Original Article

Evaluation of Dementia: The Case for Neuroimaging All Mild to Moderate Cases

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Abstract

Introduction: The aim of this study was to assess the usefulness of 4 clinical prediction rules, the neuroimaging guidelines from the Canadian Consensus Conference on Dementia (CCCAD) and the modified Hachinski's Ischaemic Score (HIS) in identifying patients with suspected dementia who will benefit from neuroimaging. Materials and Methods: Two hundred and ten consecutive patients were referred to the memory clinic in a geriatric unit for the evaluation of possible dementia. Sensitivity, specificity and likelihood ratios (LR) were calculated for each of the prediction rules and the CCCAD guidelines, in terms of their ability to identify patients with significant lesions [defined firstly as space-occupying lesions (SOL) alone and secondly as SOL or strokes] on neuroimaging. Similar analyses were applied for the HIS in the detection of strokes. Results: When considering SOL alone, sensitivities ranged from 28.6% to 100% and specificities ranged from 21.7% to 88.4%. However, when strokes were included in the definition of significant lesions, sensitivities ranged from 16.2% to 79.0% and specificities ranged from 20.9% to 92.4%. The modified HIS had a similarly poor sensitivity and specificity (43.3% and 78.9% respectively). The LR for the clinical decision tools did not support the use of any particular instrument. Conclusions: Clinical decision tools do not give satisfactory guidance for determining the need for neuroimaging patients with suspected dementia, when the detection of strokes, in addition to SOL, is regarded as important. We recommend therefore that neuroimaging be considered for all patients with suspected mild or moderate dementia in whom the potential benefits of any treatment outweigh the potential risks.

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Key words: Diagnostic medical imaging, Practice guidelines, Sensitivity and specificity

Introduction

Dementia has been reported to affect 4% to 13% of individuals above the age of 65, with the difference in prevalence rates being dependent on the screening tools used and the criteria adopted for the diagnosis of dementia.[1,2] The evaluation of any individual presenting with suspected dementia has a threefold purpose: (1) to establish or exclude a diagnosis of dementia, (2) to identify conditions that may be amenable to interventions that may reverse or alter the progression of cognitive decline, and (3) to allow for prognostication.[3] The requirement for neuroimaging as a routine component of the evaluation process has been widely debated, with proponents citing its requirement for the categorisation of dementia type and its usefulness in detecting space-occupying lesions (SOL) as well as silent strokes as some of the reasons for its necessity,[4] and opponents citing the need for clinical judgement and cost considerations as major reasons for adopting a selective approach.[5]

Opponents to routine neuroimaging have cited their ability to "reliably" identify patients with SOL using clinical prediction rules as reasons for limiting imaging to those patients who fulfilled their respective criteria.[6-8] However, the usefulness of such prediction rules are limited by the fact that they were mostly derived from highly selected populations and were designed solely for the detection of SOL (Table 1). Subsequent testing of such tools in different clinical settings have shown widely differing sensitivities and specificities.[9]

Concurrently, the impact of silent strokes, and possibly

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white matter lesions (WML), on cognitive decline is increasingly being recognised.[10-12] This, coupled with emerging evidence suggesting the possibility of modulating the progression of mixed dementia and vascular dementia,[13,14] as well as the evidence concerning secondary preventive measures for strokes,[15] suggest that neuroimaging may indeed have an important role to play in the evaluation of dementia, apart from the identification of SOL. This suggestion has been well attested to by studies conducted by Chui and Zhang[16] and Massoud et al.[17]

In view of the above, we undertook a study to examine the clinical utility of the 4 clinical prediction rules previously studied by Martin et al,[9] together with the clinical practice guidelines for neuroimaging that were drawn up by the Canadian Consensus Conference on Dementia (CCCAD),[18] in detecting significant lesions (SOL in isolation, and together with strokes) in a cohort of patients presenting to an outpatient memory clinic with the presenting complaint of progressive forgetfulness. In addition, we calculated the modified Hachinski's Ischaemic Score (HIS)[19] for our patients to compare its clinical utility in detecting underlying strokes. Our primary objective was to appraise the evidence and to formulate a clinically useful recommendation for primary care physicians who are increasingly being called upon to evaluate possible cases of dementia. This study was approved by our institutional medical research ethics committee.

