INFORMATICS JOURNALS

Short Review

Journal of Natural Remedies
DOI: 10.18311/jnr/2026/50239

eISSN: 2320-3358 | Vol 26(2), 2026 | pp. 353-364

Alzheimer's Disease Revisited: An Integrative Review of Modern Neurology and *Ayurvedic* Therapeutics

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Abstract

Background: Alzheimer's Disease (AD) is a progressive, irreversible neurodegenerative disorder primarily affecting older adults, characterised by a gradual decline in memory, cognition, and behaviour. It is the leading cause of dementia worldwide, and its prevalence is rising rapidly with ageing populations. From a biomedical perspective, AD involves pathological hallmarks such as Amyloid-β (Aβ) plaque deposition, neurofibrillary tangles of hyperphosphorylated tau protein, cholinergic neuronal loss, oxidative stress, mitochondrial dysfunction, and chronic neuroinflammation. **Aim:** To present an integrative review of Alzheimer's disease from both modern neurological and *Ayurvedic* perspectives, highlighting potential therapeutic convergence and the role of *Ayurvedic* herbs in neuroprotection. **Methods:** Relevant scientific and classical *Ayurvedic* literature was reviewed using electronic databases, focusing on keywords such as "Alzheimer's disease", " neuroprotection", and "*Medhya Rasayana*". Studies describing *Ayurvedic* formulations and their pharmacological mechanisms relevant to AD were critically analysed and compared with modern pharmacotherapeutic approaches. **Results:** Current modern therapies, such as cholinesterase inhibitors (donepezil, rivastigmine) and NMDA receptor antagonists (memantine), primarily offer symptomatic relief with limited disease-modifying potential. *Ayurvedic* management, described under *Smritibhramsha*, employs *Medhya Rasayana* herbs including *Bacopa monnieri, Withania somnifera*, *Centella asiatica*, *Convolvulus pluricaulis*, and *Curcuma longa,* which exhibit antioxidant, anti-inflammatory, and anti-amyloid activities. Integrative insights suggest these botanicals may enhance neuronal resilience and cognitive function. **Conclusion:** Integrating *Ayurvedic* neuroprotective principles with modern therapeutic understanding offers a holistic approach to AD management. Standardisation, clinical validation, and mechanistic studies are required to substantiate these traditional claims.

Major Findings: This review explores AD's pathophysiology, current diagnostic and therapeutic strategies, *Ayurvedic* understanding and interventions, and suggests how integrative approaches may offer new directions in AD management.

Keywords: Alzheimer's Disease, *Ayurvedic* Medicine, Cognitive Decline, Cholinesterase Inhibitors, *Rasayana* Therapy, *Smritibhramsha*

Abbreviations: AD – Alzheimer's Disease; Aβ – Amyloid-beta; APOE – Apolipoprotein E; ARIA – Amyloid-related Imaging Abnormalities; BBB – Blood-brain Barrier; CNS – Central Nervous System; CSF – Cerebrospinal Fluid; CT – Computed Tomography; FDA – Food and Drug Administration; FDG-PET – Fluorodeoxyglucose Positron Emission Tomography; MMSE – Mini-mental State Examination; MoCA – Montreal Cognitive Assessment; MRI – Magnetic Resonance Imaging; NMDA – N-Methyl-D-Aspartate; RCT – Randomised Controlled Trial.

(Article Chronicle: Article Received on: 16.06.2025; Revised on: 05.11.2025; Accepted on: 27.01.2026)

1. Introduction

Alzheimer's Disease (AD) is a progressive and irreversible neurodegenerative disorder that primarily affects the elderly, leading to the gradual deterioration of memory, thinking, behaviour, and the ability to perform daily activities[1]. It is recognised as the most common cause of dementia worldwide, responsible

