International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

Dolna 17, Warsaw,
Poland 00-773
+48 226 0 227 03
editorial_office@rsglobal.pl

ARTICLE TITLE	DEMENTIA – DIFFERENTIATION AND THE SIGNIFICANCE OF EARLY DIAGNOSIS
DOI	https://doi.org/10.31435/ijitss.3(47).2025.3735
RECEIVED	14 July 2025
ACCEPTED	11 September 2025
PUBLISHED	30 September 2025
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

© The author(s) 2025.
This article is published as open access under the Creative Commons Attribution 4.0 International License (CC BY 4.0), allowing the author to retain copyright. The CC BY 4.0 License permits the content to be copied, adapted, displayed, distributed, republished, or reused for any purpose, including adaptation and commercial use, as long as proper attribution is provided.

DEMENTIA – DIFFERENTIATION AND THE SIGNIFICANCE OF EARLY DIAGNOSIS

Joanna Mazurek *(Corresponding Author, Email: joannamazurek26@gmail.com)*
1st Military Clinical Hospital with Polyclinic SPZOZ in Lublin, Poland
ORCID ID: 0009-0005-0300-7798

Wojciech Gąska
Stefan Cardinal Wyszyński Provincial Specialist Hospital SPZOZ in Lublin, Aleja Kraśnicka 100, 20-718 Lublin, Poland
ORCID ID: 0009-0005-7621-3533

Ignacy Rożek
Stefan Cardinal Wyszyński Provincial Specialist Hospital SPZOZ in Lublin, Aleja Kraśnicka 100, 20-718 Lublin, Poland
ORCID ID: 0009-0005-5731-6983

Izabela Lekan
St. John Paul II Provincial Hospital in Siedlce, Mazovia Province, Siedlce, Poland
ORCID ID: 0009-0000-5079-9795

Agnieszka Brzezińska
Stefan Cardinal Wyszyński Provincial Specialist Hospital SPZOZ in Lublin, Aleja Kraśnicka 100, 20-718 Lublin, Poland
ORCID ID: 0000-0001-5730-8813

Weronika Tuszyńska
University Clinical Center of the Medical University of Warsaw, Banacha 1a Street, 02-097 Warsaw, Poland
ORCID ID: 0000-0002-2395-6748

Alicja Sodolska
Ophthalmology s.c. Primary Care Clinic, Mickiewicza Street 28, Lublin, Poland
ORCID ID: 0009-0008-3689-7004

Michał Lenart
University Clinical Hospital No. 1 in Lublin, Poland
ORCID ID: 0009-0006-5103-7251

Barbara Madoń
Medical University of Lublin, Poland
ORCID ID: 0000-0003-1054-6405

Barbara Teresińska
Medical University of Lublin, Poland
ORCID ID: 0000-0002-1101-3566

ABSTRACT

Dementias are a complex group of neurodegenerative diseases characterized by a progressive decline in cognitive functions, significantly affecting patients' daily lives. Differentiating between types of dementia, such as Alzheimer's disease, vascular dementia, Lewy body dementia, and frontotemporal dementia, is essential for appropriate therapy planning, disease prognosis, and optimizing care.

Diagnosis is based on a comprehensive assessment of clinical symptoms, neuroimaging studies, and analysis of fluid biomarkers. Clinical symptoms, such as memory impairment, attention deficits, executive dysfunction, and behavioral changes, are the basis of diagnosis; however, differentiation requires support from brain imaging techniques such as magnetic resonance imaging (MRI) and positron emission tomography (PET), which allow the identification of characteristic structural and metabolic changes. Biomarkers, particularly tau and beta-amyloid proteins measured in cerebrospinal fluid and serum, are important tools for confirming the diagnosis and monitoring disease progression.

Early diagnosis of dementia is crucial for the effectiveness of therapy and improving patients' quality of life. It enables the implementation of interventions that slow disease progression and provides appropriate psychosocial support for patients and their families.

In recent years, artificial intelligence (AI) has played an increasing role in the diagnosis of dementia. Advanced algorithms analyzing clinical, neuroimaging, and biomarker data support more accurate and faster differentiation of dementia types and help identify at-risk individuals at early disease stages. The implementation of AI in clinical practice opens new opportunities for personalized treatment and patient care.

KEYWORDS

Dementia, Alzheimer's Disease, Vascular Dementia, Lewy Body Dementia, Frontotemporal Dementia

CITATION

Joanna Mazurek, Wojciech Gąska, Ignacy Rożek, Izabela Lekan, Agnieszka Brzezińska, Weronika Tuszyńska, Alicja Sodolska, Michał Lenart, Barbara Madoń, Barbara Teresińska. (2025) Dementia – Differentiation and the Significance of Early Diagnosis. *International Journal of Innovative Technologies in Social Science.* 3(47). doi: 10.31435/ijitss.3(47).2025.3735

COPYRIGHT

© **The author(s) 2025.** This article is published as open access under the **Creative Commons Attribution 4.0 International License (CC BY 4.0)**, allowing the author to retain copyright. The CC BY 4.0 License permits the content to be copied, adapted, displayed, distributed, republished, or reused for any purpose, including adaptation and commercial use, as long as proper attribution is provided.

Introduction

Dementia syndromes are one of the neuropsychiatric disease groups with increasing prevalence due to the aging of societies.

According to the ICD-10 definition, dementia is a syndrome involving progressive disturbances of cognitive functions—particularly memory, thinking, orientation, comprehension, learning capacity, language, and judgment. Dementia is an acquired condition caused by brain disease and does not include intellectual disability [1].