Materials and Methods

A total of 210 outpatients referred to our memory clinic for the problem of progressive forgetfulness from April 1994 to January 1999 were studied. Patients were referred from family physicians and specialists in other disciplines. The clinical evaluation of all the patients comprised a detailed medical and psychiatric history, physical examination, psychometric assessment, haematological testing and neuroimaging. All neuroimaging scans were done without intravenous contrast medium on one of two spiral CT scanners with essentially identical protocols (CTi, GE Medical Systems, Milwaukee, WI, or Picker PQ 2000, Picker Medical Systems, USA, with axial sections obtained parallel to the supra-orbital line; at 5 mm interval, 0 mm gap for the posterior fossa, followed by 7 mm thickness, 0 mm gap supratentorially for the CTi and 8 mm thickness, 0 mm gap supratentorially for the Picker PQ).

The medical records of the 210 patients were reviewed retrospectively using a structured protocol to systematically extract the relevant variables of interest (see below) by 1 of 2 researchers (geriatrician or community aged care physician). Based on these variables, the patients were classified as either requiring or not requiring a brain scan in accordance with the 4 clinical prediction rules and the CCCAD guidelines. Similarly, the modified HIS was

calculated (see below) for each of the patients using the data extracted. A neuroradiologist reviewed the CT brain scans of all the above patients and recorded the findings using a semi-structured protocol. All scans were evaluated dichotomously (present/absent) for SOL (hydrocephalus, meningiomas, sub-dural haematomas, sub-dural hygromas and other SOL), strokes i.e., small vessel infarcts or lacunes (defined as well-defined areas >2 mm with CT attenuation the same as cerebrospinal fluid; lesions with these characteristics but <2 mm were considered perivascular spaces) or large vessel infarcts, and white matter lesions (defined as ill defined and moderately hypodense areas of ≥5 mm on unenhanced axial CT brain scans in the periventricular, deep and subcortical white matter). The researchers were blinded to the findings of the neuroradiologist and vice versa.

Study Variables

Study variables collected included basic demographic data as well as information required in the different clinical prediction rules and guidelines (Table 2) as well as the modified HIS. The scoring for the latter was in accordance with the recommendations reported by Rosen et al[19] (abrupt onset 2 points, stepwise deterioration 2 points, somatic complaints 1 point, emotional incontinence 1 point, history or presence of hypertension 1 point, history of strokes 2 points, focal neurological symptoms 2 points, focal neurological signs 2 points), to determine its utility in detecting strokes in this cohort of patients. In our study, patients with modified HIS scores of 3 or more were considered likely to have had strokes.

The study variables thus included the duration of cognitive decline, history of headache, presence or absence of focal signs and symptoms, speech disorders, papilloedema, history of head trauma, acuity of onset and pattern of progression, visual field defects, gait apraxia, use of anti-coagulants or history of bleeding disorders, history of malignancy, urinary incontinence, history of stroke, hypertension, emotional incontinence and somatic complaints. In addition, scores on the Chinese Mini-Mental State Examination (C-MMSE)[20] were recorded. In our study, patients were classified as having speech disorders if they had dysarthria or had been noted to have difficulty with naming objects or comprehending speech. Patients were deemed to have urinary incontinence if they had a history of urinary incontinence more than once weekly. Emotional incontinence was deemed to be present if the patients had a history of increasing agitation, inappropriate jocularity or had been noted to cry easily. Somatic complaints were defined as non-specific complaints that could not be attributed to any defined illness that the patients were suffering from.

Table 1. Clinical Prediction Rules for Neuroimaging: Clinical Setting and Patient Type

Source	Clinical setting	No.	Patient type
Bradshaw et al[6]	Neuroradiology unit	500	Patients referred for evaluation of dementia. Referrals strictly from hospital specialists. Patients with predominantly psychotic features excluded.
Dietch[7]	Neuroradiology unit	200	Patients at least 50 years of age, who have had a neurologic examination and had findings suggestive of dementia studied. First 100 patients who were referred for neuroimaging and fulfilled specified inclusion criteria were compared with the first 100 patients who failed to meet one or more of the inclusion criteria.
Larson et al[8]	Outpatient clinic	107	Consecutive patients seen in outpatient clinic for the problem of suspected dementia.