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for approximately 60–70% of all dementia cases. Recent estimates indicate that around 55 to 57 million people were affected by dementia in 2020–2021, and projections suggest that this number could rise to over 139 million by 2050 due to the global increase in life expectancy and ageing populations. The rising prevalence of AD presents a major public health concern, imposing significant economic and emotional burdens on patients, families, and healthcare systems[2]. From the standpoint of modern biomedicine, AD is defined by several key pathological features, including extracellular accumulation of Amyloid-β (Aβ) plaques, intracellular neurofibrillary tangles made of hyperphosphorylated tau protein, widespread neuronal loss, mitochondrial dysfunction, oxidative stress, cholinergic system deficits, and chronic neuroinflammation[3-5]. These changes especially impact the hippocampus and associated cortical areas, which are responsible for memory and cognition. Current treatments are symptomatic and include cholinesterase inhibitors such as donepezil, rivastigmine, and galantamine, which increase acetylcholine availability, and memantine, an NMDA receptor antagonist that reduces glutamate-mediated excitotoxicity[6-8]. In recent years, disease-modifying agents like anti-amyloid monoclonal antibodies (*e.g.*, aducanumab and lecanemab) have been approved, but their clinical efficacy and safety remain under debate due to mixed results, high costs, and risks of adverse effects such as Amyloid Related Imaging Abnormalities (ARIA). Because these modern interventions offer only modest symptomatic relief and limited disease modification, there has been a growing interest in complementary and alternative medical systems that may offer holistic and preventive strategies. *Ayurveda*, the ancient Indian system of medicine, provides a unique conceptual framework for understanding cognitive decline. It does not categorise Alzheimer's as a distinct disease but describes similar symptoms under the conditions of *Smritibhramsha* (memory loss), *Medha Kshaya* (loss of intellect), and disorders of *Manovaha Srotas* (mental channels), which are largely associated with vitiation of *Vatadosha*, particularly in the elderly. According to *Ayurvedic* philosophy, maintaining the balance of *Vata*, *Pitta*, and *Kapha* doshas is essential for mental and physical health, and the imbalance of *Vata* in the ageing brain contributes to neurodegeneration, mental fog, and memory loss. Treatment in *Ayurveda* focuses on lifestyle management, dietary modifications, and most importantly, the use of *Medhya Rasayana*-a category of rejuvenating herbs known for their cognition-enhancing and neuroprotective effects. Prominent among these are *B. monnieri* (*Brahmi*), *W. somnifera* (*Ashwagandha*), *C. asiatica* (*Mandukaparni*), *C. pluricaulis* (*Shankhpushpi*), and *Curcuma longa* (Turmeric)[9]. These herbs are traditionally used to enhance intellect, stabilise mood, reduce anxiety, and promote longevity. Modern preclinical research supports their efficacy; for instance, *Brahmi* is shown to improve synaptic activity and learning behaviour, *Ashwagandha* exhibits anti-inflammatory and neuroregenerative effects, and Turmeric's active component, curcumin, inhibits Aβ aggregation and oxidative stress. *C. asiatica* is known to enhance neurogenesis and blood flow to the brain, while *Shankhpushpi* is valued for its calming and anti-anxiety properties. Collectively, these herbs exhibit antioxidant, anti-inflammatory, anti-amyloidogenic, and neurotrophic properties that align well with the key pathogenic mechanisms of AD. Thus, *Ayurvedic* formulations, when scientifically validated and appropriately standardised, may provide neuroprotective support and enhance the quality of life in AD patients. This review aims to provide a comprehensive understanding of Alzheimer's disease by integrating both modern medical and *Ayurvedic* perspectives. It covers the pathophysiology of AD, current diagnostic and therapeutic practices, *Ayurvedic* interpretation of cognitive decline, and the pharmacological potential of *Medhya Rasayana* herbs[10]. A comparative analysis will be included to evaluate similarities and differences in objectives, mechanisms, and therapeutic approaches between the two systems. Moreover, this review will explore the scope for integrative treatment models that combine the strengths of both disciplines. While modern medicine excels in acute diagnosis and targeted molecular interventions, *Ayurveda* emphasises preventive care, systemic balance, and individualised treatment. Merging these strengths could pave the way for more effective and holistic management of Alzheimer's disease. However, rigorous clinical research, standardisation of herbal formulations, and regulatory support are essential to validate and integrate *Ayurvedic* treatments into mainstream Alzheimer's

care. By fostering interdisciplinary collaboration and evidence-based evaluation, a truly integrative strategy for tackling AD may be realised in the future.

2. Materials and Methods

This review was conducted through a detailed survey of the scientific literature related to Alzheimer's disease, focusing on both modern and *Ayurvedic* approaches. A comprehensive literature search was carried out using databases such as Google Scholar, PubMed, and ScienceDirect. Relevant search terms included "Alzheimer's Disease", "*Ayurveda*", "*Rasayana*", "*Ashwagandha*", "Cognitive decline", "Neurodegeneration", "*Panchakarma*" and "Cholinesterase inhibitors". Articles published in English between 2000 and 2024 were considered, with particular emphasis on those published after 2015. Only articles discussing the disease's pathophysiology, diagnosis, therapeutic interventions, herbal treatments, *Ayurvedic* formulations, and integrative or comparative frameworks were included. Studies not available in full text, lacking scientific validation, or containing duplicate data were excluded. Data from selected articles were organised thematically to highlight various aspects of AD management. *Ayurvedic* therapies were aligned with corresponding biomedical concepts where possible. Ethical approval was not required as this review is based solely on existing published literature.

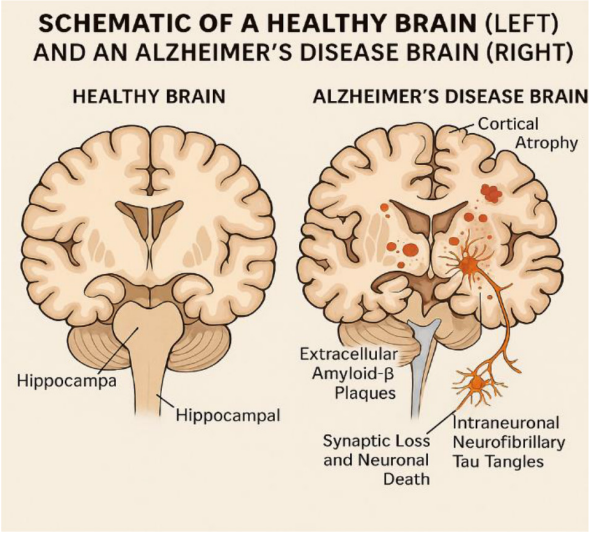


Figure 1. Schematic comparison ofa healthy brain and an Alzheimer's disease brain.

