



## **Guidance on the use of neuro-imaging in the assessment of dementia in Primary Care**

## Introduction

This guidance for Clinical Commissioning Groups and Health and Wellbeing Boards covers the role of neuro-imaging in the assessment and diagnosis of dementia in primary care.

Over the past few years, prompted by the dementia strategy and more recently the Prime Minister's challenge has led to dementia being considered a 'long term condition' with a move away from secondary care led diagnosis and management to a primary care based service resorting to specialist services only in the more challenging cases.

A group of specialists and generalists in the South West of England considered the role of neuro-imaging in the diagnosis of dementia in primary care, taking into account recent research data, best practice, and NICE guidance.

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## Authors

These guidelines were written by the Diagnostic Pathway Expert Reference Group (DPERG), Neuro-imaging work group. This group is part of the Dementia Network South West, one of three networks within the Strategic Clinical Network for Mental Health, Dementia and Neurological Conditions South West.

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# Recommendations on the use of neuro-imaging in the assessment of dementia in Primary Care

Structural neuro-imaging should be used routinely in the assessment of people with suspected dementia to:

1. Exclude other (potentially reversible) pathologies (1-10%)
2. Establish the sub-type of dementia.

Multi slice CT scans with coronal reformatted views are the most cost effective option first line of the structural neuro-imaging options available.

Besides excluding other potentially reversible pathologies, it can provide a measure of the extent of any cerebro-vascular disease and an estimation of hippocampal atrophy ( a recognised bio-marker for Alzheimer's disease).

To maximise the diagnostic value of the scan, it is important that the scan is reported by a radiologist with skills and experience in the field, and that there are close working relationships between the requesting clinicians and radiologists.

Scan images should be made routinely available to view, on a PACS system, by the requesting clinician as they are a valuable patient / clinician education tool.

While scanning is essential in cases of early or atypical dementia there are cases where scanning may not be appropriate:

In patients with severe dementia (See Appendix 1):

1. Where diagnosis is not in doubt.
2. In patients with significant co morbid physical illness where life expectancy is less than a year.
3. In patients who have had structural cerebral neuro-imaging in the previous year that could be re-reported

In cases where the diagnosis remains unclear further imaging may be appropriate:

- MRI scans are better at detecting subtle vascular changes and may be more helpful in the detection of rarer conditions, eg multiple sclerosis, progressive supra-nuclear palsy, cortico-basilar degeneration and prion diseases.
- In patients with suspected pre senile Alzheimer's disease, MRI may be required to detect atrophy in the posterior parietal regions. We would suggest that, as with the functional scans described below, that these patients would require specialist assessment.

Functional neuro-imaging with FDG-PET or HMPAO-SPECT can help in diagnosing and differentiating Alzheimers disease from Fronto-temporal dementia and DATscans™ can assist in the diagnosis of Lewy body dementia. Given the cost of these interventions we would suggest they are reserved for use in a specialist memory assessment service.

Reference costs for a CT scan are about £100

MRI is more expensive at £160

Functional Neuro-imaging varies from £500 to > £1000

There may be scope for savings under an AQP arrangement.

Depending of practice population demographics it is likely that an individual GP would come across 3-4 incident (undiagnosed) cases of dementia a year.

# Appendix 1: Factors to suggest severe dementia

## Factors to suggest severe dementia

Nursing home residents

Needing assistance with ADLs (hygiene, diet , continence, etc)

MMSE <12/30

Clear Hx of decline over several years

Lack of insight

Older, frequently >80years

## Appendix 2: The evidence base

Neuroimaging is emerging as the most important ancillary investigation in the diagnostic workup of dementia with most clinical guidelines recommending at least one structural imaging procedure in every patient suspected of dementia.<sup>1,2</sup> The traditional purpose of imaging was to exclude potentially treatable causes for cognitive impairment such as tumours, haematomas and hydrocephalus with Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) performing as well in this regard.<sup>3</sup> The yield for this purpose varies between 1% and 10% and may be even lower.<sup>4,5</sup> However, Gifford et al showed that there is considerable uncertainty in the evidence behind clinical prediction rules to identify which patients with dementia should undergo neuroimaging and the application of these rules may miss patients with potentially reversible conditions, hence it is widely accepted that a structural imaging procedure should be performed routinely in each patient with suspected dementia.<sup>6</sup>

With advances in technology, neuroimaging is also being used to include diagnosis of the dementia sub-type. Hippocampal atrophy is seen as a sensitive and specific marker of Alzheimer's Disease (AD)<sup>7,8</sup> and is indeed recognised as a well-validated biomarker of neuronal injury in the new National Institute on Aging and the Alzheimers Association workgroup (NIA-AA) diagnostic criteria.<sup>9</sup> The overall sensitivity and specificity of hippocampal atrophy for detecting mild to moderate AD versus controls were 85% and 88% in a meta-analysis.<sup>10</sup>

In the widely used National Institute for Neurological Disorders and Stroke with the Association Internationale pour la Recherche et l'Enseignement en Neurosciences (NINDS-AIREN) diagnostic criteria for Vascular Dementia (VaD), structural neuroimaging is thought to be essential for the diagnosis by demonstrating a link between the dementia and cerebrovascular disease in the form of large vessel infarcts; single strategically placed infarcts; multiple basal ganglia and white matter lacunae; or extensive peri-ventricular and deep white matter ischaemia.<sup>11</sup>

Dementia with Lewy Bodies (DLB) may not be associated with diagnostic structural imaging changes but functional imaging with dopaminergic single-photon emission computed tomography (SPECT), also called the DATScan™, is useful to differentiate DLB from AD with sensitivity and specificity of around 85%;<sup>12</sup> with low dopamine transporter uptake on the DATScan also being included as a suggestive feature in the Third DLB Consortium Consensus Diagnostic Criteria.<sup>13</sup>

As per the new International Consensus Criteria for behavioural variant fronto-temporal dementia (bvFTD), Probable bvFTD can only be diagnosed if, in addition to the clinical

findings, there are imaging co-relates of at least one of frontal and / or anterior temporal lobe atrophy on MRI / CT or frontal and / or anterior temporal hypo-perfusion or hypo-metabolism on hexamethylpropyleneamine oxime (HMPAO) SPECT or fluorodeoxyglucose-positron emission tomography (FDG-PET).<sup>14</sup>

There is no clear guidance on whether CT or MRI should be the first-line structural imaging procedure. Although they are equally good in excluding potentially treatable conditions, MRI is more sensitive to subtle vascular changes such as strategic infarcts and to changes indicative of specific conditions such as multiple sclerosis, prion disease, the Parkinson's plus syndromes and fronto-temporal lobar degeneration.<sup>15,16</sup> Early age of onset AD patients often have non-amnesic presentations and here MRI may be needed to localise atrophy to the more posterior regions of the pre-cuneus and posterior cingulate cortex.<sup>17</sup> However, CT may be more widely available and cheaper to access than MRI and the modern multi-slice CT has shown excellent reliability, compared to MRI, for detecting hippocampal atrophy, global cortical atrophy and white matter changes.<sup>18</sup>

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