Potential for misdiagnosis in community-acquired PET scans for dementia

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Summary

We reviewed records of patients seen in a tertiary Neurobehavior Clinic to identify those who had community-acquired PET scans as part of their dementia diagnostic evaluation with the goal of assessing factors influencing diagnostic accuracy. We compared outside radiologist PET diagnoses to our consensus clinical diagnosis and collected data regarding clinical variables, ordering reasons, and specialties of interpreting and ordering physicians. Among 1,580 total patients seen in our clinic, 46 met our inclusion criteria. There was disagreement between outside diagnosis based on PET and our consensus diagnosis in 65% ($n = 30$) of patients. Community-acquired PET scans may have lower diagnostic value in dementia evaluation than suggested by prior research and may be associated with significant risks including misdiagnosis with an incurable neurodegenerative disease.

The accurate diagnosis of dementia is challenging even for experienced clinicians. The standard dementia evaluation is extensive, and American Academy of Neurology (AAN) guidelines include a complete medical history, physical examination, neuropsychological testing, laboratory tests, and structural imaging such as MRI or CT.¹ Although the AAN recommendations in 2001 did not endorse functional imaging in the diagnosis of dementia, complementary testing such as fluorodeoxyglucose PET (FDG-PET) has since been proposed to be helpful in distinguishing Alzheimer disease (AD) and frontotemporal dementia (FTD).^{2,3} In 2004, the US Food

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and Drug Administration (FDA) approved brain FDG-PET as an adjunctive measure for diagnostic testing. This decision was based upon a number of studies reporting that FDG-PET can distinguish these 2 disorders with adequate sensitivity and specificity.⁴⁻⁶

A meta-analysis of PET studies in AD conducted prior to 2004 found overall sensitivity and specificity of this test to be 86% each.⁷ However, these studies were not specifically designed to assess how PET would perform in community settings. In particular, study populations were not meant to be representative of community referrals and were not recruited from primary care or other community settings and the study radiologists were highly trained in nuclear medicine at a level beyond most community practitioners.^{7,8} Subsequently, there has been only one PET study of community-dwelling patients suspected of dementia ($n = 102$). This study showed values similar to previous studies assessing accuracy of PET for AD diagnosis (specificity = 81%; sensitivity 78%), but not for diagnosis of FTD (specificity > 95%; sensitivity = 53%).⁹ This was not meant to be a study of practice patterns or performance in community settings and there were important differences from typical community use of PET including specific recruitment of younger patients and interpretations by physicians with specialty training in nuclear medicine.

To date, there have been no studies of actual practice patterns and accuracy of FDG-PET scans for dementia diagnosis as performed in the community. The primary goal of our study was to assess the potential accuracy of FDG-PET as performed in the community setting. We had the following objectives: 1) to estimate the sensitivity and specificity of FDG-PET as obtained and read in the community and 2) to identify factors contributing to the diagnostic value of FDG-PET.

METHODS

This study was approved by the Colorado Multiple Institutional Review Board. A retrospective medical records review was conducted, examining a 6-year period beginning September 15, 2004 (the date of FDA FDG-PET approval) and ending September 15, 2010. The study sample consisted of consecutive patients seen in consultation at the Neurobehavior Clinic of the University of Colorado Hospital (UCH) who underwent PET scans before referral to UCH for the evaluation of dementia. As part of our records review, the following variables were extracted: referral reason, referral source, symptoms as reported by the patient or caregiver, age at symptom onset, education level, occupation, and residing location. We also extracted the following data related to PET scans: specialty of ordering physician, ordering reason, specialty of interpreting physician, duration of symptoms prior to date of PET scan, areas of hypometabolism in specific brain regions, as well as PET reading and impression. Consensus diagnosis was determined by 2 fellowship-trained behavioral neurologists (B.M.K. and C.M.F.) based upon standard diagnostic procedures including a comprehensive history and examination, structural imaging, cognitive testing using the Mini-Mental State Examination and Frontal Assessment Battery, neuropsychological test results when available, and longer term follow-up or response to treatments. When available, PET scans were read by behavioral neurologists (B.M.K. and C.M.F.) to identify areas of hypometabolism as well as reach an independent interpretation based upon standard diagnostic criteria for AD and FTD.³ Behavioral neurologists were not blinded to clinical information or diagnosis.