3. Pathophysiology of Alzheimer's Disease

The hallmark pathologies of AD are extracellular Amyloid-β (Aβ) plaques and intracellular neurofibrillary tangles of hyperphosphorylated tau protein. According to the amyloid cascade hypothesis[11], Aβ accumulation triggers tau pathology and neuronal injury[12]. However, failures of anti-amyloid trials suggest a more complex picture. Current views posit that Aβ and tau oligomers both contribute, possibly in parallel, to synaptic dysfunction and cell death. For example, Aβ oligomers disrupt synaptic signalling and induce oxidative stress, while tau tangles impair neuronal transport. Neuroinflammation also plays a critical role: chronic activation of microglia and astrocytes around Aβ plaques releases cytokines that exacerbate neurodegeneration. In fact, persistent glial-driven inflammation is now seen as a primary driver of neuronal loss in AD. Another key feature is degeneration of the cholinergic system. Loss of cholinergic neurons in the basal forebrain leads to depletion of acetylcholine, which contributes to memory and attention deficits. This "cholinergic hypothesis" is supported by symptomatic improvement from acetylcholinesterase inhibitor drugs[13]. Other factors in AD pathogenesis include oxidative damage (from excess free radicals) and vascular changes. High oxidative stress markers and reduced antioxidants correlate with cognitive decline. Genetic risk factors also influence pathology: the APOE ε4 allele is the highest known genetic risk for late-onset AD. Carriers of APOE ε4 show increased Aβ deposition and earlier onset of disease. "Figure 1" shows the AD brain exhibits marked cortical and hippocampal atrophy with enlarged ventricles, as well as extracellular amyloid-β plaques (red deposits) and intraneuronal neurofibrillary tau tangles (orange aggregates). These pathological features lead to synaptic loss and neuronal death.

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4. Modern Approach to Diagnosis

Diagnosis of AD in modern medicine combines clinical assessment with biomarkers. Clinicians first evaluate symptoms, perform cognitive tests (*e.g.* MMSE, MoCA) and rule out reversible causes. Neuroimaging is then used: Brain MRI or CT may reveal atrophy (particularly in the medial temporal lobes/hippocampus)[14,15] and exclude other lesions. More definitive biomarkers include PET scans and fluid assays. PET imaging can detect reduced glucose metabolism (FDG-PET) or directly visualise amyloid or tau aggregates using specific tracers. For example, amyloid PET can identify amyloid accumulation early. Measurements of Aβ42 and phosphorylated tau in Cerebrospinal Fluid (CSF) are also diagnostic[16]. Importantly, recent advances have produced blood-based biomarkers: assays for plasma phospho-tau can now predict AD pathology with high accuracy, allowing less invasive screening. The 2024 NIA-AA criteria formally incorporate these biomarkers into AD diagnosis, emphasising a biological (not purely clinical) definition. Treatment is primarily symptomatic, as no cure exists. The mainstays are cholinesterase inhibitors (donepezil, rivastigmine, galantamine) for mild-to-moderate AD and memantine (an NMDA antagonist) for moderate-to-severe AD. Cholinesterase inhibitors boost acetylcholine levels and have been shown in multiple trials to produce modest cognitive benefit (*e.g.* ADAS-Cog score improvements) (Figure 2). A Cochrane review concluded that all three approved cholinesterase inhibitors are effective in mild-to-moderate AD, though effects are small. Memantine, which modulates glutamate signalling, also slows decline in moderate/severe AD (often used in combination with a cholinesterase inhibitor). Recently, disease-modifying therapies targeting amyloid have emerged. Monoclonal antibodies such as lecanemab and donanemab have FDA approval for early AD. These agents can reduce amyloid burden and modestly slow cognitive decline. For instance, lecanemab (Leqembi) and donanemab (Kisunla), given intravenously, have shown significant amyloid clearance, though their clinical benefits are still debated and they carry risks (*e.g.* ARIA brain swelling). Aducanumab (Aduhelm) was earlier approved with controversy; more recently, donanemab also received conditional approval. Aside from drugs, lifestyle interventions (*e.g.* cognitive stimulation, exercise, Mediterranean diet) are recommended to reduce risk and support patients. For example, higher adherence to a Mediterranean diet has been associated with a ~11% lower dementia risk. Overall, modern management of AD involves a combination of cognitive tests, imaging/biomarkers for diagnosis, and mainly pharmacological treatments to manage symptoms, while continuing research seeks better preventive and disease-modifying solutions[17].

