nakazaki and colleagues conducted a randomized, double-blind, placebo-controlled trial involving 100 adults aged 50–85 years with age-associated memory impairment. Participants received citicoline 500 mg daily or placebo for 12 weeks. The study reported statistically favorable changes in episodic memory and a composite memory score, while comparable benefits were not demonstrated across all cognitive domains [9]. These results are relevant because they demonstrate a measurable cognitive signal in a population without established dementia.

Nevertheless, the interpretation of these findings requires consideration of their limitations. The study evaluated a selected population and used computerized cognitive tests, while the extent to which the observed improvements translate into clinically meaningful changes in daily functioning remains uncertain. Moreover, a subsequent scientific evaluation by the European Food Safety Authority considered the broader available evidence and concluded that a causal relationship between citicoline consumption and improvement or maintenance of memory had not been established [10]. Thus, citicoline represents a potentially interesting intervention with positive clinical findings, but the current evidence does not justify routine use in all patients with isolated subjective memory complaints.

5.3. Phosphatidylserine-Containing Formulations

Phosphatidylserine is a phospholipid component of neuronal membranes and has been investigated for potential effects on cognitive performance. Its proposed role in membrane integrity and neuronal signaling has encouraged studies of memory performance in older adults. However, clinical evidence must be interpreted according to the exact formulation used, since several investigations have evaluated phosphatidylserine in combination with other compounds rather than as an isolated intervention.

Vakhapova and colleagues conducted a randomized, placebo-controlled trial involving 157 older adults without dementia who reported memory complaints. The investigated formulation combined phosphatidylserine with omega-3 fatty acids, and treatment was administered for 15 weeks. The study reported favorable effects on immediate verbal recall, with additional findings emerging from selected analyses [11]. These results suggest that the combined formulation may influence specific memory outcomes in a population resembling patients commonly encountered in clinical practice.

However, the evidence remains insufficient to establish a general recommendation. The observed benefit cannot automatically be attributed to phosphatidylserine alone, and differences in formulation, dose, population, and cognitive endpoints may affect reproducibility. Larger independent trials assessing patient-reported memory improvement and functional outcomes are needed before routine clinical use can be supported.

5.4. *Bacopa monnieri*

Bacopa monnieri is a botanical preparation traditionally associated with cognitive enhancement and has been investigated in several randomized clinical trials. Interest in this intervention has focused particularly on memory acquisition, verbal learning, delayed recall, and attention. Although favorable findings have been reported, the available studies differ in participant characteristics, extract standardization, treatment duration, and outcome assessment, making direct comparisons difficult.

Morgan and Stevens conducted a randomized, double-blind, placebo-controlled trial involving 98 older adults and reported favorable findings in selected measures of memory performance [12]. These results contributed to interest in *Bacopa monnieri* as a potential intervention for age-associated cognitive complaints. Nevertheless, improvement in specific neuropsychological outcomes should not automatically be interpreted as clinically meaningful improvement in everyday memory functioning.

More recent evidence has introduced additional uncertainty. Lopresti and Smith evaluated a standardized *Bacopa monnieri* preparation in a randomized trial involving adults with self-reported memory and attention difficulties. The intervention did not demonstrate significant improvements in the principal cognitive outcomes, although selected secondary findings were favorable [13]. Accordingly, *Bacopa monnieri* represents an intervention with conflicting clinical evidence, and further adequately powered trials are needed before routine use can be recommended.

5.5. Creatine

Creatine plays an important role in cellular energy metabolism, including processes relevant to brain function. This biological rationale has prompted investigations of its potential effects on cognitive performance under different physiological conditions. Some clinical trials and pooled analyses have reported favorable results in selected memory outcomes, suggesting that creatine may influence particular aspects of cognitive functioning.

Xu and colleagues published a systematic review and meta-analysis evaluating randomized controlled trials of creatine supplementation and cognitive performance in adults. The analysis included 16 trials involving 492 participants and reported favorable findings in selected memory-related outcomes, although improvements were not consistent across all cognitive domains [14]. A corrigendum was subsequently published and should be considered when interpreting the quantitative findings [15].

