

# Neuroimaging of Dementia

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## KEYWORDS

- Magnetic resonance imaging (MRI) • Alzheimer disease (AD)
- Positron emission tomography (PET) • Frontotemporal lobar degeneration (FTLD)
- Lewy body dementia (LBD) • Prion disease • Multiple sclerosis (MS)
- Vascular cognitive impairment

## KEY POINTS

- Routine use of structural neuroimaging with computed tomography (CT) or MRI is recommended in the evaluation of patients with dementia.
- The latest criteria for the diagnosis of AD incorporate imaging biomarkers to support a clinical diagnosis. Imaging biomarkers for AD include PET and volumetric MRI.
- Florbetapir, an amyloid-binding PET tracer, was recently approved by the US Food and Drug Administration for use in the assessment of patients with cognitive impairment. Although a positive scan is nonspecific and of limited clinical usefulness, a negative scan may be clinically relevant and suggests the presence of a non-AD cause of cognitive decline.
- The best conventional MRI modality for prion disorders is diffusion-weighted imaging (DWI). The hyperintense DWI signal is caused by restricted diffusion in the vacuolar (spongiform) areas. The pulvinar sign is part of the World Health Organization diagnostic criteria for variant Creutzfeldt-Jakob disease.
- Cognitive deficits in multiple sclerosis (MS) can be attributed not only to visible white matter lesions but also to affected gray matter and normal-appearing brain tissue.
- In MS (or any immunocompromised host), the appearance of patchy, confluent T2 hyperintense signal that extends to the subcortical U-fibers is worrisome for the development of progressive multifocal leukoencephalopathy.
- Neurosarcoidosis or central nervous system (CNS) lupus may be difficult to differentiate from MS on MRI. In neurosarcoidosis, the pituitary stalk and hypothalamus may be involved. Leptomeningeal enhancement, when present, is characteristic of neurosarcoidosis. Lesions in CNS lupus tend to be subcortical, round and patchy (not linear), and spare the callosum.
- MRI is the preferred imaging modality to characterize the pathologic correlates to vascular cognitive impairment, which can be caused by large-vessel infarction, lacunar infarct(s), chronic microvascular ischemia, watershed ischemia, or hemorrhage.

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## INTRODUCTION

Dementia is a common cause of morbidity and mortality worldwide, particularly in the elderly population. The worldwide prevalence of dementia in 2010 was estimated to be more than 35 million, with a projected prevalence in 2050 of more than 115 million.<sup>1</sup> In the United States, 13% of people 65 years and older, and almost 50% of those 85 years and older, have Alzheimer disease (AD).<sup>2</sup> The prevalence of AD in the United States is expected to increase from 5.4 million in 2012 to as much as 16 million in 2050.<sup>2</sup> Although dementia remains a clinical diagnosis, neuroimaging is an increasingly valuable clinical and research tool in the assessment of patients with cognitive symptoms. The introduction of CT in the 1970s allowed for the first time routine visualization of cerebral anatomy in vivo, to assess for structural lesions that could mimic degenerative forms of dementia. MRI, PET, and other imaging modalities allow for improved visualization of the pathophysiologic processes associated with dementia and are routinely used in clinical practice.

## IMAGING GUIDELINES

The indications for utilization of neuroimaging in the routine evaluation of dementia have evolved over the past several decades (**Table 1**). In the 1990s, guidelines published by American and Canadian organizations recommended that neuroimaging be considered in the evaluation of patients with dementia, but routine use of CT or MRI was not recommended for all patients.<sup>3,4</sup> However, initial and subsequently revised guidelines by the European Federation of Neurological Societies, and revised guidelines by the American Academy of Neurology recommended the use of structural neuroimaging (either CT or MRI) in the routine evaluation of patients with dementia.<sup>5–7</sup>

Entity	Year	Recommendations
AAN <sup>a</sup>	1994	Neuroimaging is not routinely recommended (option <sup>b</sup> )
CCCD <sup>c</sup>	1999	Neuroimaging (head CT) is recommended in select clinical situations, such as age <60 y, rapid progression, or gait disturbance (level B <sup>d</sup> )
AAN <sup>a</sup>	2001	Structural neuroimaging (noncontrast CT or MRI) is appropriate in the routine initial evaluation of patients with dementia (guideline <sup>e</sup> )
EFNS <sup>f</sup>	2007	Structural imaging is recommended in every patients suspected of dementia: <ul style="list-style-type: none"> <li>• Noncontrast CT can identify surgically treatable lesions and vascular disease (level A<sup>g</sup>).</li> <li>• To increase specificity, MRI should be used (level A<sup>g</sup>).</li> </ul>
EFNS <sup>f</sup>	2012	Structural imaging (CT or MRI) is recommended in the routine evaluation of every patient with dementia, to exclude secondary causes of dementia (level A <sup>g</sup> )

<sup>a</sup> American Academy of Neurology.

<sup>b</sup> Practice option: unclear clinical certainty (inconclusive or conflicting evidence or opinion).

<sup>c</sup> Canadian Consensus Conference on Dementia.

<sup>d</sup> Level B: based on fair evidence.

<sup>e</sup> Practice guideline: recommendation that reflects moderate clinical certainty, usually class II evidence or strong consensus of class III evidence.

<sup>f</sup> European Federation of Neurological Societies.

<sup>g</sup> Level A: established as effective, based on at least 1 convincing class I study or at least 2 convincing class II studies.

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