5. *Ayurvedic* Perspective on Alzheimer's Disease

Ayurvedic medicine views cognitive decline holistically, attributing it to imbalances in the body's humours (*doshas*) and toxins (*Ama*) affecting the mind (*Manas*) and memory (*Smriti*). In *Ayurveda*, the brain and nervous system are governed by *Medha, Smriti*, and *Buddhi*. Impairment of memory is often conceptualised as *Smritibhransha* (loss of memory) caused by aggravated *Vata dosha* and/or accumulation of *Ama*. The treatment focus is restoration of balance and rejuvenation (*rasayana therapy*). *Ayurvedic* therapies for AD emphasise cleansing procedures (*Panchakarma*) and formulations meant to enhance nerve strength and cognitive function[18]. *Medhya Rasayana* are specialized herbal combination historically prescribed for memory and intelligence. Key *Ayurvedic* herbs with evidence of cognitive benefits include *B. monnieri* (*Brahmi*), *C. asiatica* (*Mandukaparni*), *W. somnifera* (*Ashwagandha*), *C. pluricaulis* (*Shankhpushpi*), and *Curcuma longa* (Turmeric). These plants contain phytochemicals (*e.g.* bacosides, withanolides, asiaticosides, curcuminoids) with antioxidant, anti-inflammatory, and neuroprotective properties. For instance, a PNAS study showed that *Ashwagandha* extract administered orally for 30 days reversed amyloid pathology and rescued cognitive deficits in Alzheimer-model mice. Similarly, *Bacopa* extract has been found to reduce β-amyloid plaques by up to 60% in a transgenic AD mouse model and improve memory performance. *C. asiatica* is used in *Ayurveda* for memory enhancement; in 5xFAD AD mice, 3-month treatment with *Centella* extract improved memory and reduced amyloid plaque burden. *Shankhpushpi* (*C. pluricaulis*) has shown memory-boosting effects in rodents. Curcumin, the active component of turmeric,

is studied for its ability to inhibit Aβ aggregation and tau phosphorylation; in animal models, oral curcumin prevented plaque formation and cognitive decline[19]. *Ayurvedic* formulations often combine these herbs. For example, *Brahmi Ghrita* (medicated ghee with *Brahmi* and other herbs) is a classical nootropic preparation. Classical texts and modern overviews note that plants like *Brahmi, Ashwagandha, Shankhpushpi, Gotu Kola* (*Centella*), and *Guggulu* (*Commiphora*) are used to "slow brain ageing and enhance memory". The *Ayurvedic* approach also includes supportive practices: diet, meditation, and lifestyle adjustments aimed at balancing *Vata* (often implicated in neurological disorders) and nourishing *Dhatus* (tissues). Importantly, *Ayurveda* emphasises individual constitution (*Prakriti*), so treatments are personalised. Overall, the *Ayurvedic* perspective combines ancient knowledge of *Medhya Rasayana* herbs and holistic principles to address the mind-body aspects of dementia[20]. Scientific research on these herbs is growing: many have demonstrated neuroprotective activities (antioxidant, anti-inflammatory, anti-amyloid, and acetylcholinesterase inhibitory effects). However, high-quality clinical evidence in AD patients remains scarce. Nonetheless, these plants' multi-target actions suggest they might complement conventional therapies, for instance, by reducing oxidative stress or inflammation beyond what single-target drugs achieve (Figure 3).

6. Modern Treatments

Alzheimer's treatments today are largely symptomatic, aiming to slow decline and ease symptoms. FDA-approved drugs include cholinesterase inhibitors and NMDA receptor antagonists. Cholinesterase inhibitors (donepezil, rivastigmine, galantamine) boost acetylcholine levels and can modestly improve memory and daily function in mild-to-moderate AD[21]. Memantine, an NMDA antagonist, is used in moderate-to-severe stages to regulate glutamate activity and may prolong independence. Combination therapies (*e.g.* Namzaric, donepezil + memantine) and adjuvant drugs (*e.g.* rivastigmine patch, atypical antipsychotics like brexpiprazole for agitation) provide additional symptomatic relief. Cholinesterase inhibitors: Donepezil, rivastigmine, and galantamine are first-line drugs for mild–moderate AD[22]. They prevent acetylcholine breakdown, offering brief cognitive and functional improvement, though effects wane as the disease progresses. NMDA antagonist: Memantine treats moderate-to–severe AD by dampening glutamate excitotoxicity. It is often added when patients no longer respond to cholinesterase inhibitors and can modestly slow the loss of daily living abilities. Amyloid-targeting antibodies: In 2021–2023, the FDA approved monoclonal antibodies (lecanemab, donanemab) for early AD. These drugs bind β-amyloid plaques and have been shown to slightly slow cognitive decline and clear amyloid on PET scans. Patients require amyloid confirmation (via PET or CSF) before treatment, and must be monitored for Amyloid-Related Imaging Abnormalitie (ARIA) that can cause brain swelling or haemorrhage. Aducanumab (Aduhelm), approved in 2021 under an accelerated pathway, was similarly aimed at amyloid reduction, but its modest benefit and high cost led Biogen to discontinue it in 2024. Other interventions: Supportive therapies