Nevertheless, the studied populations were heterogeneous, and findings obtained in healthy individuals or under specific experimental conditions cannot be directly extrapolated to patients with persistent subjective memory complaints. In addition, the relationship between changes in cognitive test scores and meaningful improvements in daily functioning remains insufficiently established. Creatine should therefore be regarded as an investigational intervention in this clinical context rather than an established treatment.

5.6. *Ginkgo biloba*

Ginkgo biloba extracts have been investigated extensively for cognitive symptoms, particularly in older adults. Some studies have reported favorable effects on selected cognitive measures, while others have failed to demonstrate meaningful benefits. Interpretation is further complicated by differences in extract composition, dose, duration, and participant characteristics, as well as the inclusion of patients with dementia or other forms of cognitive impairment.

A Cochrane systematic review published in 2026 included 82 studies involving 10,613 participants across different cognitive conditions. However, only three studies involving 597 participants specifically addressed subjective cognitive complaints, and evidence regarding improvement in global clinical status remained very uncertain. In patients with mild cognitive impairment, the review found little or no benefit in several cognitive and functional outcomes, whereas selected improvements were reported in dementia populations [16].

For individuals presenting with isolated subjective memory complaints, the available evidence therefore remains insufficient to support routine use. Results from patients with established neurodegenerative disorders should not be generalized to cognitively unimpaired adults. The potential for adverse effects and clinically relevant interactions, particularly involving medications affecting hemostasis, should also be considered.

5.7. Omega-3 Fatty Acids

Omega-3 fatty acids have been investigated in relation to neuronal membrane composition, cardiovascular health, and cognitive performance. Several studies have explored whether supplementation may improve memory or reduce cognitive decline, with results varying according to age, nutritional status, baseline cognitive function, and the composition of the intervention.

Although selected investigations have reported favorable cognitive outcomes, consistent evidence supporting omega-3 supplementation specifically for isolated subjective memory complaints remains insufficient. Benefits observed in particular populations should not be assumed to apply universally. In clinical practice, omega-3 intake should be considered within the broader context of nutritional and cardiovascular health rather than prescribed indiscriminately as a specific treatment for subjective forgetfulness [5, 6].

5.8. Vitamins and Correction of Nutritional Deficiencies

Nutritional abnormalities may contribute to cognitive symptoms, particularly when deficiencies affect neurological function or general health. Vitamin B12 deficiency is clinically relevant because it may be associated with neurological manifestations, anemia, and cognitive difficulties. Folate deficiency and other nutritional disorders may also contribute to symptoms in selected patients, especially when dietary restrictions, malabsorption, or relevant medical conditions are present.

Correction of a documented deficiency is an appropriate therapeutic intervention, but this should be distinguished from supplementation in individuals with normal nutritional status. Evidence does not consistently demonstrate that routine administration of B vitamins, vitamin D, vitamin E, or multivitamin preparations improves isolated subjective memory complaints in the absence of deficiency. Therefore, supplementation should be guided by clinical indications and laboratory findings rather than by the presence of forgetfulness alone [4, 5].

5.9. Other Pharmacological Agents

Several pharmacological compounds have been proposed for cognitive enhancement, including piracetam and other agents commonly described as nootropics. However, studies have often involved heterogeneous populations or cognitive disorders that differ from isolated subjective cognitive decline. Consequently, the evidence supporting their routine use in patients with normal objective cognitive performance remains inadequate.

Acetylcholinesterase inhibitors and memantine require particular consideration because they are established treatments for selected neurodegenerative conditions. Their therapeutic indications should not be extended to patients reporting subjective forgetfulness without an appropriate diagnosis. In this setting, potential adverse effects and the absence of demonstrated clinical benefit argue against their routine prescription [4].

6. Practical Management in Everyday Clinical Practice

A practical approach to subjective memory complaints should begin by determining whether the patient has an isolated perception of cognitive decline or evidence of objective impairment. The initial consultation should combine symptom characterization, functional assessment, medication review, and evaluation of sleep, mood, and relevant systemic conditions. Brief cognitive screening may provide additional information, but results should always be interpreted in relation to the clinical history and educational background.

