Audit of practice versus guidelines: neuro-imaging in a dementia clinic

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INTRODUCTION

The diagnosis of dementia remains clinical, in other words there is no definitive diagnostic test or biomarker. Criteria for the diagnosis of dementia exist, such as those enshrined in the 'Diagnostic and statistical manual of mental disorders' (DSM),[1] which require the 'development of multiple cognitive deficits that include memory impairment … sufficiently severe to cause impairment in occupational or social functioning.' These criteria require clinical experience and expertise to be used appropriately.

Guidelines for the diagnosis of dementia have been published by expert bodies, including the American Academy of Neurology in 2001,[2] and the European Federation of Neurological Societies in 2007.[3] These recommend the use of certain investigations to supplement clinical assessment in possible dementia cases, including neuro-imaging.

Audit Standard

'Structural imaging should be used in the assessment of people with suspected dementia to exclude other cerebral pathologies and to help establish the subtype diagnosis'

National Institute for Health and Clinical Excellence/Social Care Institute for Excellence (NICE/SCIE) guidelines of 2006, (paragraph 1.4.3.2)[4]

Various neuro-imaging modalities exist, which may broadly be classified as either structural or functional. The former includes computed tomography (CT) and magnetic resonance imaging (MRI); the latter includes single photon emission computed tomography (SPECT) and positron emission tomography (PET). Structural neuro-imaging facilities are increasingly widespread, with concomitantly easier access, even for primary care physicians in some localities.

There is a very broad differential diagnosis of causes of cognitive impairment generally, and of dementia specifically, although neurodegenerative disorders such as Alzheimer's disease (AD) with or without concurrent cerebrovascular disease are the most common in the Walton Centre Cognitive Function Clinic (CFC).[5] Other neurodegenerative disorders seen include the frontotemporal lobar degenerations (FTLDs) which present with either linguistic (progressive nonfluent aphasia, semantic dementia) or behavioural syndromes (behavioural variant FTLD), and dementia with Lewy bodies (DLB) which presents with cognitive decline and a mild Parkinsonian syndrome.

In the field of cognitive disorders, neuro-imaging was initially thought relevant simply to exclude other pathologies which cause cognitive decline, particularly so-called potentially reversible causes such as subdural haematoma, brain tumour, and normal pressure hydrocephalus. However, more sophisticated MR imaging techniques may be used to quantitate cortical atrophy, which may be a signature of pathological neurodegeneration.[6]

An audit of neuro-imaging practice was undertaken in the Cognitive Function Clinic at the Walton Centre for Neurology and Neurosurgery (WCNN), a regional neuroscience centre with a broad catchment population in northwest England and north Wales. Although the clinic does not operate any age restriction on referrals, the case mix of patients seen is generally younger than that typical in old-age psychiatry memory clinics.[7] For the majority of referrals, the service is a one-stop clinic at which clinical, 'bedside' neuropsychological, and neuro-imaging (CT) assessment take place on the same day. More complex cases may need further appointments for further investigation ('formal' neuropsychological assessment, more sophisticated neuro-imaging, neurophysiological tests or cerebrospinal fluid analysis).

PATIENTS AND METHODS

This was a prospective observational study of 100 consecutive patients referred to CFC between March and August 2010. Data collected were reports of neuro-imaging performed prior to and after referral to the clinic.

RESULTS

Patient demographics showed a slight male preponderance (M:F = 55:45) with a broad age range typical of previous cohorts (age range 23-85 years, median 60.5 years)

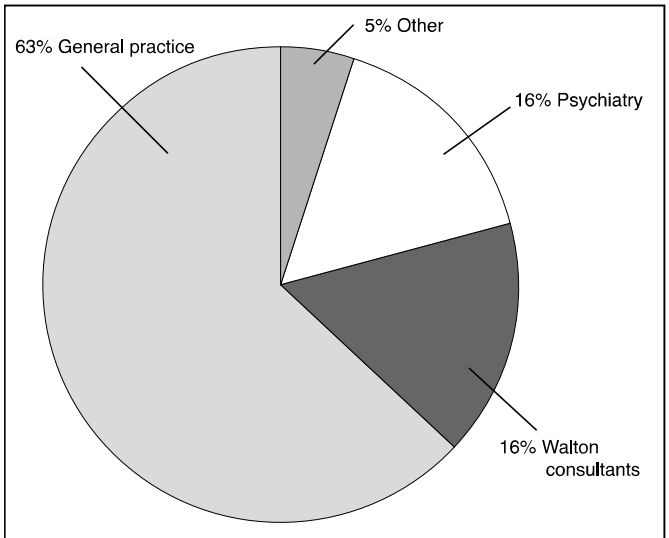

Figure 1 Source of referral for 100 consecutive patients to the Walton Centre's Cognitive Function Clinic

The majority of patients were not demented by DSM-IV criteria, although there was evidence for a neurological or neurodegenerative process causing cognitive impairment short of dementia in some of these, for example patients seen early in the course of DLB may have typical neuropsychological deficits and neurological findings without meeting DSM-IV criteria for dementia. The spectrum of neurodegenerative disorders seen is typical of this CFC; the absence of cases of Parkinson's disease dementia reflects the fact that these patients generally continue to be seen in movement disorders clinics at WCNN.