Table 2. Variables Included in Clinical Prediction Rules

Prediction rule	Variables included/Recommendations
Bradshaw et al[6]	Dementia >1 month or <1 year plus any of the following: headache, focal signs, speech disorder, papilloedema Neuroimaging recommended for patients who meet criteria.
Dietch[7]	Dementia present >1 month, no head trauma in preceding week, gradual onset (>48 hours), no history of strokes, no history of seizures, no history of urinary incontinence, no focal signs, no papilloedema, no visual field defects, no apraxia/ataxia of gait, no headache Neuroimaging not recommended for patients who meet *all* the criteria.
Larson et al,[8] high risk	Acute deterioration and recent onset (<12 months) or mild dementia (C-MMSE >20) Neuroimaging recommended for patients who meet criteria.
Larson et al,[8] low risk	No acute deterioration or have longer duration (>36 months) and severe dementia (C-MMSE <15) Neuroimaging not recommended for patients who meet criteria.
CCCAD guidelines	Age <60, use of anti-coagulants or history of bleeding disorder, recent head trauma, history of carcinoma in sites that metastasize to brain, rapid unexplained decline (<2 years), urinary incontinence and gait disturbance early in course of dementia, localising signs, gait ataxia Patients meeting any of the stated criteria should have neuroimaging.
Modified HIS	Abrupt onset (2 points), history of stroke (2 points), focal neurological signs (2 points), focal neurological symptoms (2 points), stepwise deterioration (1 point), hypertension (1 point), somatic complaints (1 point), emotional incontinence (1 point) Patients with total score ≤2 likely to have Alzheimer's disease.

CCCAD: Canadian Consensus Conference on Dementia; C-MMSE: Chinese Mini-Mental State Examination; HIS: Hachinski's Ischaemic Score

Statistical Analysis

Patients were classified as either requiring or not requiring neuroimaging in accordance with the 4 clinical prediction rules and the CCCAD guidelines. The sensitivity and specificity for each of the instruments were then calculated in terms of the presence or absence of significant findings on neuroimaging. Significant findings were defined, firstly, as any SOL that may be amenable to surgical intervention (i.e., hydrocephalus, meningiomas, sub-dural haematomas, sub-dural hygromas and other SOL), and secondly, as SOL or strokes. The likelihood ratios for positive (LR+) and negative (LR–) "test results" were similarly calculated for both definitions of significant findings. In a separate analysis, sensitivity, specificity, and LR+ and LR– values were calculated for the modified HIS to determine its clinical utility in identifying patients with strokes. All analyses were done with the statistical programme STATA version 7.0 (Stata Corp., College Station, Tx).

Results

Of the 210 patients studied, 79 were male and 131 were female. There was no significant age difference between the 2 gender groups (males: mean age 72.5, standard deviation 9.8; females: mean age 74.7, standard deviation 7.9, $P = 0.2$). SOL were found in 7 patients (2 meningiomas, 4 sub-dural hygromas and 1 metastatic deposit from cervical carcinoma). One hundred and twenty patients were found to have strokes on neuroimaging, out of which 70 (58.3%) did not have a previous history of strokes and did not demonstrate any focal neurological signs.

The neuroimaging findings are summarised in Table 3. If the prediction rule recommended by Bradshaw et al[6] had been applied, 158 CT scans would have been avoided. However, 5 SOL and 85 strokes would have been missed. Similarly, 112 scans could have been avoided with the application of Dietch's recommendations, but at the expense of missing 2 SOL and 51 strokes. The corresponding

Table 3. Neuroimaging Findings According to Prediction Rules

Prediction rule	No. of scans indicated	No. of SOLs detected	No. of strokes detected	No. of scans avoided	No. of SOLs missed	No. of strokes missed
Bradshaw et al[6]	52	2	35	158	5	85
Dietch[7]	98	5	69	112	2	51
Larson et al,[8] high risk	25	3	17	185	4	103
Larson et al,[8] low risk	123	4	63	87	3	57
CCCAD[9]	166	7	94	64	0	26