When functional independence is preserved, cognitive screening is reassuring, and no neurological warning signs are identified, the clinician may initially focus on potentially reversible contributors. Laboratory investigations should be individualized, and management should address identified sleep disorders, psychological symptoms, chronic pain, medication effects, and relevant metabolic abnormalities. This strategy may provide clinical benefit while avoiding unnecessary imaging, extensive laboratory panels, and inappropriate pharmacological treatment.

Patients frequently request medication even when no specific underlying disorder has been identified. In such circumstances, discussion should acknowledge that several compounds have produced favorable results in selected studies, while explaining that these findings do not establish consistent clinical efficacy. Decisions should consider the patient's symptoms, comorbidities, concurrent medications, potential adverse effects, and the degree of uncertainty surrounding the intervention. Treatment should not be presented as capable of preventing dementia unless that outcome has been demonstrated in an appropriate population.

Follow-up is an essential component of management. For clinically stable patients with reassuring initial findings, reassessment after approximately 8–12 weeks may be reasonable, particularly when interventions have been directed toward modifiable contributors. This interval represents a pragmatic clinical suggestion rather than a universally established guideline requirement. The follow-up consultation should evaluate symptom progression, daily functioning, treatment response, and the emergence of new neurological or cognitive findings. Persistent deterioration,

objective impairment, or functional decline should prompt additional investigation and consideration of specialist referral [3, 4].

7. Discussion

Subjective memory complaints represent a heterogeneous clinical phenomenon rather than a single disease entity. Their evaluation requires a balance between recognizing potentially important early cognitive changes and avoiding unnecessary diagnostic testing or pharmacological treatment. A structured clinical approach can help distinguish reversible contributors from objective cognitive impairment while providing reassurance and an appropriate plan for follow-up.

An important observation from the available therapeutic literature is that favorable cognitive outcomes have been reported for several pharmacological and nutraceutical interventions. Citicoline, phosphatidylserine-containing formulations, *Bacopa monnieri*, and creatine provide examples of compounds for which positive clinical findings have been documented [9, 11, 12, 14]. These findings are scientifically relevant because they suggest potential therapeutic effects that warrant further investigation. However, the presence of a statistically significant improvement in a cognitive test does not necessarily indicate that patients will experience meaningful improvement in everyday memory functioning.

The distinction between preliminary efficacy signals and established therapeutic benefit is particularly important. Small samples, short treatment durations, heterogeneous populations, secondary outcomes, and inconsistent replication may limit confidence in otherwise promising results. Furthermore, commercial formulations may differ substantially from those evaluated in clinical trials. These limitations emphasize the need to consider both favorable and unfavorable findings when interpreting the evidence. The EFSA evaluation of citicoline, the conflicting trials involving *Bacopa monnieri*, and the recent Cochrane review of *Ginkgo biloba* illustrate the importance of interpreting individual positive studies within the broader evidence base [10, 13, 16].

A major limitation of the therapeutic literature is the frequent inclusion of populations that differ from patients with isolated subjective cognitive decline. Improvements observed in healthy volunteers, individuals with age-associated memory impairment, or patients with objective cognitive disorders cannot automatically be generalized to patients whose principal complaint is perceived forgetfulness. In addition, studies often emphasize neuropsychological test performance rather than patient-reported improvement or functional outcomes.

From a practical perspective, the strongest justification for intervention remains the identification of a clinically relevant contributing condition. Treating sleep disorders, addressing psychological distress, correcting nutritional deficiencies, reviewing potentially contributing medications, and optimizing chronic disease management may offer broader benefits than prescribing an isolated cognitive-enhancing product. Nevertheless, the existence of positive trials should not be disregarded; instead, these studies should be presented transparently, with clear differentiation between evidence supporting further research and evidence sufficient for routine clinical recommendation.

Future clinical trials should specifically enroll individuals with well-characterized subjective cognitive decline, use standardized interventions, and incorporate validated patient-reported outcomes alongside objective cognitive assessments. Longer follow-up periods are also needed to determine whether short-term changes in cognitive test performance translate into meaningful improvements in everyday functioning or influence subsequent cognitive trajectories.

