

Assessment of Cognitive Impairment: The Role of CT

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ABSTRACT: The value of CT as a routine screening procedure in the investigation of cognitive impairment is being increasingly challenged. To address this issue, we reviewed the records of 175 patients with intellectual deficits admitted to a Behavioural Neurology Unit over a two-year period. In the vast majority of cases, ie. 82%, the CT served essentially to confirm the clinical impression and added no new diagnostic information that impacted the management of the presenting problem. In 15% of cases the CT scan was helpful for diagnosis, especially in the differentiation between Alzheimer's disease and multi-infarct dementia.

RÉSUMÉ: *Évaluation d'un déficit cognitif: rôle du CT scan.* La valeur de CT scan comme procédé de dépistage de routine dans l'investigation de déficits cognitifs est de plus en plus contestée. Pour examiner cette question, nous avons révisé les dossiers de 175 patients présentant un déficit intellectuel admis à l'Unité de Neurologie du Comportement pendant une période de deux ans. Dans la grande majorité des cas, soit 82%, le CT scan a servi essentiellement à confirmer l'impression clinique et n'ajoutait aucune information diagnostique nouvelle ayant un impact sur la conduite du traitement en ce qui a trait au motif de la consultation. Dans 15% des cas, le CT scan a aidé au diagnostic, spécialement dans la différenciation entre la maladie d'Alzheimer et la démence par infarctus cérébral multiple.

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CT of the brain is a standard procedure for identifying reversible causes of dementia such as hydrocephalus and intracranial masses.^{1,2,3,4} In addition, CT may also help differentiate between Alzheimer's disease (AD) and multi-infarct dementia (MID), an important distinction due to the differences in approach to management, prognosis and genetic implications.^{5,6} The value of CT as a routine screening procedure in the investigation of cognitive deterioration is, however, being increasingly challenged.^{2,7,8,9} To address this issue we examined the clinical impact of CT on diagnosis and management in a consecutive series of patients admitted for evaluation of cognitive impairment to a specialized Behavioral Neurology assessment unit in a geriatric hospital.

METHODS

The hospital charts of 190 consecutive patients admitted to the Behavioral Neurology Unit at Baycrest Hospital between March, 1986 and March, 1988 were reviewed with regards to admission diagnosis, CT results, discharge diagnosis and pertinent operative notes and surgical follow-up. One hundred and

sixty-six patients were referred for assessment of dementia. The remaining 24 had less widespread cognitive or language impairment due to focal brain lesions (eg. aphasia following cerebral infarction). There were 84 females with a mean age of 72.6 ± 9.3 years (range: 43-88 years) and 106 males with a mean age of 70.5 ± 11.3 years (range: 20-92 years).

The clinical evaluation of the patients admitted to the unit included a medical history, general physical and neurological examination, and a detailed mental status examination. The assessments were carried out by a neurologist in all cases. When indicated, patients were referred for additional evaluation by a neuropsychologist, speech pathologist, or medical subspecialist (eg. psychiatrist, geriatrician).

Laboratory evaluation included CT of the brain, EEG and blood tests (ie. CBC, sedimentation rate, thyroid function studies, serum B12 and folate levels, serum calcium and phosphorus, liver function tests and renal function tests). Neurological diagnoses were made according to standard criteria.¹⁰ In addition, all patients with AD met NINCDS-ADRDA criteria for probable AD.¹¹ All CT scans were carried out at Mount Sinai Hospital.

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Among the 190 patients admitted to the unit, 15 had CT scans done prior to admission and were not included in this retrospective analysis. The remaining 175 who had a scan done after admission comprised the study sample. These CT scan reports were reviewed and classified under one or more of seven categories as: 1) supporting the initial clinical diagnosis; 2) differentiating between two or more equally plausible diagnoses; 3) suggesting an unexpected primary clinical diagnosis; 4) leading to a new investigation or to a change in patient management; 5) resulting in a change in prognosis; 6) providing an incidental diagnosis; and 7) providing information that suggested a misleading diagnosis that was subsequently found to be incorrect.

CT scans were classified as supporting the initial diagnosis if there was a lesion, such as a tumor, that was clinically suspected. Alternatively, a CT that was normal or showed atrophy was also considered as supporting the initial diagnosis in disorders such as AD. The CT scans were interpreted using standard neuroradiological criteria for lesions such as infarct, atrophy and tumor.