CCCAD: Canadian Consensus Conference on Dementia; SOL: space-occupying lesion

Table 4. Performance of Prediction Rules in Detecting Significant Lesions

Space-occupying lesions				
Prediction rule	Sensitivity, % (95% CI)	Specificity, % (95% CI)	LR+	LR–
Bradshaw et al[6]	28.6 (3.7, 71.0)	75.4 (68.8, 81.1)	1.16	0.95
Dietch[7]	71.4 (29.0, 96.3)	54.2 (47.1, 61.2)	1.56	0.53
Larson et al,[8] high risk	50.0 (11.8, 88.2)	88.4 (83.0, 92.6)	4.31	0.57
Larson et al,[8] low risk	47.1 (18.4, 90.1)	41.4 (24.5, 48.5)	0.97	1.04
CCCAD[9]	100.0 (59.0, 100.0)	21.7 (16.2, 28.0)	1.28	0
Space-occupying lesions or strokes				
Prediction rule	Sensitivity, % (95% CI)	Specificity, % (95% CI)	LR+	LR–
Bradshaw et al[6]	29.0 (21.2, 37.9)	81.4 (71.6, 89.0)	1.56	0.87
Dietch[7]	58.9 (49.7, 67.6)	70.9 (60.1, 80.2)	2.02	0.58
Larson et al,[8] high risk	16.2 (10.1, 24.2)	92.4 (84.2, 97.2)	2.13	0.91
Larson et al,[8] low risk	53.2 (44.1, 62.2)	33.7 (23.9, 44.7)	0.80	1.39
CCCAD[9]	79.0 (70.8, 85.8)	20.9 (12.9, 31.0)	1.0	1.0

95% CI: 95% confidence interval; CCCAD: Canadian Consensus Conference on Dementia

LR+ : Likelihood ratio of positive "test result" i.e., proportion of subjects with a positive finding and positive "test result" compared to proportion of subjects without a positive finding but having a positive "test result".

LR– : Likelihood ratio of negative "test result" i.e., proportion of subjects with a positive finding but a negative "test result" compared to proportion of subjects without a positive finding and having a negative "test result".

number of scans avoided and the number of lesions missed for the remaining prediction rules and guidelines are as follows: Larson et al[8] (high risk) – 185 scans avoided, 4 SOL and 103 strokes missed; Larson et al[8] (low risk) – 87 scans avoided, 3 SOL and 57 strokes missed; CCCAD guidelines – 44 scans avoided, no SOL and 26 strokes missed.

The sensitivity, specificity and likelihood ratios of the clinical prediction rules and the CCCAD guidelines are summarised in Table 4. The CCCAD guidelines had the highest sensitivity in detecting significant findings (SOL in isolation, as well as SOL or strokes). However, when the prediction rules and clinical guidelines were considered in terms of their likelihood ratios (which embrace the dimensions of both sensitivity as well as specificity), the LR+ ranged from 0.97 (Larson et al,[8] low risk) to 4.31 (Larson et al,[8] high risk) for SOL alone, and 0.80 (Larson

et al,[8] low risk) to 2.13 (Larson et al,[8] high risk) for SOL or strokes. The corresponding LR– values ranged from 0 (CCCAD[18]) to 1.04 (Larson et al,[8] low risk) and 0.58 (Dietch[7]) to 1.39 (Larson et al,[8] low risk).

The modified HIS had a sensitivity of 43.3% (95% CI, 34.3, 52.7) and a specificity of 78.9% (95% CI, 69.0, 86.8) for detecting strokes in our patients when we used a cutoff of ≥3 to identify patients with higher vascular risk factors and hence, a greater risk of strokes. The corresponding LR+ and LR– values were 2.05 and 0.72, respectively. Using a modified HIS score of ≥3 to determine the necessity for neuroimaging, we would have missed 68 patients with strokes.

Discussion

An important element in the diagnostic approach to a patient presenting with suspected dementia is the

identification of conditions, the treatment of which results in the reversal of the cognitive impairment, or the identification of conditions for which treatment may result in amelioration of the progression of cognitive decline. In the first instance, reversible dementias may be caused by metabolic or mood disorders, or SOL which may be amenable to surgical intervention (e.g., meningiomas, subdural collections and normopressure hydrocephalus). While the earlier group of conditions can be detected through careful clinical examination and blood investigations, the latter category can only be reliably diagnosed through neuroimaging. It is for this latter group that most of the clinical prediction rules cited in this study were proposed, with the common objective of facilitating a rational, more selective decision process for requesting brain scans.