8. Conclusions

Subjective memory complaints are frequent in clinical practice and often occur without demonstrable cognitive impairment or established neurological disease. Nevertheless, these symptoms should not be dismissed without an appropriate clinical evaluation, particularly when they are persistent, progressive, or associated with functional changes.

A practical evaluation should include careful symptom characterization, functional assessment, medication review, sleep and mood evaluation, cognitive screening, and individualized laboratory investigations. Non-pharmacological interventions and correction of identifiable contributors remain the foundation of management.

Selected pharmacological and nutraceutical interventions have demonstrated favorable cognitive outcomes in clinical studies. However, evidence remains insufficient to support routine prescription for isolated subjective cognitive decline. A balanced, individualized approach may reduce unnecessary investigations, avoid inappropriate treatment, and facilitate early recognition of patients requiring specialized cognitive evaluation.

Declarations

Ethics approval and consent to participate: Not applicable. This study is a narrative review of previously published literature and involved no new human or animal research.

Consent for publication: Not applicable.

Availability of data and materials: No original datasets were generated. The evidence discussed was obtained from previously published scientific literature.

Competing interests: None.

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Use of artificial intelligence: The manuscript was initially conceptualized in Portuguese and subsequently developed in English with AI-assisted language support (ChatGPT OpenAI). Responsibility for scientific interpretation, accuracy of the references, and approval of the final manuscript remains with the author.

Table 1: Clinical assessment of subjective memory complaints: potential contributors, clinical clues, and recommended screening instruments

Clinical domain	Symptoms or clinical clues	Suggested instruments or assessments	Clinical implications
Subjective cognitive decline	Persistent perceived memory deterioration, forgetfulness, word-finding difficulties	SCD-I clinical criteria; structured cognitive history; informant interview	Characterize onset, progression, and subjective–objective discrepancy [1, 3]
Objective cognitive performance	Difficulty learning new information, disorientation, progressive cognitive symptoms	Mini-Mental State Examination (MMSE); Montreal Cognitive Assessment (MoCA)	Identify objective impairment; interpret scores according to age, education, and validated cutoffs [4]
Functional independence	Difficulty managing medications, finances, appointments, or household activities	Lawton–Brody Instrumental Activities of Daily Living Scale; Katz ADL Index	Distinguish preserved function from emerging functional impairment [4]
Insomnia and sleep quality	Difficulty initiating or maintaining sleep, nonrestorative sleep, fatigue	Insomnia Severity Index (ISI); Pittsburgh Sleep Quality Index (PSQI)	Identify sleep disturbances potentially contributing to cognitive complaints [5, 6]
Excessive daytime sleepiness	Daytime drowsiness, fatigue, reduced concentration	Epworth Sleepiness Scale (ESS)	Quantify sleepiness; does not independently diagnose sleep apnea
Obstructive sleep apnea	Snoring, witnessed apnea, obesity, hypertension, daytime sleepiness	STOP-Bang Questionnaire; Berlin Questionnaire	Estimate apnea risk and determine need for formal sleep evaluation
Depression	Low mood, anhedonia, fatigue, impaired concentration	Patient Health Questionnaire-9 (PHQ-9); Geriatric Depression Scale (GDS)	Identify depressive symptoms requiring assessment and treatment
Anxiety	Excessive worry, restlessness, subjective cognitive difficulties	Generalized Anxiety Disorder-7 (GAD-7)	Evaluate anxiety as a potential contributor to cognitive symptoms
Chronic pain and fibromyalgia	Widespread pain, fatigue, fibro fog, nonrestorative sleep	Numeric Rating Scale (NRS); Revised Fibromyalgia Impact Questionnaire (FIQR)	Assess pain burden, functional impact, and associated cognitive complaints [7]
Medication-related cognitive effects	Sedation, impaired attention, recent medication changes, polypharmacy	Medication reconciliation; anticholinergic burden assessment	Identify potentially reversible medication-related contributors [4, 6]
Sensory impairment	Hearing or visual difficulties interfering with communication and testing	Hearing and vision assessment	Identify sensory limitations affecting cognitive evaluation [6]
Progressive cognitive disorder	Objective impairment, functional deterioration, informant-confirmed decline	Serial cognitive testing; formal neuropsychological evaluation when indicated	Consider neuroimaging and specialist referral [3, 4]

Abbreviations: ADL, activities of daily living; SCD-I, Subjective Cognitive Decline Initiative.
Note: Screening questionnaires do not replace clinical diagnosis. The instruments listed are established assessment options; references [1,3–7] support the overall clinical approach, but individual instrument-validation publications should be added to the manuscript bibliography.