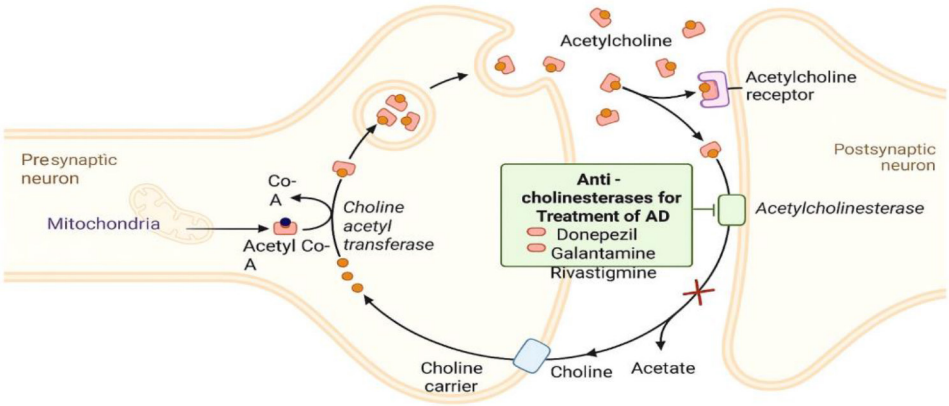


Figure 2. The anti-cholinesterases mechanism of action in Alzheimer's disease (AD).

include cognitive stimulation/training, management of behavioural symptoms, and caregiver education[23]. Addressing vascular and metabolic risk factors (hypertension, diabetes), ensuring social engagement, and providing occupational therapy are standard care. Non-pharmacological strategies complement drugs. Healthy lifestyle changes–especially aerobic exercise and a Mediterranean-style diet–are strongly recommended to delay cognitive decline. For example, the WHO endorses ≥150 minutes/week of moderate exercise to improve brain health. Though trial evidence is mixed, eating fruits, vegetables, whole grains, and unsaturated fats (Mediterranean diet) correlates with larger brain volume and better cognition[24]. Cognitive training, social activities, and structured routines can also help maintain function in early stages. Despite these approaches, no cure exists. All current drugs provide only modest, short-lived benefits. Cholinesterase inhibitors and memantine can slow symptom progression by months, but ultimately do not halt neurodegeneration. The new amyloid antibodies offer proof-of-concept for disease modification, but their long-term efficacy and safety remain uncertain. Challenges include high cost, restrictive eligibility, and risk of serious side effects (*e.g.* ARIA with antibodies).

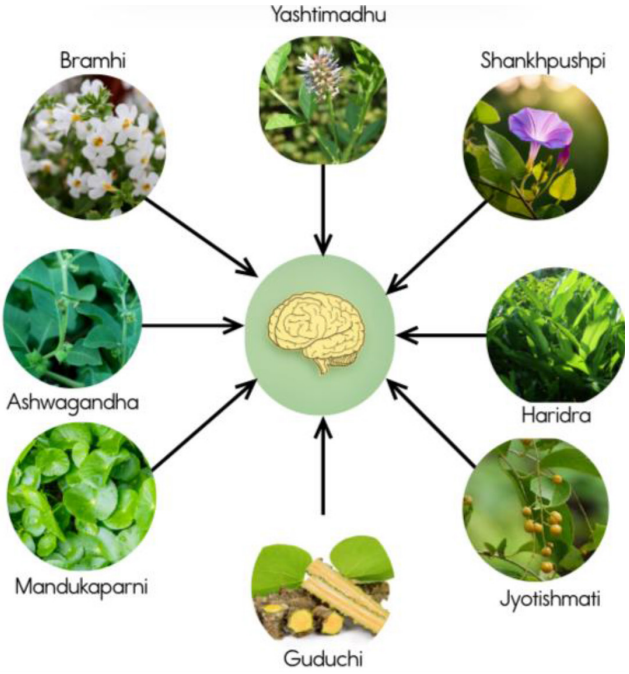


Figure 3. Shows *Ayurvedic* herbs used in the management of Alzheimer's disease.

Modern treatments focus on managing symptoms and delaying decline, but more effective, safer therapies are urgently needed. These various pharmacological and non-pharmacological approaches are used in combination to manage the disease. A summary of these key modern therapeutics is provided in Table 1.

7. *Ayurvedic* Treatment

In *Ayurveda*, dementia-like symptoms are framed as age-related "*Smritibhramsha*" (memory loss), often linked to *doshic* imbalances. Cognitive decline in old age (*Jarjariavastha*) is seen as an aggravation of *Vata* (and sometimes *Pitta*) *dosha*, leading to depletion of *Medha* (brain tissue) and *Ojas* (vitality). Treatment thus aims to balance doshas and rejuvenate the mind-body system. Classical *Ayurvedic* texts emphasise *Rasayana* (rejuvenation) therapy and *Panchakarma* (purification) procedures to slow degeneration.

7.1 *Rasayana* Therapy

Medhya Rasayana herbs ("memory enhancers") are cornerstone treatments. Key herbs include *Brahmi* (*B. monnieri*), *Mandukaparni* (*C. asiatica*), *Shankhpushpi* (*C. pluricaulis*), *Ashwagandha* (*W. somnifera*), *Guduchi* (*Tinospora cordifolia*)[25-29], *Vacha* (*Acorus calamus*), *Tagara* (*Valeriana wallichii*), and others. These plants are believed to nourish the brain (*Balya*) and enhance *Smriti* (memory). They exhibit antioxidant, anti-inflammatory, and anti-amyloid effects in studies. *Ayurvedic* formulations often use medicated ghritas (clarified butter preparations) and herbal powders – for example, *Brahmi Ghrita*, *Mahapanchagavya Ghrita*, *Chyawanprash*, and *SaraswataChurna* – to deliver these *rasayanas* deeply into neural tissues. *Ghrita* itself (especially cow-derived ghee) is valued as an *anupana* (conduit) to carry medicated oils to the brain.