Based on their original data sets, both Dietch[7] and Larson et al[8] were able to achieve 100% sensitivity in detecting SOL using their respective clinical prediction tools, with corresponding specificities ranging from 52.9% to 85.7%. However, when the same prediction tools were applied in the setting of an academic geriatric centre, Martin et al[9] found that the criteria formulated by Dietch had a sensitivity of 87.5% while that formulated by Larson and colleagues had a sensitivity of only 25%. More recently, in their review of clinical prediction rules for neuroimaging, Gifford and colleagues[21] tested the different prediction rules and the CCCAD guidelines on a hypothetical population and arrived at similarly divergent sensitivities and specificities. Using sensitivity and specificity data collated by Gifford et al, the LR+ values for SOL would have ranged from 0.6 for "Bradshaw's criteria"[6] to 2.26 for the CCCAD guidelines,[18] while the LR- values would have ranged from 0.25 (CCCAD guidelines) to 1.17 (Larson et al,[8] high risk) for the population studied by Martin and colleagues.[9]

In our study population, the CCCAD guidelines had the highest sensitivity (100%) for the detection of SOL but also resulted in the highest number of scans being required (166 out of 210). The clinical prediction rule with the second highest sensitivity of 71.4% was that recommended by Dietch[7] while the prediction rule formulated by Bradshaw et al[6] had the lowest sensitivity. The LR+ values in our study population ranged from 1.16 (using the prediction rule formulated by Bradshaw et al[6]) to 4.31 (Larson et al,[8] high risk). This means that, at best, Larson's "high risk" criterion is 4.31 times more likely to be fulfilled in a person with a positive SOL on neuroimaging than in a person without a SOL. The LR+ value for the CCCAD guidelines[18] was 1.28 i.e., the CCCAD guidelines are only 1.28 times more likely to be fulfilled in a person with a SOL than in a person without such a lesion. Conversely, the LR- value for the CCCAD guidelines was 0 in our study population,

suggesting that patients who did not fulfill the CCCAD guidelines for neuroimaging were highly unlikely to have SOL within the brain.

It has been stated by Wasson and colleagues[22] that "the ultimate measure of a clinical prediction rule is its effect on patient care", and that in using such rules, "one should try to minimise the chance of serious error in patient care". Similarly, Gifford et al[21] recommended that clinical prediction rules used for the assessment of dementia patients should possess a high sensitivity to minimise the proportion of false-negative findings.[21] Our finding that the CCCAD guidelines have a high sensitivity and low LR- value in identifying patients with SOL concur with the results cited by Freter et al[23] and suggest that the CCCAD guidelines would be useful in identifying patients with SOL in the setting of a memory clinic for assessing patients with suspected dementia. The widely varying sensitivities, specificities for the other clinical prediction rules that were demonstrated by Martin's group and reaffirmed in our study population, together with the relatively low likelihood ratios of the different "rules" suggest that they have limited usefulness in actual clinical practice.

Patients with SOL however, constitute only a small percentage of all patients presenting with suspected dementia (3.3% in our study population, 3.1% in the study reported by Freter et al[23]) and furthermore, the benefits of surgical intervention in such circumstances are not well substantiated, with patients suffering from mild cognitive decline being more likely to improve with intervention.[23] Conversely, there is growing evidence concerning the importance of cerebrovascular disease in patients with dementia – Snowdon et al[11] demonstrated that strokes impact negatively on the cognitive function of patients with AD, while Vermeer et al showed recently that silent brain infarcts more than doubled the risk of dementia and led to a steeper decline in cognitive function in patients who were so affected.[10] In addition, we also recently reported that WML may impact negatively on functional outcomes in patients with dementia.[12]

In view of these developments, we elected to study the utility of the clinical prediction rules and the CCCAD guidelines in detecting clinically significant lesions, using a definition that incorporated strokes in addition to SOL. As noted, the CCCAD guidelines had the highest sensitivity (79.0%). However, 26 patients with strokes were missed when the decision for neuroimaging was based on these guidelines. When strokes were incorporated within the definition of clinically significant lesions, Larson's "high risk" criteria was found to have the lowest sensitivity (16.2%) and would have resulted in 99 strokes being missed. The LR+ values for the prediction tools ranged from 0.8 (Larson et al,[8] low risk) to 2.13 (Larson et al,[8] high

risk[8]), while the LR- values ranged from 0.58 (Dietch[7]) to 1.39 (Larson et al, low risk). As can be seen, the performance of the clinical prediction rules and clinical guidelines in determining the need for neuroimaging were far from satisfactory when a more encompassing (but not unreasonable) definition of clinically significant lesions was applied.