In 2020, the number of people suffering from dementia was estimated at 55 million. It is projected that this number will rise to 139 million by 2050. Over 60% of people with dementia currently live in low- and middle-income countries, and this proportion is expected to increase to 71% by 2050 [2]. Currently, dementia is the seventh leading cause of death worldwide and one of the major causes of disability and dependency among older people. According to the World Health Organization, in 2019, global costs associated with dementia were estimated at 1.3 trillion USD, with approximately 50% attributed to informal care provided by family members or close friends, who devote an average of five hours per day to caregiving and supervision [3].

Early diagnosis of dementia allows for the implementation of therapies that slow disease progression, facilitates care planning, supports families, and improves patient quality of life. Despite significant medical advances, a large proportion of cases—especially in the early stages—still remain undiagnosed. The first symptoms may go unnoticed by patients or be ignored and attributed to normal aging. Additionally, some individuals may avoid reporting symptoms due to fear of diagnosis.

Another challenge for physicians is the differential diagnosis of dementia types—such as Alzheimer's disease, vascular dementia, Lewy body dementia, or frontotemporal dementia—due to frequently overlapping clinical symptoms. In clinical practice, screening tools, brain imaging, and biomarkers play a particularly important role. Modern technologies, such as artificial intelligence and blood-based early biomarkers of neurodegeneration, are also gaining significance.

Methodology

This systematic review was based on scientific papers searched on the PubMed platform and in the medical literature in March, April and May 2025. Mainly studies published from 2019 to 2025 were included in the analysis. Articles written in English were used. The studies were screened based on the title and abstract and then selected for full- text review by the first author.

Discussion

Classification and Etiologies of Dementias

Dementia is a syndrome characterized by impairments in higher cognitive functions such as memory, thinking, orientation, calculation, learning capacity, language, and judgment. Emotional control, impulse regulation, behavior, and motivation can also be affected, while consciousness remains intact. These symptoms severely impact daily functioning, impairing even basic activities such as personal hygiene, dressing, and eating. Dementias may result from various conditions with different pathophysiological mechanisms. Depending on the cause, the location of damage in the central nervous system, and the disease progression, several major types of dementia syndromes can be distinguished. They are broadly classified as primary or secondary dementias.

Primary dementias arise from conditions that directly affect brain tissue. These include Alzheimer's disease, Lewy body dementia, and frontotemporal dementia. Secondary dementias occur as a consequence of systemic illnesses affecting the brain as one of many organs or result from head trauma. This group includes post-stroke dementia, vascular dementia, Creutzfeldt–Jakob disease, and long-term consequences of traumatic brain injuries [4]. Proper classification of dementia type is crucial for determining the appropriate diagnostic and therapeutic approach.

Alzheimer's Disease

Alzheimer's disease (AD) accounts for 60% to 80% of all dementia cases. Key pathological features of AD include the accumulation of beta-amyloid plaques in brain tissue and blood vessels and the presence of neurofibrillary tangles composed of hyperphosphorylated tau protein within neurons. Additionally, neuron and synapse loss, as well as gliosis, are typical [5].

The exact mechanisms underlying the disease remain incompletely understood. Vascular abnormalities, mitochondrial dysfunction, oxidative stress, reduced glucose utilization in the brain, and neuroinflammation are considered significant contributors. Mitochondrial dysfunctions interact complexly with other pathological processes. These dysfunctions may be caused by environmental toxins, metabolic disorders, defects in oxidative phosphorylation (OXPHOS), or mutations in mitochondrial DNA (mtDNA).

Consequences include reduced ATP production, excessive reactive oxygen species (ROS) generation, impaired mitophagy, accumulation of defective organelles, impaired mitochondrial transport and fusion/fission balance, disruptions in protein and metabolite transport, and calcium dysregulation. These changes exacerbate energy deficits, oxidative stress, calcium imbalance, abnormal protein deposition, and excitotoxicity—leading to loss of mitochondrial membrane potential, cytochrome c release, neuronal death, and cognitive decline. These processes are interlinked and mutually reinforcing, contributing to Alzheimer's disease progression [6].

There are two forms of Alzheimer's disease: early-onset (before age 65) and late-onset. A small proportion of AD cases are genetic, associated with autosomal dominant mutations in the APP, PSEN1, and PSEN2 genes—typically resulting in early-onset AD. However, the majority of patients develop sporadic late-onset Alzheimer's disease (LOAD), which occurs in older age. While not inherited in a traditional sense, certain genetic factors increase susceptibility—most notably the presence of the APOE ε4 allele, which occurs in about 16% of the population [7].

Lewy Body Dementia

Lewy body dementia (LBD) is considered the second most common cause of dementia, accounting for approximately 7% to 34% of cases [4]. The hallmark pathology is the accumulation of misfolded α-synuclein protein in the form of Lewy bodies and Lewy neurites in neurons and glial cells. Under normal conditions, α-synuclein is found in presynaptic nerve terminals, where it regulates synaptic vesicle trafficking. However, in pathological states, it undergoes phosphorylation, nitration, truncation, and ubiquitination—forming toxic aggregates [8]. Structurally, α-synuclein has three domains: an N-terminal region responsible for tetramerization, a central aggregation-prone region, and a proline-rich acidic C-terminal region [9].

The accumulation of α-synuclein leads to the loss of cholinergic neurons—causing memory and learning impairments—and dopaminergic neurons—resulting in motor, mood, sleep, behavioral, and cognitive disturbances. LBD often coexists with other neurodegenerative pathologies, including β-amyloid and tau deposition, which may worsen clinical outcomes.