RESULTS

Table 1 lists the diagnoses of the 175 patients evaluated, along with the number of patients in each group. Table 2 shows the number of patients in each CT category. There was a high level of confidence about the diagnosis based upon the history and physical examination in 145 cases (83%). In all but one of these cases, ie. in 99%, the CT supported the initial diagnosis. In the exceptional case, the initial working diagnosis was multiple sclerosis. The CT showed no evidence of demyelination in this disorder. Although the scan did not show positive evidence of multiple sclerosis, it did not rule out the diagnosis since CT may not show features of demyelination in this disorder. One of the cases in which the CT supported the initial diagnosis had an unexpected finding of an acoustic neuroma. In another case the

Table 1: Diagnoses of Patients

Diagnosis	Number
Alzheimer's Disease	68
Multi-infarct Dementia	26
Parkinson's Disease and Dementia	25
Stroke	23
Alcoholism	3
Multiple Sclerosis Dementia	2
Huntington's Disease	1
Normal Pressure Hydrocephalus	3
Pick's Disease	1
Olivopontocerebellar Atrophy	2
Progressive Supranuclear Palsy	3
Tumor	2
Head Injury	1
Aneurysm	3
Anoxia	3
Hypoglycemia	1
Depression	1
Senile Chorea	1
Undiagnosed	6
	175

CT added information by suggesting that a tumor appeared resectable.

In the remaining 30 cases CT was ordered to help differentiate between two or more plausible differential diagnoses and was helpful in 27 patients. These 27 cases comprised 15% of the total sample. In 22 cases the differentiation involved MID versus AD. In all but one case the CT was negative, ie. showed no infarcts and a presumptive diagnosis of AD was made. It was recognized, however, that patients with negative scans could still have had small brain infarcts that were not seen on CT and thus could still have had MID or MID combined with AD. Among the remaining cases, one patient with a frontal lobe syndrome had a CT which showed bilateral basal ganglia calcification which was not suspected clinically and which may comprise a treatable disorder (see discussion). Four patients had clinical features suggestive of NPH. In all four, the CT provided evidence that led to further investigations, ie. a CSF flow study. Two patients had a positive CSF flow study and went on to have a ventriculo-peritoneal shunt inserted. One of these patients subsequently showed improvement in memory. Two patients had a negative or equivocal CSF flow study and were not shunted. In these cases the CT can be considered to have provided misleading information. CT was also very helpful in a patient with a differential diagnosis of Huntington's disease vs senile chorea with cognitive impairment. The absence of caudate atrophy weighed heavily against a diagnosis of Huntington's disease. This was subsequently supported by a normal SPECT scan.

The five patients in whom the CT findings had an impact on prognosis were a patient whose tumor was shown to appear resectable, a patient who was diagnosed as senile chorea with cognitive impairment as opposed to Huntington's disease, the two cases who were diagnosed as having normal pressure hydrocephalus rather than Alzheimer's disease and a patient with an incidental acoustic neuroma.

There were a relatively large number of patients with incidental findings. These included radiological diagnoses such as cerebellar atrophy. Leuko-ariosis was also noted.

DISCUSSION

Our retrospective survey showed that a working clinical diagnosis could be made in the vast majority of cases (ie. 83%) referred to an interdisciplinary neurobehavioural assessment team for in-patient evaluation of cognitive impairment. In all but one of these cases the CT findings served only to confirm the clinical impression and added no new diagnostic information

Table 2: CT Findings

CT Classification	Frequency
Supporting the Initial Diagnosis	144 (82%)
Differentiating Between 2 Diagnoses	27 (15%)
Suggesting an Unexpected Primary Diagnosis	1 (0.06%)
Leading to a New Investigation or Change in Management	4 (2%)
Resulting in a Change in Prognosis	5 (3%)
Providing an Incidental Diagnosis	40 (23%)
Suggesting a Misleading Diagnosis	2 (1%)

that impacted on the presenting problem. In 27 of 30 cases, on the other hand, the CT was important for diagnosis by differentiating between equally plausible clinical disorders. In the majority of these cases, the differential diagnosis involved AD versus MID.