To further assess the possibility of reducing the need for neuroimaging through the use of "clinical decision tools", we studied the ability of the modified HIS to detect brain infarcts in our study population. The HIS was derived from patient characteristics and cardiovascular risk factors and has been widely used, in both its original and modified forms, to differentiate patients with pure AD from those with VAD.[24-26] It has been shown to be comparable to the criteria proposed by the National Institute of Neurological Disorders and Strokes and the Association Internationale pour la Recherche et l'Enseignement en Neurosciences (NINDS-AIREN)[27] or the Alzheimer's Disease Diagnostic and Treatment Centers (ADDTC) of the State of California[28] in its ability to exclude patients with pure AD. However, it has also been demonstrated that the HIS fares poorly in identifying patients with mixed dementia i.e., patients with AD and concomitant brain infarcts.[24] In view of the fact that the HIS was based largely on known cardiovascular risk factors, we thought it reasonable to study its usefulness in predicting the presence of cerebrovascular disease. We had chosen to use the lower cutoff score of 3 or more (modified HIS) as the HIS has been shown to be more useful in identifying pure AD and excluding vascular lesions at such a cutoff.[24] As in the case of the other prediction rules, the modified HIS performed poorly in identifying patients with brain infarcts, with a sensitivity of 43.3% and an exclusion of 68 patients with brain infarcts when the cutoff of ≥3 was applied.

Our study has shown that while some of the clinical prediction rules (especially the CCCAD guidelines) can be useful for detecting SOL, these same rules would generally have resulted in a sizeable proportion of patients with strokes (21.6% to 85.8%) being missed. However, given the importance of detecting underlying strokes in those presenting with possible dementia, these findings then lead to the question of whether neuroimaging should therefore be routinely recommended for all patients presenting for the evaluation of possible dementia. We believe this question is best answered by considering the relative merits of making such diagnoses, in terms of the relative benefits and risks associated with currently available treatment modalities for secondary stroke prevention. For example, while antiplatelet therapy is widely recognised to be effective in lowering the risk of recurrent strokes (2 recurrent strokes may be prevented for every 100 patients treated for 2 years), the risks associated with such treatment should not be overlooked (1 gastrointestinal bleed may be encountered in the same group of patients within the same period).[29,30] Furthermore, it should be noted that the majority of patients presenting with possible dementia are elderly, and the risk of gastrointestinal haemorrhage from antiplatelet treatment increases with age.[31-33]

Given the above context, we suggest that neuroimaging for the primary purpose of excluding the more commonly encountered strokes, should not be considered for those patients with advanced dementia in whom the prospects of improvement in cognition, functional status or quality of life would be slim. In this group, the topic of secondary stroke prevention would also lose its cogency, with the risks associated with antiplatelet use clearly outweighing benefits.

The same consideration will prevail when considering the need for neuroimaging for the primary purpose of excluding SOL. In this regard too, the time-line of 2 years that is stated within the CCCAD guidelines may be considered as an additional useful guideline: a patient with significant dementia of more than 2 years' duration is thus very unlikely to benefit from any neuro-radiological evaluation for either SOL or strokes.

Our study is not without its limitations. Firstly, the retrospective nature of our study means that our patients did not undergo a strictly uniform protocol for neuroimaging, even though both scanners that were used applied scan parameters that were essentially identical except for a minimal difference in slice thickness supratentorially. However, it has previously been shown that use of different CT scanners with different scan protocols and slice thickness has not impeded scan evaluation.[34] We have tried to minimise the resultant limitations by having all films reviewed by a single neuro-radiologist using a standardised qualitative assessment format.