PET interpretation was classified as follows: true positive (PET interpretation matched consensus diagnosis); true negative (PET exclusion of dementia matched consensus diagnosis); false positive (PET interpretation rendered a new diagnosis that did not match consensus diagnosis); and false negative (PET interpreted as normal, but patient had a diagnosis of dementia). Documented readings of areas of hypometabolism were also compared to readings of our behavioral neurologists to determine accuracy of reported readings separate from official interpretations. The χ^2 test, or Fisher exact test when cell size was less than 5, was used to determine whether factors of interest influenced PET accuracy, and p values less than 0.05 were considered significant.

Table 1 Demographic information

	Total (n = 46)	True positive (n = 13)	True negative (n = 3)	False positive (n = 22)	False negative (n = 8)
Men, %	72	77	67	68	75
Age, y, mean (SD)	63.4 (9.7)	63.8 (9.41)	69.7 (8.5)	61.6 (10.1)	65.1 (9.6)
Age at onset, y, mean (SD)	58 (12.3)	59 (9.99)	68 (9.5)	55 (14.2)	60 (10.3)
MMSE score, max 30, mean (SD)	25.6 (5.5)	22.5 (4.13)	27.3 (2.5)	26.4 (6.8)	27.1 (2.4)
FAB score, max 18, mean (SD)	13.2 (4.5)	12.2 (3.05)	16 (2.6)	13.6 (5.4)	12.3 (1.53)
Consensus diagnosis ^a	Alzheimer disease (n = 11; 24%)	Alzheimer disease (n = 11; 84.6%)	Psychiatric (n = 2; 67%)	FTD (n = 2; 9%)	FTD (n = 1; 12.5%)
	FTD (n = 4; 9%)	FTD (n = 1; 7.7%)	Other (n = 1; 33%)	Psychiatric (n = 6; 27%)	Other (n = 7; 87.5%)
	Psychiatric (n = 8; 17%)	Other (n = 1; 7.7%)		Normal (n = 2; 9%)	
	Normal (n = 2; 4.3%)			Other (n = 12; 54.5%)	
	Other (n = 21; 46%)				

Abbreviations: FAB = Frontal Assessment Battery; FTD = frontotemporal dementia; MMSE = Mini-Mental State Examination.

^aOther diagnoses include reversible and nonreversible causes of dementia such as vitamin B₁₂ deficiency, medication effect, progressive supranuclear palsy, corticobasal degeneration, Parkinson disease dementia, Lewy body dementia, alcoholic dementia, Creutzfeldt-Jakob disease, mild cognitive impairment, and mixed dementia.

RESULTS

We screened 1,580 consecutive patients seen in consultation at our Neurobehavior Clinic during the study period, and identified 49 patients who underwent FDG-PET as part of their diagnostic evaluation. After excluding 3 patients with incomplete records, we examined characteristics of the remaining 46 patients (72% male; mean age 63.4 [$\pm$ 9.7] years). Twenty-seven (n = 27/46; 59%) patients had full neuropsychological testing done either before their first visit or after being seen in our clinic and the results of this testing were utilized in making the consensus diagnosis. Thirteen (n = 13/46; 28%) patients had some form of follow-up in the Neurobehavior Clinic, with the shortest follow-up time being 1 month and longest 5 years and 10 months. Demographic and clinical characteristics of this sample are given in table 1.