7.2 *Panchakarma* Purification

Before *Rasayana*, gentle detoxification is used to remove accumulated toxins and prepare the body. Practices include *Mridu Virechana* (mild purgation), *Sneha Basti* (oil enemas), *MatraVasti* (small-quantity oil enemas), *Shirovasti* (retaining medicated oil on the head), and *Nasya* (herbal oils dropped into the nostrils). These therapies are tailored to the individual's age and

strength, aiming to alleviate *Vata* without weakening *Dhatus*. For example, *Shirovasti* with *Brahmi*-infused ghee or *Nasya* with herbal oils calms the nervous system and supports memory[30].

7.3 Lifestyle and Support

Ayurveda also prescribes a *sattvic* (pure), easy-to-digest diet and daily routines (*dinacharya*) for brain health. Practices include *yoga asanas*, *pranayama* (breathing exercises), meditation, and mantra therapy (*Satvavajaya Chikitsa*) to stabilise the mind (*Manas*) and improve *Ojas*. Proper sleep hygiene and stress management are stressed. *Ayurvedic Rasayana* therapy explicitly aims to improve memory, stamina, sense perception, and longevity. *Ayurvedic* psychologists advocate a calm environment, positive social support, and individualized counseling to mitigate anxiety and agitation[31].

Ayurvedic clinicians report that combined *Rasayana* and *Panchakarma* protocols can improve the quality of life in dementia patients. Several clinical studies have validated aspects of *Ayurvedic* medicines: For example, *Ashwagandha* root extract has shown neurite regeneration, synaptic restoration, and reduction of amyloid in animal models, while *Brahmi* (*Bacopa*) extracts have improved learning/memory and reduced amyloid in trials. Polyherbal formulations like *Saraswata Churna* and *Manasamitra Gutika* are documented to inhibit acetylcholinesterase, reduce oxidative stress, and enhance cognition in preclinical models.

Nonetheless, most evidence is preliminary or experiential. *Ayurveda* emphasises early, long-term treatment; its interventions tend to act slowly and holistically, aiming to preserve brain tissue, fortify immunity, and support caregivers as part of care. This holistic regimen aims to preserve brain tissue and support caregivers. These key *Ayurvedic* interventions are summarised in Table 2.

8. Modern Treatments *vs*. Herbal (Allopathy *vs*. *Ayurveda*) Treatment

Allopathic medicine for AD is largely reductionist: It identifies specific pathologic targets (acetylcholine deficiency, amyloid plaques, tau tangles) and uses single-agent drugs to address them. For example, cholinesterase inhibitors act only on one neurotransmitter system, and anti-amyloid monoclonals target one protein. In contrast, *Ayurveda* employs a holistic, systemic approach. It seeks to restore the overall balance of the body's *doshas* and *dhatus* (tissues) and strengthen innate healing (*Agni, Ojas*). Rather than one drug-one target, *Ayurvedic* regimens use multi-herb "*Rasayana*"

Table 1. Key modern therapeutics in Alzheimer's disease

Sr. No.	Drug / Intervention	Class	Mechanism of Action	Main Benefits
1	Donepezil	Cholinesterase inhibitor	Inhibits acetylcholinesterase → ↑ acetylcholine	Modest cognitive and functional gains
2	Rivastigmine (patch/tablet)	Cholinesterase inhibitor	Inhibits acetylcholinesterase and butyrylcholinesterase	Comparable efficacy, the patch improves compliance
3	Galantamine	Cholinesterase inhibitor	Inhibits acetylcholinesterase; modulates nicotinic receptors	Cognitive improvement, possible neuroprotection
4	Memantine	NMDA receptor antagonist	Blocks pathologic NMDA-mediated excitotoxicity	Slows functional decline,well-tolerated
5	Lecanemab	Anti-amyloid monoclonal antibody	Binds aggregated Aβ → microglial clearance	Reduces amyloid burden, slows decline
6	Donanemab	Anti-amyloid monoclonal antibody	Targets pyroglutamate Aβ → plaque remodelling	Significant amyloid clearance, cognitive benefit
7	Cognitive Stimulation	Behavioral intervention	Engages neural circuits → synaptic plasticity	Improves memory and daily function
8	Mediterranean-style Diet	Lifestyle / Nutritional therapy	↑ antioxidants and anti-inflammatory nutrients → neuroprotection	Delays decline, vascular support

formulas and lifestyle changes that are believed to nourish mind-body networks.