Table 2: Laboratory and complementary investigations in patients with subjective memory complaints

Investigation	Main clinical purpose	When to request	Interpretation or clinical action
Complete blood count	Detect anemia and hematological abnormalities	Common initial evaluation	Investigate abnormalities according to clinical context
Thyroid-stimulating hormone (TSH)	Identify thyroid dysfunction	Common initial evaluation	Assess thyroid status and treat confirmed dysfunction
Vitamin B12	Identify potentially reversible deficiency	Common initial evaluation	Investigate and correct deficiency when confirmed
Fasting glucose or HbA1c	Assess diabetes or dysglycemia	According to risk and routine care	Optimize glycemic management
Creatinine and estimated GFR	Evaluate renal dysfunction	Common initial evaluation	Assess metabolic contributors and medication safety
Sodium, potassium, and calcium	Identify electrolyte and metabolic abnormalities	Common initial evaluation	Correct clinically significant abnormalities
Liver function tests	Assess hepatic or systemic disease	According to clinical context	Investigate abnormal results
Folate	Identify nutritional deficiency	Dietary risk, malabsorption, macrocytosis	Correct documented deficiency
Ferritin and iron studies	Investigate iron deficiency associated with fatigue	Suspected iron deficiency	Treat confirmed deficiency
Vitamin D	Assess deficiency when independently indicated	Relevant risk factors or clinical indications	Not recommended solely to investigate subjective forgetfulness
HIV and syphilis serology	Investigate selected infectious etiologies	Relevant exposure, clinical features, or unexplained cognitive syndrome	Manage according to confirmed diagnosis
ESR and CRP	Evaluate inflammatory or systemic disease	Clinical suspicion of inflammation	Further investigation according to findings
Brain MRI	Identify structural, vascular, or other intracranial abnormalities	Objective impairment, progressive symptoms, focal signs, or atypical presentation	Consider specialist evaluation
Formal neuropsychological testing	Characterize cognitive domains in detail	Persistent unexplained symptoms, diagnostic uncertainty, or discordant screening	Clarify cognitive profile and establish baseline

Abbreviations: CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; GFR, glomerular filtration rate; MRI, magnetic resonance imaging.
Note: The investigation should be individualized. A broader diagnostic panel may be appropriate when objective cognitive impairment or a cognitive-behavioral syndrome is suspected [4]. Routine extensive testing is not required for every patient with isolated subjective complaints.

Table 3: Pharmacological and nutraceutical interventions with at least one reported favorable cognitive finding

Intervention	Dose and duration evaluated	Evidence of favorable outcomes	Common or clinically relevant adverse effects	Interpretation
Citicoline	500 mg/day for 12 weeks	Improved episodic and composite memory outcomes in older adults with age-associated memory impairment [9]	Headache, gastrointestinal discomfort, nausea; generally well tolerated in studied doses	Positive randomized trial, but broader evidence remains inconclusive [10]
Phosphatidylserine combined with omega-3 fatty acids	Combination formulation evaluated for 15 weeks; component doses should be confirmed from the original trial	Improved immediate verbal recall in older adults with memory complaints [11]	Gastrointestinal discomfort, nausea, possible intolerance to formulation components	Favorable findings apply to the combined formulation, not necessarily phosphatidylserine alone
Bacopa monnieri	Approximately 300 mg/day of standardized extract for 12 weeks in the positive trial	Favorable findings in selected memory outcomes [12]	Nausea, abdominal cramps, increased bowel movements, diarrhea	Positive trial exists; subsequent trial did not confirm primary cognitive benefit [13]
Creatine	Variable regimens across trials, commonly 3–20 g/day; durations varied	Favorable memory outcomes in pooled analysis of heterogeneous populations [14, 15]	Gastrointestinal discomfort, bloating, water retention, weight increase	Potential cognitive signal; no established treatment regimen for isolated SCD
Ginkgo biloba	Standardized extracts commonly studied at 120–240 mg/day; duration varied	Selected favorable outcomes reported in some cognitive populations [16]	Headache, gastrointestinal symptoms, dizziness; possible bleeding-related interactions	Positive findings in some populations, but evidence remains very uncertain for isolated subjective complaints
Omega-3 fatty acids	EPA/DHA doses and durations varied across studies	Selected favorable cognitive outcomes in heterogeneous populations	Fishy aftertaste, gastrointestinal discomfort; possible bleeding-related concerns at some doses	No established dose or indication specifically for isolated SCD [5, 6]