When comparing outside interpretation of PET scans to our consensus diagnosis, 65% (n = 30/46) were misdiagnosed. There were 13 true positives (28%), 3 true negatives (6.5%), 22 false positives (48%), and 8 false negatives (17%; table 1). The estimated sensitivity and specificity values of outside PET diagnostic impressions in our cohort were 61.9% and 12%, respectively. The false positive group included those who were cognitively normal (n = 2/22; 9%) or who had reversible causes of dementia, such as vitamin deficiency (n = 1/22; 5%) and psychiatric disorders (n = 6/22; 27%). Among all patients, 16 (n = 16/46; 35%) had the scan mistakenly interpreted as AD.

When available, PET images (n = 41) were read by our behavioral neurologists and compared to our consensus diagnosis. While acquired through different scanners and presented with variable software, all available images were of diagnostic quality. Based on our own PET readings, 37% (n = 15/41; 37%) of patients were misdiagnosed. Of these 41 scans, 17 (n = 17/41; 41%) were true positive, 9 (n = 9/41; 22%) were true negative, 3 (n = 3/41; 7%) were false positive, and 12 (n = 12/41; 29%) were false negative. The

Table 2 PET characteristics of patients presenting to the Neurobehavior Clinic

	True positive (n = 13)	True negative (n = 3)	False positive (n = 22)	False negative (n = 8)	True positive (n = 13)
Ordering physician	Neurologist (n = 37; 80%)	Neurologist (n = 12; 92%)	Neurologist (n = 2; 67%)	Neurologist (n = 15; 68%)	Neurologist (n = 8; 100%)
	PCP (n = 7; 15%)	Missing information (n = 1; 8%)	PCP (n = 1; 33%)	PCP (n = 6; 27%)	
	Missing information (n = 2; 4%)			Missing information (n = 1; 5%)	
Interpreting physician ^a	Neuroradiologist (n = 5; 11%)	Neuroradiologist (n = 2; 15%)	Neuroradiologist (0%)	Neuroradiologist (n = 2; 9%)	Neuroradiologist (n = 1; 13%)
	Radiologist (n = 37; 80%)	Radiologist (n = 9; 69%)	Radiologist (n = 3; 100%)	Radiologist (n = 18; 82%)	Radiologist (n = 7; 87%)
	Other (n = 1; 2%)	Other (n = 1; 8%)		Missing information (n = 2; 9%)	
	Missing information (n = 3; 7%)	Missing information (n = 1; 8%)			
Initial PET impression ^b	Alzheimer disease (n = 26; 56.5%)	Alzheimer disease (n = 10; 77%)	Normal (n = 3; 100%)	Alzheimer disease (n = 16; 73%)	Normal (n = 3; 37.5%)
	FTD (n = 3; 6.5%)	FTD (n = 2; 15%)		FTD (n = 1; 4.5%)	Inconclusive (n = 5; 62.5%)
	Psychiatric (0%)	Other (n = 1; 8%)		Other (n = 5; 22.5%)	
	Normal (n = 6; 13%)				
	Other (n = 11; 24%)				
Symptom to PET duration, y, mean (SD)	5.1 (±8.2)	3.7 (±3.3)	1.8 (1.1)	6.6 (±11.2)	4.5 (4.3)
Reason for ordering PET ^c	AD vs FTD (n = 3; 6.5%)	AD vs FTD (n = 1; 8%)	AD vs FTD (n = 1; 33.5%)	AD vs FTD (n = 1; 5%)	AD vs FTD (n = 0)
	Other (n = 43; 93.5%)	Other (n = 12; 92%)	Other (n = 2; 66.5%)	Other (n = 21; 95%)	Other (n = 8; 100%)

Abbreviations: AD = Alzheimer disease; FTD = frontotemporal dementia; PCP = primary care physician.

^aOther interpreting physician included cardiologist.

^bOther impressions included Lewy body dementia, multi-infarct dementia, cerebral vascular disease, dementia not otherwise specified, inconclusive, and uncertain significance.