In Parkinson's disease dementia, cortical α-synuclein plays a primary role, while in LBD, β-amyloid is also significant. LBD shares genetic links with both Parkinson's and Alzheimer's diseases. Mutations in SNCA and LRRK2 can lead to various forms of dementia or parkinsonism, and the APOE ε4 allele increases LBD risk (though to a lesser extent than in AD), whereas ε2 may have a protective effect. Neuropathologically, LBD is distinguished by widespread cortical Lewy bodies, unlike AD.

Clinically, differentiating LBD from AD can be challenging. However, LBD is more likely to present with fluctuating cognition, visual hallucinations, parkinsonism, and REM sleep behavior disorder. Despite advanced α-synuclein pathology, some individuals do not exhibit clinical symptoms, suggesting that Lewy bodies may not be inherently toxic—or could even serve a protective function. The molecular mechanisms of LBD are still poorly understood, and current cellular models fail to fully replicate mature Lewy body formation. Vascular pathology does not appear to play a major role in LBD development, although it often coexists with Alzheimer-type changes [10].

Frontotemporal Dementia

Frontotemporal dementia (FTD) is a neurodegenerative disorder involving the degeneration of the frontal and/or temporal cortical regions. It leads to behavioral changes, speech difficulties, and psychiatric symptoms. FTD includes various clinical subtypes: the behavioral variant (bvFTD), the semantic variant of primary progressive aphasia (svPPA), the non-fluent variant (nfvPPA), the right temporal variant (rtvFTD), and FTD associated with motor neuron disease (FTD-MND).

Related conditions also include tauopathies such as corticobasal syndrome (CBS) and progressive supranuclear palsy (PSP), which can affect frontal lobe functions [11].

FTD exhibits high genetic heterogeneity. Around 40% of patients have a family history of dementia, psychiatric disorders, or motor symptoms, and 10% display autosomal dominant inheritance. The most common genetic mutations occur in C9orf72, MAPT, and GRN, accounting for approximately 30% of hereditary FTD cases. TBK1 is now recognized as the fourth most frequent mutation, while others collectively represent less than 5%. Inheritance patterns vary by subtype, with bvFTD and FTD-ALS showing the strongest genetic associations, and the semantic variant (svPPA) showing the weakest. Pathologically, FTD is characterized by abnormal protein folding and the formation of toxic aggregates in neurons and glial cells. Commonly involved proteins include TDP-43 (especially types A and B), tau, and FUS. Neurodegeneration progresses in a prion-like manner, spreading from cell to cell. Genetic mutations disrupt protein homeostasis, impairing organelles like lysosomes, mitochondria, and the endoplasmic reticulum, as well as processes such as autophagy, RNA transport, and inter-organelle signaling. These disruptions lead to the accumulation of pathological proteins, increased cell vulnerability, and eventual neuronal death [12].

Vascular Dementia

Vascular dementias are a diverse group of brain disorders caused by cerebrovascular pathologies. Four primary types of vascular changes leading to dementia have been identified: post-stroke dementia, multi-infarct dementia, mixed dementia, and subcortical ischemic vascular dementia [13]. Vascular dementia (VaD) develops due to several key processes: restricted blood flow and brain hypoxia, blood–brain barrier disruption, impaired cerebrospinal fluid outflow, and vascular inflammation. Each of these factors can lead to changes in the brain. Prolonged oxygen deprivation, particularly in the white matter, damages neurons and weakens memory and other cognitive functions. Disruption of the blood–brain barrier promotes inflammation and degeneration of neural tissue. When the outflow of cerebrospinal fluid is impaired, toxic substances are not effectively removed, leading to the accumulation of proteins and further damage. Vascular inflammation, in turn, impairs cerebral circulation and accelerates disease progression [14].

In addition to the main brain-related mechanisms, systemic risk factors play a crucial role in the development and exacerbation of vascular dementia. These include hypertension, elevated cholesterol levels, obesity, and diabetes. Such conditions promote systemic inflammation by increasing cytokine production, which can damage the blood–brain barrier, activate the brain's immune cells (microglia), and intensify inflammation in the central nervous system [15].

Differential Diagnosis

Proper diagnosis is essential for selecting appropriate treatment, determining prognosis, and providing support for both the patient and their family. This section outlines current diagnostic approaches to distinguish among the major types of dementia. Accurate differential diagnosis requires a detailed patient history, including age, biological sex, medical background, and current symptoms. It also necessitates cognitive assessments, neuroimaging, and laboratory testing. It is important to consider both neurodegenerative diseases and reversible conditions such as depression, delirium, vitamin deficiencies, thyroid disorders, or side effects of medications [16].

Clinical Symptoms

Dementia presents a wide range of neuropsychiatric and cognitive symptoms. A detailed analysis of clinical manifestations should be one of the first steps in diagnosing the disease. Below are the characteristic symptoms of the most common forms of dementia:

Alzheimer's Disease

Alzheimer's disease is marked by a broad range of clinical symptoms that progress gradually. Its development can be divided into three stages: asymptomatic, prodromal (often referred to as mild cognitive impairment), and dementia stage [17]. The earliest symptoms include amnestic cognitive impairments and short-term memory difficulties. As Alzheimer's progresses, cognitive deficits become increasingly complex. Patients may experience problems with executive functions such as maintaining concentration, planning, and problem-solving. Additional difficulties include expressive language issues and visuospatial disorientation. These changes significantly affect the patient's daily functioning and independence.