Demographics	Prospective study (March-August 2010)
N	100
M:F	55:45 (55% male)
Age range	23-85 years, median 60.5 years
Dementia: No dementia	33:67 (dementia prevalence = 33%)
Patients with dementia diagnoses	
Alzheimer's disease / Down syndrome	15
Frontotemporal lobar degenerations	8
Dementia with Lewy bodies	3
Vascular dementia	2
Others	5
Patients without dementia but with probable underlying neurodegenerative process	
Mild cognitive impairment	10
Progressive nonfluent aphasia	2
Dementia with Lewy Bodies	2
HIV related	1

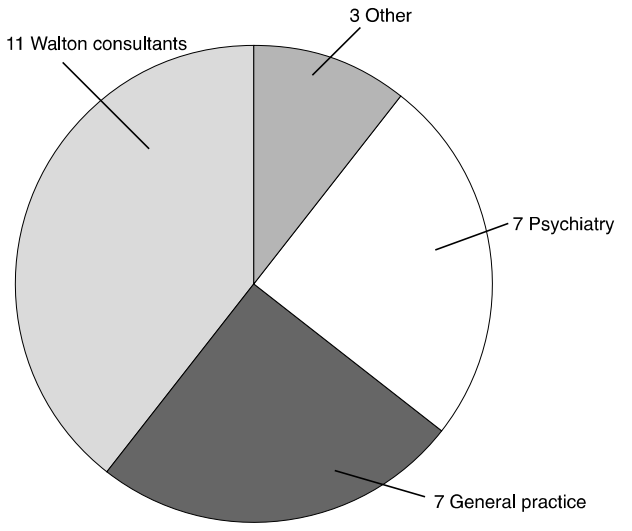

Figure 2 Origins of the 28 patients who had been scanned before referral

Breakdown of neuro-imaging by patient diagnosis showed that patients receiving a diagnosis of dementia were more likely to be imaged (15/33 = 45%) than those not demented (13/67 = 19%), although notably the latter group included some of those with underlying neurological/neurodegenerative disease.

Following CFC consultation, all remaining patients (72) underwent neuro-imaging. In no case was revision of clinical diagnosis (eg, not dementia revised to dementia, or a change of dementia subtype) forthcoming as a result of CFC neuro-imaging.

DISCUSSION

The data indicated that CFC is compliant with NICE/SCIE guidelines on neuro-imaging. Whilst this might be deemed a laudable achievement, clinicians will probably wish to know whether this made any difference to clinical management. Finding potentially reversible causes of cognitive decline is, sadly, exceedingly rare, the most common structural cause seen in CFC being intracranial dural arteriovenous fistula.[8] Therefore, it is not clear what added value structural neuro-imaging brings, although use of longitudinal imaging (eg, follow-up scans) and more sophisticated techniques might contribute more to diagnostic formulation.[6] Our limited experience of functional neuro-imaging with SPECT suggests that it may be helpful in the differentiation of AD and FTLDs.[9]

Should neuro-imaging be a prerequisite for referral to memory clinics, part of the minimum dataset available to the CFC clinician prior to assessment? Probably not. We see a steady trickle of patients referred to CFC with 'cortical atrophy' diagnosed on radiology reports of brain scans undertaken for other purposes, usually headache. Although this label of 'atrophy' may be technically correct according to neuroradiological terminology (Global Cortical Atrophy scale, grade 1 = opening of sulci),[10] it is invariably a qualitative

Figure 3 Global Cortical Atrophy, grade 1: is this non-pathological change appropriate for patient age (75 years) or pathological change suggestive of early AD? Distinction cannot be made without clinical information. Cognitive assessment in this patient (MMSE = 21/30) indicated the latter diagnosis

judgement (see figure 3). Moreover, it is seldom of clinical relevance in our experience, serving only to generate significant and understandable anxiety in patients and primary- and secondary-care physicians unfamiliar with the uses of neuro-imaging in assessment of cognitive problems. As with all imaging findings, clinical-radiological correlation is essential, which privileges the primacy of clinical assessment.

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Peer review

Dr Mike Flanagan, consultant radiologist at Royal Lancaster Infirmary, comments on Andrew Larner's article.

In his discussion, the author asks, 'Should neuro-imaging be a prerequisite for referral to memory clinics?' and then answers, '… probably not'. The radiologists in this Trust [University Hospitals of Morecambe Bay] would almost certainly agree – although it must be said that we have not tried to reach a consensus on the issue.

The main reason to agree with him concerns poor scientific method. Assessment of, say, cortical atrophy is highly subjective with wide inter- and intra-observer variability. We perform structural imaging only; we use an old quad slice CT scanner. MR scanning is reserved for activity with a higher perceived value such as tumour staging or others such as neuro-imaging where the clinical history, signs and symptoms make it a preferable tool to CT. We do not have a radiologist who has a commitment to dementia imaging and do not have the same depth and breadth of connection with colleagues who manage dementia as we have in other specialisms. We scan a population of increasingly elderly patients who all show 'normal' involutional changes in the brain with time. How can a radiologist be sure when an observation gives a reliable indication of abnormal brain in dementia? Well, it's a big challenge, especially because our reports can sometimes carry unjustifiable dogma to the non-expert reader. ('The report says "atrophy" so this must be true.') Poor science, the nature of our resources and the low priority given to this work mean that dementia imaging is a Cinderella slice of our activity pie. It's easy to say that these CT scans are pointless since we hardly ever find any reversible structural abnormality.

And yet, we must remind ourselves that that's the way we are now, not where the science and service might be in a decade. Who is to say what's over the horizon? Better access to functional brain imaging might allow research which improves structural imaging, even in a district general hospital (DGH).

We have had in our midst, for over a year, an exceptional locum colleague. He is a fully trained neuro-radiologist who has transformed our understanding of neuro-imaging and how we look at even the most basic CT scans. He is giving us tertiary-level opinions on site in our DGH. We have access to very high levels of knowledge in anatomy, pathology and clinical diseases of the brain. We are learning how and where to look for help in functional imaging. He has raised the 'brain imaging bar' to a level which will be unsustainable after his departure but which affords a view of how much might be achieved with the right skills, knowledge and equipment in several clinical areas.

Dementia imaging could be one of these but would require a major, sustained and holistic reform. Commissioners please note.