^cOther reasons for ordering PET include dementia, memory loss, cognitive impairment, evaluate AD, evaluate FTD, evaluate Lewy body dementia.

estimated specificity and sensitivity of PET as read by our behavioral neurologists including knowledge of clinical data were 75% and 58.6%. The accuracy of behavioral neurologist reading was significantly higher than that of the community impressions (χ^2 , $p = 0.02$).

Table 2 summarizes our examination of factors potentially associated with PET accuracy. PET scans were most often ordered by neurologists (n = 37/46; 80%) followed by primary care physicians (n = 7/46; 16%). There were 32 different ordering physicians, suggesting that our effects were not driven by particular individuals. Scans were ordered for clarification of AD vs FTD in only 7% of cases (n = 3/46), and scans were most often ordered for

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nonspecific reasons (e.g., dementia evaluation). In 7% of cases ($n = 3/46$), scans were ordered for indications not known to be resolvable by PET (e.g., Lewy body dementia). Neuroradiologists read scans in only 11% of cases ($n = 5/46$), 80% were read by radiologists ($n = 37/46$), and 9% ($n = 4/46$) were read by other physicians (e.g., cardiologist) or had missing data. There were 28 different physicians who interpreted the scans (5 patients were missing interpreting physician information), suggesting that our effects were not driven by particular individuals. There was no significant difference in accuracy based on ordering physician specialty, reading physician specialty, ordering indication, or duration of symptoms prior to PET scan (Fisher exact test, $p > 0.05$).

DISCUSSION

To our knowledge, this is the first report on the diagnostic accuracy of PET scans for dementia as ordered and read in the community. Compared to our consensus diagnosis, 65% of patients were misdiagnosed based upon the outside radiologist diagnosis. The diagnostic accuracy of PET we observed is much lower than would be predicted from prior research studies, and seems to result from a combination of factors, including misreading and misinterpretation of scans and acquisition of scans for unclear and non-FDA-approved indications. While discordant with the research studies leading to FDA approval, our findings will come as no surprise to practicing behavioral neurologists who frequently “undiagnose” AD diagnoses based on PET scans.

We begin our discussion by noting that our study has a number of important limitations. First, there may be referral and selection bias in the patients referred to our Neurobehavior Clinic, such that those patients with more complex or contradictory PET findings may be over-represented in our sample. It is possible that patients seen in the community but not referred to us may have had more accurate PET scans, but we have no way of addressing this question with our data. It is also notable that only 49 of over 1,500 patients seen at our clinic had PET scans. We are unaware of any data as to how this compares to national averages of dementia evaluations. Second, we have pathologic confirmation or long-term follow-up in very few of our cases to prove or disprove the accuracy of our consensus diagnosis. However, a study from our Neurobehavior Clinic in which accuracy of clinical diagnosis was assessed in 58 patients suggests that our clinical diagnoses agree with neuropathology in approximately 90% of cases, similar to the accuracy reported from other academic medical centers and case series (C.M.F., unpublished data). However, it is possible that patients “misdiagnosed” with AD based on PET may eventually develop AD. Finally, the sample size for this study is small, limiting our ability to sensitively assess patterns or factors that may have made less obvious contributions to PET accuracy. Nonetheless, we believe several important lessons can be gleaned from our work.

One factor potentially contributing to the inaccuracy of PET scans is misinterpretation on the part of the radiologist. This misinterpretation may occur for several reasons, including lack of specialty training, low volume of scans, inexperience with the diseases being evaluated, absence of clear standards to determine areas of hypometabolism, and lack of important