Although neither AD nor MID are reversible causes of dementia, it is still important to distinguish between these disorders due to differences in genetic implications and in approaches to management. Families are becoming increasingly concerned about the hereditary implications of a diagnosis of Alzheimer's disease. In terms of management, patients with MID require careful assessment of the cause of their multiple infarcts and may benefit from treatment aimed at controlling risk factors such as hypertension, diabetes and cardiac disease.

Whereas there are clinical indicators to distinguish AD from MID,^{12,13} the differentiation between these disorders is not always clear on the basis of history and physical examination. For example, multiple brain infarcts may occur without a clinical history of stroke or physical deficits suggestive of a focal lesion.^{14,15} In contrast, focal neurological changes may occur in the absence of any identifiable neurologic disease, especially in the elderly.¹⁶ Also, the elderly often have focal deficits due to neurological lesions that are unrelated to brain damage (eg. plantar extensor response due to cervical spondylosis). It is well known that CT does not demonstrate the presence of small infarcts in all cases of MID and that CT is not as sensitive as MRI for identifying infarcts. CT is, nevertheless, more readily available than MRI and does provide helpful information.

Among the remaining cases in whom CT contributed to the diagnosis, the patient who presented with psychosis, dementia and a movement disorder characterized by dyskinesia provides a good example of the important role of CT in the evaluation of cognitively impaired patients. CT showed bilateral basal ganglia calcification. This disorder has been associated with psychotic behavior, dementia and a movement disorder and has been reported to respond to lithium.¹⁷ Basal ganglia calcification is, therefore, a potentially treatable disorder which could only have been identified by appropriate neuroimaging. Similarly, in the patient with suspected Huntington's disease, the CT was an important factor in making a diagnosis of senile chorea.

In conclusion, it must be stressed that our results are based upon findings in the setting of a specialized neurobehavioural unit and may, therefore, not generalize to other settings such as general medical and psychiatric units. Our findings do, nevertheless, raise the question whether CT can be used in a

more cost effective manner if it is restricted to those patients in whom the clinical evaluation is insufficient to make a diagnosis despite a detailed neurobehavioural assessment. It must be emphasized, however, that well designed prospective studies are needed to confirm our findings and also to determine whether they apply to other clinical settings.

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REFERENCES

1. Fox J. Use of CT scan in senile dementia. *J Neurol, Neurosurg Psychiatry* 1975; 38: 948-953.
2. Dietch JT. CT scanning in cases of dementia. *West J Med* 1983; 138: 835-837.
3. Lotz PR. Neurologic disorders: The many new uses of CT. *Geriatrics* 1985; 40: 40-53.
4. Wells C. Chronic brain disease: An overview. *Am J Psychiatry* 1978; 135: 1-12.
5. Cummings JL, Benson DF. *Dementia: A clinical approach*. Butterworths, 1983.
6. Antiplatelet Trialists' Collaboration. *Br Med J* 1988; 296: 320-331.
7. Martin DC. Clinical prediction rules for CT scanning in senile dementia. *Arch Int Med* 1987; 147: 77-80.
8. Larson EB. Diagnostic evaluation of 200 elderly outpatients with suspected dementia. *J Gerontol* 1985; 40: 536-543.
9. The investigation of dementia. *Med Aust* 1976; 2: 397-398.
10. Adams RD, Victor M. *Principles of neurology*. McGraw-Hill, 3rd Edition, 1985.
11. McKahn G, Drachman D, Folstein M, et al. Clinical diagnosis of Alzheimer's disease: report on NINCDS-ADRDA work group under Auspices of Department of Health and Human Services Task Force on Alzheimer's disease. *Neurology* 1984; 34: 939-944.
12. Hashinski VC. Cerebral blood flow in dementia. *Arch Neurol* 1975; 32: 632-637.
13. Rosen WG et al. Pathologic verification of ischemic score in differentiation of dementias. *Ann Neurol* 1980; 7(5): 486-488.
14. Jorgensen L. Ischemic cerebrovascular disease in an autopsy series. Part 1. *J Neurol Sci* 1966; 3: 490-509.
15. Fisher CM. Lacunes: small deep cerebral infarcts. *Neurology* 1965; 15: 76-80.
16. Drachman DA. Neurological evaluation of the elderly patient. In: Albert ML, ed. *Clinical Neurology of Aging*. Oxford University Press 1984.
17. Munir KM. The treatment of psychotic symptoms in Fahr's disease with lithium carbonate. *J Clin Psychopharmacol* 1986; 6: 36-38.