Abbreviations: DHA, docosahexaenoic acid; EPA, eicosapentaenoic acid; SCD, subjective cognitive decline.
Notes: This table includes interventions with at least one reported favorable finding, even when other studies have produced negative or inconclusive results. Such conflicting evidence is addressed in the manuscript discussion rather than by repeating these interventions in Table 4. The doses are descriptive of research exposure, not prescribing recommendations. Adverse-effect profiles require confirmation against formulation-specific clinical trial reports and authoritative drug information. References [9–16] support the corresponding efficacy discussions.

Table 4: Pharmacological interventions without established positive evidence for isolated subjective cognitive decline

Intervention	Doses used in other clinical indications or investigated populations	Evidence in isolated subjective memory complaints	Common or clinically relevant adverse effects	Clinical recommendation
Donepezil	5–10 mg/day in approved dementia indications	No established positive evidence supporting treatment of isolated SCD	Nausea, diarrhea, vomiting, insomnia, muscle cramps, bradycardia, syncope	Not recommended solely for subjective memory complaints
Rivastigmine	Oral 1.5–6 mg twice daily; transdermal patches according to approved labeling	No established positive evidence supporting isolated SCD	Nausea, vomiting, diarrhea, weight loss, dizziness, bradycardia	Not recommended without an appropriate neurological indication
Galantamine	8–24 mg/day, depending on formulation and titration	No established positive evidence supporting isolated SCD	Nausea, vomiting, diarrhea, anorexia, weight loss, bradycardia	Not recommended solely for subjective complaints
Memantine	Commonly titrated to 20 mg/day in approved dementia indications, with renal adjustment when needed	No established positive evidence supporting isolated SCD	Dizziness, headache, constipation, confusion	Not recommended without an appropriate neurological indication
Piracetam	Regimens vary widely by indication and jurisdiction; no validated SCD dose	Insufficient reliable evidence establishing benefit specifically in isolated SCD	Nervousness, agitation, insomnia, gastrointestinal discomfort	Routine use unsupported
Vitamin E without deficiency	Variable doses in cognitive studies; no established SCD regimen	No established symptomatic benefit for isolated SCD	Gastrointestinal symptoms; potential bleeding risk at high doses	Not recommended specifically for memory enhancement
Vitamin D without deficiency	Variable supplementation regimens; no established SCD regimen	No established symptomatic benefit for isolated SCD	Excessive intake may cause hypercalcemia and renal complications	Supplement only when clinically indicated
B-complex vitamins without deficiency	Variable formulations and doses	No established consistent benefit for isolated SCD	Generally well tolerated at nutritional doses; high-dose vitamin B6 may cause neuropathy	Correct deficiencies rather than prescribing indiscriminately
Unstandardized nootropic combinations	No standardized regimen	Insufficient reliable evidence	Variable adverse effects, interactions, and product-quality concerns	Avoid routine use without adequate evidence

Abbreviations: SCD, subjective cognitive decline.
Notes: The absence of established positive evidence for isolated SCD does not mean that every drug listed has produced exclusively negative trials in every cognitive disorder. Donepezil, rivastigmine, galantamine, and memantine have separate indications in selected neurological conditions. The doses shown describe use in other populations and must not be interpreted as recommended doses for subjective memory complaints. The general diagnostic and treatment distinction is consistent with reference [4]; drug-specific dosing, contraindications, and adverse effects should additionally be supported by prescribing information or dedicated pharmacological references before submission.

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