In addition to cognitive symptoms, Alzheimer's often involves neuropsychiatric issues. Early signs include low mood, chronic anxiety, apathy, and lack of motivation, which can be mistaken for depression or emotional exhaustion. In more advanced stages, more serious behavioral and perceptual disturbances may occur, including delusions, hallucinations, aggression, irritability, and emotional instability [5].

Dementia with Lewy Bodies

Patients with dementia with Lewy bodies experience neuropsychiatric symptoms such as visual and sensory hallucinations, systematic delusions, apathy, aggression, anxiety, and depression. Often, patients lack insight into their symptoms, making caregiver reports essential. Other distinctive neuropsychiatric features include cognitive fluctuations (i.e., varying severity of symptoms throughout the day), sleep disturbances, restlessness, aggressive behavior, or suspiciousness. Common sleep problems include both insomnia and excessive daytime sleepiness, as well as REM sleep behavior disorder—where patients have vivid dreams and physically act them out, which can be dangerous. Motor symptoms typical of this type of dementia include bradykinesia, muscle rigidity, postural instability, gait disturbances, and falls. Numerous autonomic dysfunctions also occur, such as orthostatic hypotension. Gastrointestinal symptoms may include drooling, dysphagia, gastroparesis, and constipation. Other possible symptoms include urinary urgency, frequency, incontinence, and excessive sweating [18].

Frontotemporal Dementia (FTD)

Frontotemporal dementia is a disease with a varied clinical presentation. FTD is often mistaken for Alzheimer's, especially in its early stages. While memory impairments dominate in Alzheimer's, FTD primarily involves behavioral or language changes, with memory relatively preserved. FTD can also resemble dementia with Lewy bodies due to fluctuations in cognition, hallucinations, and parkinsonism. Because of behavioral changes—such as emotional indifference, loss of empathy, or impulsiveness—many FTD patients initially see a psychiatrist and may be misdiagnosed with depression, schizophrenia, or bipolar disorder. Symptoms may mimic depression but typically lack characteristic sadness.

FTD symptoms can also indicate the specific subtype. In the behavioral variant (bvFTD), suspicion is raised when at least three of the following are present: early impulsiveness, lack of empathy, apathy, stereotypical behaviors, appetite changes, and difficulties with planning and organizing—despite preserved memory. In the language variants of FTD—so-called primary progressive aphasias (PPA)—speech impairment predominates. In nfvPPA (non-fluent variant), patients struggle with sentence construction; their speech is slow and effortful, and they have trouble understanding complex syntax, though they understand individual words. In svPPA (semantic variant), typical symptoms include difficulty naming objects, failure to understand word meanings, loss of factual knowledge, while grammar and fluency are preserved [19].

Vascular Dementia

The symptoms of vascular dementia depend on the location and type of cerebrovascular damage. Subcortical dementia develops gradually and is characterized by progressive executive dysfunction—difficulties with planning, organizing, decision-making, and a general slowing of thought processes. Patients often struggle with maintaining attention and processing information, while episodic memory may remain relatively preserved in the early stages. Mobility issues may also occur, and some patients develop Parkinsonian symptoms such as muscle stiffness, bradykinesia, and postural instability.

In post-stroke dementia, symptoms appear directly after a stroke or within six months of it and tend to be permanent. The nature of symptoms depends on the stroke's location and may include language impairments, memory problems, attentional deficits, or spatial disorientation. These are often accompanied by focal neurological signs, such as paresis, hemiplegia, and visual or sensory disturbances on one side of the body.

In multi-infarct dementia, symptoms develop in a stepwise fashion, worsening after each subsequent vascular event. Cortical symptoms are typical, such as aphasia, apraxia, agnosia, visual field deficits, and spatial neglect.

Mixed dementia commonly combines features of vascular dementia with Alzheimer's disease. Clinically, prominent memory disturbances are observed—especially difficulties with learning new information and rapid forgetting—but symptoms typical of vascular damage may also be present, such as psychomotor slowing, attention deficits, and impaired executive functions [20].

Screening Tools and Neuropsychological Tests

Screening tools and neuropsychological tests play a crucial role in the diagnosis of dementias, allowing for early detection of cognitive impairment and differentiation between dementia types. Among the most commonly used screening tools are the Mini-Mental State Examination (MMSE) and the Montreal Cognitive Assessment (MoCA).

The Mini-Mental State Examination (MMSE) is a brief neuropsychological tool used to assess overall cognitive function. In individuals with mild cognitive impairment, this test is often supplemented with more detailed assessments, which may evaluate language, praxis, and executive functioning. The MMSE is simple and quick to administer, carries no risk for the patient, and is widely respected among professionals involved in dementia diagnosis and care [21].

The Montreal Cognitive Assessment (MoCA) is a short cognitive test lasting about 10 minutes. It evaluates short-term memory, visuospatial abilities, executive functions, attention, concentration, working memory, language, and orientation. Compared to the MMSE, MoCA provides a more detailed assessment of executive functioning. Recently, MoCA has been considered a non-specialist alternative to the MMSE, especially since the MMSE is now copyright-protected and subject to fees. However, MoCA is a more challenging test, and its scores are not directly equivalent to MMSE results [22].

Studies have shown that sociodemographic variables such as age, gender, education, literacy, and language significantly influence MMSE and MoCA scores. These factors may interact to affect test outcomes and the rate of cognitive decline, complicating score interpretation in diverse populations [23].