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historical or physical examination details from ordering physicians. That the majority of scans were read by general radiologists suggests that the lack of specialty training may contribute, although neuroradiologists did not prove significantly more accurate. Compared to our readings, outside physicians made errors of reading (e.g., reporting areas of hypometabolism that were in fact normal) and interpretation (e.g., reporting bifrontal hypometabolism as consistent with AD). The general reliance on interpretation by visual gestalt remains a major issue in the clinical applicability of PET. This approach may cause low reliability because of the unavailability of clear cutoffs between normal and pathologic findings, and may result in significant bias.^{9–11} A 2007 study stated that FDG-PET imaging is unreliable as the sole basis of determining the cause of dementia, and should only be used as an adjunct to other components of the diagnostic evaluation.³ While standards exist for the reading and interpretation of FDG-PET that may be useful for community radiologists,^{11,12} this study suggests that neurologists consider reading their own scans or work more closely with a neuroradiologist, similar to the suggestions of a recent study on the use of MRI in dementia.¹³

One innovation that could improve the accuracy of PET scan interpretation is an objective imaging analysis procedure that can be applied across multiple centers.^{11,12} Better results are achieved when stereotactic surface projection (SSP) maps are used to display FDG-PET data than with traditional transaxial images, and visual interpretation of SSP images shows diagnostic accuracy of 89.6%, with 97.6% specificity and 86% sensitivity values.⁴ It is possible that PET accuracy in community settings would improve if PET services for dementia evaluation were limited to centers with neuroradiologists with either experience or performance-based qualifications and use better defined standards for interpretation.

Another factor contributing to the inaccuracy of PET scans appears to be a tendency of physicians, mainly neurologists, to order the scan for unclear and non-FDA-approved indications. Our impression is that neurologists frequently order PET scans when there is diagnostic uncertainty, but the uncertainty rarely involves distinguishing AD from FTD. While PET is useful for differentiating AD from FTD,³ evidence is insufficient to show its usefulness for conditions such as Lewy body dementia, vascular dementia, or other less common dementias. These disorders have no signature PET patterns, and use of the test to evaluate possible diagnoses such as these would therefore lead to uninterpretable or inaccurate PET scans even in the most experienced hands.

The potential inaccuracy of PET presents a number of undesirable implications. The patient and family may experience significant emotional stress and psychological damage because of misdiagnosis of an incurable neurodegenerative disease. A related problem is the risk of insurance coverage being denied to a healthy person who is wrongly diagnosed by this test. Denial of insurance coverage is a real possibility that we encountered in 2 patients in this cohort. Another problem is that despite the potentially high false-positive rate, physicians often have a false sense of confidence in the diagnosis beyond a standard clinical evaluation, and this misdiagnosis can lead to reduced vigilance for reversible conditions such as vitamin deficiency and depression. A final problem is the high cost of PET scans, ranging between \$1,901 and \$2,535.¹⁴ This

is a huge expense, particularly given that the main indication for PET is to distinguish between 2 diseases for which there are currently no curative treatments.

PET was first developed as a research tool, and studies to date were not designed to accurately assess its performance in the community. Previous studies were primarily designed to answer research questions, and cannot be expected to predict how PET will perform in the real world, where patients present with challenging clinical problems. We endorse the implementation of more ecological studies assessing PET accuracy in community centers to derive a clearer picture of the issues clinicians face when attempting to make a dementia diagnosis, including population-based studies. While we agree that FDG and other forms of PET may have significant value in research, including the selection of groups of participants for clinical trials, we have significant misgivings about using studies for diagnostic purposes in individual patients. Community practice studies may also be relevant for florbetapir PET, recently approved by the FDA for AD diagnosis. Outside of potential issues in terms of radiologist training and interpretation, there is a known potential for false-negative diagnoses, as seen in certain genetic AD kindreds,¹⁵ and false-positive results in cognitively normal individuals or those with non-AD dementias who may have significant brain amyloid even on autopsy studies.^{16,17}

In light of these findings, we suggest that neurologists and other clinicians carefully consider the potential risks and benefits of PET scans in dementia diagnosis before ordering them. While clinicians and patients have many legitimate reasons for greater diagnostic certainty, currently available options including PET and *APOE4* testing have not been proven to improve upon the opinion of astute clinicians and may instead result in ethical or clinical quandaries.

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