8.1 Efficacy and Mechanisms

Western drugs can yield measurable short-term gains on cognitive scales (*e.g.*, 1–3 point MMSE improvement), but these gains are usually temporary. Their mechanisms are well-characterised (enzyme inhibition, receptor blockade)[31]. *Ayurvedic* remedies have broad-spectrum effects – they often contain antioxidants, polyphenols and neuromodulators that can reduce neuroinflammation, oxidative stress, and even amyloid burden, though in gentler ways. *Ayurveda* also addresses psychological and social factors, using counselling (*Satvavajaya*) and spiritual practices (*Daivavyapashraya*) alongside herbs. This can improve mood, compliance, and quality of life, effects not captured by typical drug trials[32].

8.2 Strengths

Modern therapies have the advantage of standardised dosing and clinical trial support for at least symptomatic benefit. They can act quickly on target symptoms. *Ayurvedic* treatments are praised for having fewer acute side effects (when properly prescribed) and for potentially slower, sustained benefits. The polyherbal formulas are believed to "work synergistically" through multiple biochemical pathways, and are often more affordable and culturally familiar in regions where *Ayurveda* is practiced[33,34]. Because *Ayurveda* tailors regimens to the individual's constitution (*Prakriti*) and disease stage, its approach is inherently personalised, whereas Western drug regimens tend to be one-size-fits-all.

8.3 Limitations

Allopathic drugs can produce adverse effects (gastrointestinal issues with cholinesterase inhibitors; hallucinations/agitation with antipsychotics, ARIA with antibodies). They are also costly and not curative. *Ayurvedic* remedies, on the other hand, suffer from a lack of standardisation (herb quality, dosage) and limited rigorous clinical validation. The onset of action may be slow, requiring patient commitment to long courses. Herb–drug interactions are a concern when combining therapies. Regulatory oversight of herbal products is weaker than for pharmaceuticals, raising issues of purity and consistency.

Overall, allopathic AD therapy prioritises symptom management via targeted drugs, while *Ayurveda* emphasises overall rejuvenation and disease prevention. These paradigms can be viewed as complementary: combining them could leverage the precision of modern drugs with the holistic support of traditional care.

9. Future Directions

9.1 Integrative Research

Future efforts should explore synergies between conventional drugs and *Ayurvedic* herbs. Preclinical and clinical trials can test whether combining a cholinesterase inhibitor with, say, a *Bacopa* extract yields greater cognitive benefit or fewer side effects. Modern pharmacology (network pharmacology, systems biology) offers tools to unravel multi-target actions of polyherb formulas. There is growing interest in herb–drug co-therapy: For example, antioxidants from *Withania* or Turmeric may counteract oxidative stress induced by amyloid. Developing standardised herbal extracts (with known active constituents) and testing them alongside drugs could open novel adjunctive treatments[35,36].

9.2 Personalised Medicine

Alzheimer's care is moving toward precision medicine. In the future, therapies may be tailored to genetic markers (*e.g.* APOE ε4 status), biomarkers (CSF tau/amyloid levels), and patient phenotype. Similarly, *Ayurvedic* concepts of *Prakriti* (body–mind constitution) could inform personalised regimens ("ayurgenomics"). Combining genetic screening with traditional assessments might allow hybrid stratification of patients. Artificial intelligence could help integrate multi-dimensional data (genomics, metabolomics, lifestyle) to optimise individualised interventions drawing from both medical systems[37].

9.3 AI and Biomarker Integration

Emerging AI methods promise to transform AD diagnosis and treatment. Machine learning applied to neuroimaging (MRI, PET) can detect subtle early brain changes and predict progression with high accuracy. AI-driven analysis of genetic, proteomic, and digital biomarkers (speech patterns, eye movement) could

Table 2. Key *Ayurvedic* interventions in Alzheimer's disease

Sr. No.	Therapy / Herb	Category	Putative Mechanism	Traditional Indication	Level of Evidence
1	*Ashwagandha* (*W. somnifera*)	*Medhya Rasayana*	Antioxidant, anti-amyloid, and neurite regeneration	*Vata–Pitta* balance, memory support	Preclinical models; small human studies
2	*Brahmi* (*B. monnieri*)	*Medhya Rasayana*	↑ acetylcholine, antioxidant, anti-inflammatory	Learning, retention, memory	Animal models, RCTs in mild cognitive impairment
3	*Mandukaparni* (*C. asiatica*)	*Medhya Rasayana*	Neurogenesis, plaque reduction, and anti-inflammatory	Memory enhancement, wound healing	5xFAD mouse studies, limited clinical data
4	*Shankhpushpi* (*C. pluricaulis*)	*Medhya Rasayana*	GABAergic modulation, antioxidant	Cognitive support, anxiety relief	Rodent cognition models
5	*Panchakarma* (*Nasya, Basti, Shirodhara*)	Detoxification	Clears *Ama*, pacifies *Vata* in the head region	Neuroprotection, mental clarity	Traditional use; pilot clinical studies
6	Medicated *Ghrita* (*e.g., Brahmi Ghrita*)	Lipid-based delivery	Improves CNS delivery, nourishes *Majja Dhatu*	Rejuvenation, memory tonic	Textual evidence; emerging pharmacokinetic data

enable preclinical diagnosis and subgrouping. Multi-modal data integration–combining imaging, genomics, fluid biomarkers, and clinical tests–is being accelerated by deep learning. These technologies could also be used to assess responses to herbal and lifestyle interventions, identifying which combinations work best for specific patient profiles[38,39].