Neuropsychological tests offer more advanced diagnostic capabilities and are used to evaluate cognitive functioning in patients suspected of having dementia. Unlike screening tools, which provide a general overview of cognitive status, neuropsychological assessments allow for detailed analysis of specific brain functions and their impairments. These tests assess various domains, including short- and long-term memory, attention and concentration, language abilities (comprehension and expression), executive functions (planning, cognitive control, mental flexibility), visuospatial skills, processing speed, and psychomotor abilities. Tests are chosen individually based on the patient's symptoms and diagnostic goals.

Examples of commonly used neuropsychological tools include the Wechsler Memory Scale (WMS), Clock Drawing Test (CDT), Verbal Fluency Test, and Wisconsin Card Sorting Test (WCST). These results complement clinical evaluation and brain imaging and are useful for monitoring disease progression and therapy effectiveness [24].

The Wechsler Memory Scale – Fourth Edition (WMS-IV) version for older adults includes four primary indices assessing different aspects of memory: auditory, visual, immediate, and delayed. Each index is based on specific subtest results. The full test includes seven tasks measuring abilities such as recalling stories, remembering word pairs, reproducing patterns, and remembering symbol placements. This structure enables a detailed evaluation of both short- and long-term memory in verbal and visual forms. Importantly, the test does

not require reading skills, making it suitable for illiterate individuals. Scores are standardized, allowing for comparison with population norms, with higher scores indicating better memory function [25].

The Clock Drawing Test (CDT) is a multidimensional cognitive assessment tool that analyzes executive functions, planning, visuospatial abilities, memory, and attention. The digital Clock Drawing Test (dCDT) allows for more detailed cognitive evaluation by recording precise parameters [26]. A study by Schejter-Margalit and colleagues demonstrated that the digital version, based on quantitative analysis, more effectively detected early, subtle cognitive changes in patients with Parkinson's disease than traditional diagnostic tools [27]. Similarly, research by Li et al. confirmed that the digital clock mapping test can accurately identify cognitive impairment in individuals with Alzheimer's-related mild cognitive impairment and is effective as an early detection tool [28].

The verbal fluency test is a widely used neuropsychological tool for assessing various aspects of cognitive functioning, including semantic memory, language functions, and executive processes such as initiation and organization of search strategies. There are two basic types of this test: semantic fluency, which involves generating names of items belonging to a specific category (e.g., animals), and phonemic fluency, which requires producing words starting with a given letter. Studies have shown that individuals with Alzheimer's disease and amnestic mild cognitive impairment (a-MCI) achieve significantly lower scores. In contrast, elderly individuals with normal cognitive aging typically perform within the normal range, suggesting that verbal fluency can be a sensitive marker distinguishing neurodegenerative processes from physiological aging [29].

The Wisconsin Card Sorting Test is a classic neuropsychological tool used to assess executive functions such as abstract thinking, cognitive flexibility, and the ability to shift strategies in response to feedback. The task involves sorting cards according to undisclosed rules that change during the test. Key indicators, such as the number of perseverative errors and set loss, allow detailed assessment of adaptive abilities and cognitive control of the patient. The test is widely used in the diagnosis of frontal lobe dysfunctions, e.g., in Parkinson's disease, dementia, or schizophrenia [30].

Imaging Diagnostics and Biomarkers

Brain imaging (neuroimaging) is an essential part of diagnosing neurodegenerative diseases and is routinely used in clinical practice. Magnetic Resonance Imaging (MRI) allows assessment of brain structure, analysis of progressive atrophy, and identification of functional changes. Positron Emission Tomography (PET) measures brain metabolic activity using radioactive tracers. Computed Tomography (CT) uses X-ray radiation to generate cross-sectional images of the brain. Electroencephalography (EEG) records the variability of the brain's bioelectrical activity in real time [31].

Alzheimer's Disease

The most common neuroimaging finding in Alzheimer's disease patients is cortical atrophy resulting from neuronal cell death. These changes typically occur symmetrically on both sides of the brain, especially in the parietal and temporal lobes. In late-onset Alzheimer's and carriers of the APOE E4 gene variant, hippocampal atrophy is particularly evident, with relative preservation of central brain structures [32]. Patients with atypical forms of Alzheimer's—such as early-onset or without the APOE E4 variant—often show little or no hippocampal atrophy. Instead, they exhibit pronounced cortical atrophy in posterior regions, such as the precuneus, typical for early disease stages. In these patients, asymmetrical damage to parieto-occipital and posterior temporal areas, mostly on the right side, leads to early visual disturbances or spatial disorientation, which precede marked cognitive decline [33].

Fluorodeoxyglucose-PET (FDG-PET) assesses brain metabolism and indicates hypometabolic areas characteristic of different Alzheimer's phenotypes. In the typical form, changes affect the parietal and temporal lobes symmetrically; in posterior cortical atrophy, the occipital regions; and in the logopenic variant of primary progressive aphasia, the left temporoparietal region. This imaging helps differentiate Alzheimer's from other dementias, such as Lewy body dementia. PET imaging with amyloid-beta and tau tracers is also used. Amyloid imaging shows deposits regardless of clinical phenotype, whereas tau imaging reflects symptom localization—for example, temporal lobe in amnestic form, occipital lobe in posterior cortical atrophy, and left frontoparietal area in logopenic aphasia. The extent of tau ligand uptake may also indicate disease progression speed.