Secondly, the ability of CT scans to detect ischaemic cerebrovascular disease lesions (ICVD) and the clinical significance of such lesions may be questioned. We acknowledge that CT imaging is less sensitive than magnetic resonance imaging (MRI) for the detection of ICVD. However, it is impractical to apply MRI for all patients presenting with suspected dementia and we propose that our findings may in fact underestimate the "miss rate" of silent infarcts.

Thirdly, we recognise that the retrospective nature of our study may affect the precision of our results. Retrospective power calculations based on the parameters for estimating sensitivities and specificities of prediction rules in detecting SOL indicated that we would be able to attain precisions (95% confidence level) ranging from ±18% to ±30% for our sensitivity values, and precisions of ±5% to ±7% for our specificity values. Similarly, for SOL or strokes, the

precision ranged from ±6% to ±9% for sensitivities, and ±6% to ±10% for specificities.

Conclusion

The currently established clinical decision tools (with the possible exception of the CCCAD guidelines) for the use of neuroimaging in the evaluation of patients with suspected dementia for detecting SOL are of limited utility. Application of such tools to determine the requirement for neuroimaging would result in a sizeable proportion of patients with strokes being missed. We recommend therefore that neuroimaging be routinely considered in the evaluation of patients with suspected dementia, as long as the potential benefits associated with possible subsequent treatment are judged to outweigh the associated risks. This approach will thus exclude patients who, when presenting to the clinician for the first time for problems related to possible dementia, are already advanced in their cognitive impairment, especially when the cognitive state has been steadily declining for at least 2 years.

Competing Interests

None identified.