9.4 Biomarker Discovery

Advances in noninvasive biomarkers (blood-based assays for phosphorylated tau, neurofilament light, or even retinal scans) will likely be integrated with AI analytics. Such tools could allow monitoring of both Western and *Ayurvedic* treatments through objective measures of neurodegeneration or inflammation. The ultimate goal is to detect AD at its very earliest stages and apply multi-pronged interventions (pharmacological and holistic) before irreversible damage occurs[40].

9.5 Regulatory and Quality Frameworks

As integrative therapies evolve, regulatory oversight must adapt. Herbal medicines need standardised extraction, dosing, and rigorous safety testing – similar to pharmaceuticals. Current efforts are underway to authenticate *Ayurvedic* nootropics and compile scientific evidence for their use. Future legislation may require clear labelling of active constituents and evidence-based claims for cognitive benefits. Global health organisations (*e.g.* WHO Traditional Medicine Strategy) encourage integrating validated traditional treatments into healthcare systems, provided quality and safety are assured. Clinical guidelines will need to address herb–drug interactions and co-prescribing practices.

9.6 Collaboration and Innovation

Finally, addressing Alzheimer's will require collaborative, multidisciplinary research. Combining AI-driven drug discovery with ethnopharmacology may uncover new candidate molecules inspired by *Ayurvedic* herbs. Increased funding for integrative clinical trials – testing herbal formulations against placebo or in combination with standard care–is crucial. Efforts like creating open-access databases of *Ayurvedic* formulations, sharing biomarker data, and engaging both Western clinicians and *Ayurvedic* practitioners will accelerate progress. In summary, the future points toward a convergence of tradition and technology, where AI-guided, personalised interventions draw from the strengths of both modern neurology and ancient wisdom to improve outcomes in AD.

10. Conclusion

Alzheimer's Diseas (AD) continues to be a major public health challenge, characterised by progressive

neurodegeneration and significant personal and societal burden. Modern medicine has made progress through symptom-targeted therapies such as cholinesterase inhibitors and NMDA antagonists, and more recently, monoclonal antibodies aimed at amyloid plaques. However, these treatments provide only modest, temporary relief and are often limited by side effects, high costs, and lack of curative efficacy. In contrast, *Ayurveda* offers a holistic approach rooted in balancing the body's *doshas*, rejuvenating brain tissue (*Medha*), and enhancing vitality (*Ojas*). *Ayurvedic* management of AD focuses on *Rasayana* therapy using memory-enhancing herbs like *Brahmi, Ashwagandha*, and *Shankhpushpi*, supported by *Panchakarma* detoxification and lifestyle modifications, including *yoga*, meditation, and diet. These interventions aim to support the body's innate healing, improve cognitive function, and reduce stress and inflammation through systemic actions. Although the philosophical foundations of modern and *Ayurvedic* medicine differ, both systems recognise the importance of early intervention and lifestyle in preserving cognitive health. There is increasing interest in integrating these approaches. Combining pharmaceutical drugs with *Ayurvedic* herbs may yield synergistic benefits. Herbs with antioxidant, anti-inflammatory, and neuroprotective effects could potentially enhance the efficacy of standard therapies or mitigate their side effects. Future research should focus on validating *Ayurvedic* therapies through rigorous scientific trials, standardising herbal formulations, and understanding their mechanisms through tools like systems biology and network pharmacology. Artificial intelligence and biomarker-based precision medicine may further enable personalised interventions that combine insights from both systems. This convergence of tradition and innovation offers a promising path forward. By integrating modern evidence-based treatments with time-tested *Ayurvedic* principles, a more comprehensive, patient-centred approach to Alzheimer's care can be developed. Such a synthesis may lead to earlier diagnosis, more effective prevention strategies, and improved quality of life for patients and caregivers alike. As the global prevalence of AD rises, collaborative, multidisciplinary research embracing both ancient wisdom and modern science is not just desirable, it is essential. Future clinical studies are needed to validate the findings.

11. Acknowledgement

My profound appreciation goes out to Mahatma Gandhi Ayurved College, Hospital and Research Centre for providing the learning environment, materials, and support required to finish this review paper. I would especially like to express my gratitude to the research guides and faculty members for their invaluable insights and unwavering support during the process. Their advice was very helpful in forming the integrative viewpoint that this work presents.

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Author Contribution

The review's conceptualisation, integration of contemporary and *Ayurvedic* viewpoints, and critical revision of the manuscript for intellectual content were all overseen by Dr. Dhruv Modi. Dr. Devyani Dasar prepared the *Medhya Rasayana* section, helped with referencing, and contributed to the literature review on *Ayurvedic* interventions. Data on the pathophysiology, diagnosis, and contemporary treatment of Alzheimer's disease were gathered and examined by Dr. Naveen Singh. Dr. Krishna Shinde created the comparative tables and helped with formatting and editing the manuscript. All authors have read and agreed to the published version of the manuscript.