MRI assesses grey matter atrophy. In typical Alzheimer's, changes begin in the hippocampus, while atypical forms correspond to symptom location and may not involve hippocampus early on. MRI detects changes later than FDG-PET but correlates well with memory test results. Cerebrospinal fluid (CSF)

biomarkers enable early detection of Alzheimer's pathology. Reduced amyloid beta 1-42 to 1-40 ratio and elevated phosphorylated tau levels are characteristic. Neurofilament light chain is increasingly replacing total tau as a neurodegeneration marker. Other biomarkers like glial acidic protein, YKL-40, and neurogranin have diagnostic potential but are not routinely used yet. Blood biomarkers are gaining importance; plasma amyloid beta ratios and phosphorylated tau (181, 217, 231) correlate with CSF and PET results. Blood glial acidic protein may predict disease progression and help differentiate from frontotemporal dementia [34].

Lewy Body Dementia (DLB)

In diagnosing Lewy body dementia, despite the lack of direct pathological markers, several indirect imaging and neurophysiological tests are used. SPECT or PET show reduced dopamine transporter (DAT) uptake in basal ganglia, enabling differentiation of DLB from Alzheimer's with high sensitivity and specificity. Myocardial scintigraphy with ^{123}I-MIBG shows reduced cardiac sympathetic activity characteristic of DLB. Polysomnography typically reveals loss of muscle atonia during REM sleep (REM sleep without atonia), a strong marker of synucleinopathy. MRI/CT shows relative preservation of medial temporal structures (especially hippocampus), unlike the significant atrophy seen in Alzheimer's. FDG-PET reveals occipital hypometabolism and the"cingulate island sign"—relative preservation of metabolism in the posterior cingulate gyrus. EEG may show dominant pre-alpha slow waves with periodic fluctuations correlating with cognitive symptoms and supporting diagnosis [35].

Frontotemporal Dementia (FTD)

In behavioral variant frontotemporal dementia (bvFTD), neuroimaging changes—atrophy on MRI or hypometabolism on PET—are required to establish a"probable" diagnosis. Early-stage imaging may still appear normal. FDG-PET is often more sensitive than MRI and helps differentiate FTD (hypometabolism in frontal lobes, anterior cingulate, and anterior temporal lobes) from Alzheimer's (changes mainly in temporoparietal lobes and posterior cingulate). The semantic variant of primary progressive aphasia (svPPA) shows atrophy of temporal poles; the nonfluent variant (nfvPPA) has atrophy in the left perisylvian region. Progressive supranuclear palsy (PSP) shows midbrain atrophy; corticobasal degeneration syndrome (CBS) affects areas around the central sulcus.

CSF biomarkers are inconclusive in FTD but tau/amyloid-beta ratios may help differentiate FTD from Alzheimer's (sensitivity 79%, specificity 97%) [36].

Vascular Dementia

In vascular dementia, imaging—especially MRI—plays a key diagnostic role. Typical findings include diffuse hyperintensities in periventricular and subcortical white matter, which may be punctate or confluent. Lacunar infarcts in white matter, basal ganglia, and thalamus are common, as are cortical-subcortical ischemic or traumatic changes. Enlarged Virchow-Robin spaces and microbleeds, particularly visible in susceptibility-weighted imaging (SWI), are additional features. Cerebral amyloid angiopathy shows microbleeds at the gray-white matter junction, superficial siderosis (especially fronto-occipital), acute intracerebral hemorrhages, and secondary parenchymal loss seen on CT and MRI. CADASIL, a hereditary cerebral arteriopathy, reveals extensive symmetrical white matter hyperintensities, especially in anterior temporal lobes and insula. Lacunar infarcts and hemorrhages often coexist, mimicking other vascular dementias, but localization and genetic background are diagnostically characteristic [33].

Artificial Intelligence

Despite widespread use of neuroimaging techniques, their interpretation carries risk of error due to data complexity, multidimensionality, and subjective clinical assessment. Although visual rating scales, such as medial temporal lobe atrophy or white matter lesion severity scales, provide valuable information, they cannot capture all significant image features. For example, resting-state functional MRI (fMRI) generates hundreds of parameters describing functional connectivity among thousands of brain regions—data that can be effectively analyzed using machine learning methods [37].

In this context, deep learning—a sophisticated form of artificial intelligence—shows higher accuracy than traditional clinical analysis in interpreting complex neuroimaging data. AI algorithms enable automation of image analysis, potentially reducing human error and improving clinical decision-making accuracy [38].

Although an increasing number of studies confirm machine learning's effectiveness in identifying imaging features associated with cognitive disorder diagnosis or dementia risk, a clear gap remains between research findings and their implementation in routine clinical practice. Uncertainty persists about how AI algorithms should interact with physician decisions and what role they should play as supportive diagnostic tools.

Conclusions

Dementias are a heterogeneous group of neurodegenerative diseases leading to progressive cognitive decline, behavioral disturbances, and difficulties in daily functioning. Differentiating among dementia types—such as Alzheimer's disease, vascular dementia, Lewy body dementia, and frontotemporal dementia—is crucial as it determines therapeutic strategy, prognosis, and care approach.

Diagnosis relies on three main pillars: clinical symptoms, neuroimaging, and biomarker analysis. The clinical picture involves assessment of memory, executive functions, speech, orientation, perception, and behavior. Modern neuroimaging techniques—MRI, PET, and CT—enable detection of characteristic anatomical and metabolic brain changes. Fluid biomarkers, especially tau and beta-amyloid proteins in cerebrospinal fluid and serum, allow earlier and more precise dementia differentiation.

Early diagnosis is essential—it not only allows symptomatic and non-pharmacological treatment but also facilitates better care planning and emotional/social support for patients and their families.

In this context, artificial intelligence is gaining importance by increasingly supporting diagnostic processes. Machine learning algorithms analyze multidimensional data—including brain images, biomarker profiles, and clinical information—enhancing diagnostic accuracy and enabling identification of individuals at early, often preclinical stages of disease.