REFERENCES

1. Kay DW. New knowledge on the epidemiology of dementia. Rev Clin Gerontol 1999;9:163-82 (CINAHL, Accession Number: 2000005792).
2. Chong MS, Sahadevan S. An evidence-based clinical approach to the diagnosis of dementia. Ann Acad Med Singapore 2003;32:740-8.
3. Corey-Bloom J, Thal LJ, Galasko D, Folstein M, Drachman D, Raskind M, et al. Diagnosis and evaluation of dementia. Neurology 1995;45:211-8.
4. Katzman R. Should a major imaging procedure (CT or MRI) be required in the workup of dementia? An affirmative view. J Fam Pract 1990;31:401-5.
5. Clarfield AM, Larson EB. Should a major imaging procedure (CT or MRI) be required in the workup of dementia? An opposing view. J Fam Pract 1990;31:405-10.
6. Bradshaw JR, Thomson JL, Campbell MJ. Computed tomography in the investigation of dementia. BMJ 1983;286:277-80.
7. Dietch JT. Computerized tomographic scanning in cases of dementia. West J Med 1983;138:835-7.
8. Larson EB, Reifler BV, Featherstone HJ, English DR. Dementia in elderly outpatients: a prospective study. Ann Intern Med 1984;100:417-23.
9. Martin DC, Miller J, Kapoor W, Karpf M, Boller F. Clinical prediction rules for computed tomographic scanning in senile dementia. Arch Intern Med 1987;147:77-80.
10. Vermeer SE, Prins ND, den Heijer T, Hofman A, Koudstaal PJ, Breteler MM. Silent brain infarcts and the risk of dementia and cognitive decline. N Engl J Med 2003;348:1215-22.
11. Snowdon DA, Greiner LH, Mortimer JA, Riley KP, Greiner PA, Markesbery WR. Brain infarction and the clinical expression of Alzheimer disease. The Nun Study. JAMA 1997;277:813-7.
12. Sitoh YY, Sitoh YY, Sahadevan S. Clinical significance of cerebral white matter lesions in older Asians with suspected dementia. Age Ageing 2004;33:67-71.
13. Gabelli C. Rivastigmine: an update on therapeutic efficacy in Alzheimer's disease and other conditions. Curr Med Res Opin 2003;19:69-82.
14. Forette F, Seux ML, Staessen JA, Thijs L, Birkenhager WH, Babarskiene MR, et al Prevention of dementia in randomised double-blind placebo-controlled Systolic Hypertension in Europe (Syst-Eur) trial. Lancet 1998;352:1347-51.
15. Albers GW, Tijssen JG. Antiplatelet therapy: new foundations for optimal treatment decisions. Neurology 1999;53:S25-S31.
16. Chui H, Zhang Q. Evaluation of dementia: a systematic study of the usefulness of the American Academy of Neurology's practice parameters. Neurology 1997;49:925-35.
17. Massoud F, Devi G, Moroney JT, Stern Y, Lawton A, Bell K, et al. The role of routine laboratory studies and neuroimaging in the diagnosis of dementia: a clinicopathological study. J Am Geriatr Soc 2000;48:1204-10.
18. Patterson CJ, Gauthier S, Bergman H, Cohen CA, Feightner JW, Feldman H, et al. Canadian Consensus Conference on Dementia: a physician's guide to using the recommendations. CMAJ 1999;160:1738-42.
19. Rosen WG, Terry RD, Fuld PA, Katzman R, Peck A. Pathological verification of ischemic score in differentiation of dementias. Ann Neurol 1980;7:486-8.
20. Katzman R, Zhang MY, Ouang-Ya-Qu, Wang ZY, Liu WT, Yu E, et al. A Chinese version of the Mini-Mental State Examination; impact of illiteracy in a Shanghai dementia survey. J Clin Epidemiol 1988;41:971-8.
21. Gifford DR, Holloway RG, Vickrey BG. Systematic review of clinical prediction rules for neuroimaging in the evaluation of dementia. Arch Intern Med 2000;160:2855-62.
22. Wasson JH, Sox HC, Neff RK, Goldman L. Clinical prediction rules. Applications and methodological standards. N Engl J Med 1985;313:793-9.
23. Freter S, Bergman H, Gold S, Chertkow H, Clarfield AM. Prevalence of potentially reversible dementias and actual reversibility in a memory clinic cohort. CMAJ 1998;159:657-62.
24. Amar K, Wilcock GK, Scott M. The diagnosis of vascular dementia in the light of the new criteria. Age Ageing 1996;25:51-5.
25. Moroney JT, Bagiella E, Desmond DW, Hachinski VC, Molsa PK, Gustafson L, et al. Meta-analysis of the Hachinski Ischemic Score in pathologically verified dementias. Neurology 1997;49:1096-105.
26. Gold G, Giannakopoulos P, Montes-Paixao Junior C, Herrmann FR, Mulligan R, Michel JP, et al. Sensitivity and specificity of newly proposed clinical criteria for possible vascular dementia. Neurology 1997;49:690-4.
27. Roman GC, Tatemichi TK, Erkinjuntti T, Cummings JL, Masdeu JC, Garcia JH, et al. Vascular dementia: diagnostic criteria for research studies. Report of the NINDS-AIREN International Workshop. Neurology 1993;43:250-60.
28. Chui HC, Victoroff JI, Margolin D, Jagust W, Shankle R, Katzman R. Criteria for the diagnosis of ischemic vascular dementia proposed by the State of California Alzheimer's Disease Diagnostic and Treatment Centers. Neurology 1992;42:473-80.
29. Antithrombotic Trialists' Collaboration. Collaborative meta-analysis of randomised trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. BMJ 2002;324:71-86. Erratum in: BMJ 2002;324:141.
30. Derry S, Loke YK. Risk of gastrointestinal haemorrhage with long term use of aspirin: meta-analysis. BMJ 2000;321:1183-7.
31. Wolfe MM, Lichtenstein DR, Singh G. Gastrointestinal toxicity of nonsteroidal antiinflammatory drugs. N Engl J Med 1999;340:1888-99. Erratum in: N Engl J Med 1999;341:548.
32. Pilotto A, Franceschi M, Leandro G, Paris F, Niro V, Longo MG, et al. The risk of upper gastrointestinal bleeding in elderly users of aspirin and other non-steroidal anti-inflammatory drugs: the role of gastroprotective drugs. Aging Clin Exp Res 2003;15:494-9.
33. Weksler BB. Antiplatelet agents in stroke prevention. combination therapy: present and future. Cerebrovasc Dis 2000;10 Suppl 5:41-8.
34. Wahlund LO, Barkhof F, Fazekas F, Bronge L, Augustin M, Sjogren M, et al. European Task Force on Age-Related White Matter Changes. A new rating scale for age-related white matter changes applicable to MRI and CT. Stroke 2001;32:1318-22.