Incorporating AI into dementia diagnostics, alongside advances in biomarkers and neuroimaging, significantly improves quality and effectiveness of care for people with cognitive disorders.

Disclosures

Authors do not report any disclosures.

Author's contribution

Conceptualisation: Joanna Mazurek, Wojciech Gąska; methodology: Ignacy Rożek, Izabela Lekan; software: Agnieszka Brzezińska, Weronika Tuszyńska; check: Alicja Sodolska, Michał Lenart; formal analysis: Barbara Madoń, Barbara Teresińska; investigation: Ignacy Rożek Izabela Lekan; resources: Weronika Tuszyńska, Agnieszka Brzezińska; data curation: Alicja Sodolska Michał Lenart; writing-rough preparation: Joanna Mazurek, Wojciech Gąska; writing-review and editing: Barbara Madoń, Barbara Teresińska; visualisation: Michał Lenart; supervision: Joanna Mazurek; project administration:Joanna Mazurek, Wojciech Gąska

All authors have read and agreed with the published version of the manuscript.

Funding Statement: This Research received no external funding.
Institutional Review Board Statement: Not applicable.
Informed Consent Statement: Not applicable.
Data Availability Statement: Not applicable.
Conflicts of Interests: The authors declare no conflict of interest.

REFERENCES

1. World Health Organization. (2016). International statistical classification of diseases and related health problems: 10th revision (ICD-10). World Health Organization.
2. Alzheimer's Disease International. (n.d.). *Numbers of people with dementia worldwide*. https://www.alzint.org/resource/numbers-of-people-with-dementia-worldwide/ (accessed May 2, 2025)
3. World Health Organization. (n.d.). *Dementia fact sheet*. https://www.who.int/news-room/fact-sheets/detail/dementia (accessed May 2, 2025)
4. Gałecki, J. (Ed.). (2021). *Psychiatria*. PZWL.
5. Zheng, Q., & Wang, X. (2025). Alzheimer's disease: Insights into pathology, molecular mechanisms, and therapy. *Protein & Cell, 16*(2), 83–120. https://doi.org/10.1093/procel/pwae026
6. Sharma, C., Kim, S., Nam, Y., Jung, U. J., & Kim, S. R. (2021). Mitochondrial dysfunction as a driver of cognitive impairment in Alzheimer's disease. *International Journal of Molecular Sciences, 22*(9), 4850. https://doi.org/10.3390/ijms22094850
7. Rostagno, A. A. (2022). Pathogenesis of Alzheimer's disease. *International Journal of Molecular Sciences, 24*(1), 107. https://doi.org/10.3390/ijms24010107
8. Meade, R. M., Fairlie, D. P., & Mason, J. M. (2019). Alpha-synuclein structure and Parkinson's disease – lessons and emerging principles. *Molecular Neurodegeneration, 14*, 29. https://doi.org/10.1186/s13024-019-0329-1

9. Mehra, S., Gadhe, L., Bera, R., Sawner, A. S., & Maji, S. K. (2021). Structural and functional insights into α-synuclein fibril polymorphism. *Biomolecules, 11*(10), 1419. https://doi.org/10.3390/biom11101419
10. Prasad, S., Katta, M. R., Abhishek, S., et al. (2023). Recent advances in Lewy body dementia: A comprehensive review. *Disease-a-Month, 69*(5), 101441. https://doi.org/10.1016/j.disamonth.2022.101441
11. Olney, N. T., Spina, S., & Miller, B. L. (2017). Frontotemporal dementia. *Neurologic Clinics, 35*(2), 339–374. https://doi.org/10.1016/j.ncl.2017.01.008
12. Antonioni, A., Raho, E. M., Lopriore, P., et al. (2023). Frontotemporal dementia, where do we stand? A narrative review. *International Journal of Molecular Sciences, 24*(14), 11732. https://doi.org/10.3390/ijms241411732
13. Skrobot, O. A., O'Brien, J., Black, S., et al.; VICCCS group; Ben-Shlomo, Y., Passmore, A. P., Love, S., & Kehoe, P. G. (2017). The vascular impairment of cognition classification consensus study. *Alzheimer's & Dementia, 13*(6), 624–633. https://doi.org/10.1016/j.jalz.2016.10.007
14. Altahrawi, A. Y., James, A. W., & Shah, Z. A. (2025). The role of oxidative stress and inflammation in the pathogenesis and treatment of vascular dementia. *Cells, 14*(8), 609. https://doi.org/10.3390/cells14080609
15. Lecordier, S., Manrique-Castano, D., El Moghrabi, Y., & ElAli, A. (2021). Neurovascular alterations in vascular dementia: Emphasis on risk factors. *Frontiers in Aging Neuroscience, 13*, 727590. https://doi.org/10.3389/fnagi.2021.727590
16. Niotis, K., Akiyoshi, K., Carlton, C., & Isaacson, R. (2022). Dementia prevention in clinical practice. *Seminars in Neurology, 42*(5), 525–548. https://doi.org/10.1055/s-0042-1759580
17. Jack, C. R. Jr., Knopman, D. S., Jagust, W. J., et al. (2010). Hypothetical model of dynamic biomarkers of the Alzheimer's pathological cascade. *The Lancet Neurology, 9*(1), 119–128. https://doi.org/10.1016/S1474-4422(09)70299-6
18. Taylor, J. P., McKeith, I. G., Burn, D. J., et al. (2020). New evidence on the management of Lewy body dementia. *The Lancet Neurology, 19*(2), 157–169. https://doi.org/10.1016/S1474-4422(19)30153-X
19. Puppala, G. K., Gorthi, S. P., Chandran, V., & Gundabolu, G. (2021). Frontotemporal dementia – current concepts. *Neurology India, 69*(5), 1144–1152. https://doi.org/10.4103/0028-3886.329593
20. Chang Wong, E., & Chang Chui, H. (2022). Vascular cognitive impairment and dementia. *Continuum (Minneap Minn), 28*(3), 750–780. https://doi.org/10.1212/CON.0000000000001124
21. Arevalo-Rodriguez, I., Smailagic, N., Roqué-Figuls, M., et al. (2021). Mini-Mental State Examination (MMSE) for the early detection of dementia in people with mild cognitive impairment (MCI). *Cochrane Database of Systematic Reviews, 7*(7), CD010783. https://doi.org/10.1002/14651858.CD010783.pub3
22. Davis, D. H., Creavin, S. T., Yip, J. L., Noel-Storr, A. H., Brayne, C., & Cullum, S. (2021). Montreal Cognitive Assessment for the detection of dementia. *Cochrane Database of Systematic Reviews, 7*(7), CD010775. https://doi.org/10.1002/14651858.CD010775.pub3
23. Tsoy, E., Sideman, A. B., Piña Escudero, S. D., et al. (2021). Global perspectives on brief cognitive assessments for dementia diagnosis. *Journal of Alzheimer's Disease, 82*(3), 1001–1013. https://doi.org/10.3233/JAD-201403
24. Maeshima, S., Osawa, A., Kawamura, K., et al. (2024). Neuropsychological tests used for dementia assessment in Japan: Current status. *Geriatrics & Gerontology International, 24*(Suppl 1), 102–109. https://doi.org/10.1111/ggi.14678
25. Lee, S. C., Chien, T. H., Chu, C. P., Lee, Y., & Chiu, E. C. (2023). Practice effect and test-retest reliability of the Wechsler Memory Scale-Fourth Edition in people with dementia. *BMC Geriatrics, 23*(1), 209. https://doi.org/10.1186/s12877-023-03913-2
26. Wang, C., Li, K., Huang, S., et al. (2025). Differential cognitive functioning in the digital clock drawing test in AD-MCI and PD-MCI populations. *Frontiers in Neuroscience, 19*, 1558448. https://doi.org/10.3389/fnins.2025.1558448
27. Schejter-Margalit, T., Kizony, R., Shirvan, J., et al. (2021). Quantitative digital clock drawing test as a sensitive tool to detect subtle cognitive impairments in early stage Parkinson's disease. *Parkinsonism & Related Disorders, 90*, 84–89. https://doi.org/10.1016/j.parkreldis.2021.08.002
28. Li, R. X., Ma, Y. H., Tan, L., & Yu, J. T. (2022). Prospective biomarkers of Alzheimer's disease: A systematic review and meta-analysis. *Ageing Research Reviews, 81*, 101699. https://doi.org/10.1016/j.arr.2022.101699
29. McDonnell, M., Dill, L., Panos, S., et al. (2020). Verbal fluency as a screening tool for mild cognitive impairment. *International Psychogeriatrics, 32*(9), 1055–1062. https://doi.org/10.1017/S1041610219000644
30. Kopp, B., Lange, F., & Steinke, A. (2021). The reliability of the Wisconsin Card Sorting Test in clinical practice. *Assessment, 28*(1), 248–263. https://doi.org/10.1177/1073191119866257
31. Aljuhani, M., Ashraf, A., & Edison, P. (2024). Use of artificial intelligence in imaging dementia. *Cells, 13*(23), 1965. https://doi.org/10.3390/cells13231965
32. Frisoni, G. B., Bocchetta, M., Chételat, G., et al.; ISTAART's NeuroImaging Professional Interest Area. (2013). Imaging markers for Alzheimer disease: Which vs how. *Neurology, 81*(5), 487–500. https://doi.org/10.1212/WNL.0b013e31829d86e8
33. Furtner, J., & Prayer, D. (2021). Neuroimaging in dementia. *Wiener Medizinische Wochenschrift, 171*(11–12), 274–281. https://doi.org/10.1007/s10354-021-00825-x

34. Dubois, B., von Arnim, C. A. F., Burnie, N., Bozeat, S., & Cummings, J. (2023). Biomarkers in Alzheimer's disease: Role in early and differential diagnosis and recognition of atypical variants. *Alzheimer's Research & Therapy, 15*(1), 175. https://doi.org/10.1186/s13195-023-01314-6
35. McKeith, I. G., Boeve, B. F., Dickson, D. W., et al. (2017). Diagnosis and management of dementia with Lewy bodies: Fourth consensus report of the DLB Consortium. *Neurology, 89*(1), 88–100. https://doi.org/10.1212/WNL.0000000000004058
36. Pressman, P. S., & Miller, B. L. (2014). Diagnosis and management of behavioral variant frontotemporal dementia. *Biological Psychiatry, 75*(7), 574–581. https://doi.org/10.1016/j.biopsych.2013.11.006
37. Aljuhani, M., Ashraf, A., & Edison, P. (2024). Use of artificial intelligence in imaging dementia. *Cells, 13*(23), 1965. https://doi.org/10.3390/cells13231965
38. Avberšek, L. K., & Repovš, G. (2022). Deep learning in neuroimaging data analysis: Applications, challenges, and solutions. *Frontiers in Neuroimaging, 1*, 981642. https://doi.org/10.3389/fnimg.2022.981642