Occasional Review

Computed tomography in the investigation of dementia

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Abstract

To assess the value of computed tomography in investigating patients with dementia, 500 consecutive patients presenting with a provisional clinical diagnosis of dementia of recent onset were reviewed and the results analysed in a computer. Most patients had either cerebral atrophy or infarction, but 82 patients had a normal scan and 42 others had tumours. More than 10% of all patients, including 5% with no other symptoms or signs, had a treatable lesion. Various associated symptoms and signs were useful pointers to such a treatable lesion and clearly indicated computed tomography. Accurate diagnosis is the cornerstone of proper management, and if a few patients with treatable lesions can be identified then the benefits to all concerned may be incalculable.

Introduction

The incidence of dementia has been quoted differently, ranging from one in 20 by the age of 70¹ to one in six over the age of 65.² It is less common, but perhaps more serious, in younger age groups. It is said that those who are affected survive only a third as long as healthy people of the same age and sex,² and in the process both the family and the medical services become severely strained and the economy burdened. It has been calculated, for example, that by 1988 a health district of 250 000 people will have to provide services for 3000 elderly people with dementia.³ Alzheimer's disease is thought to be the most frequent cause of dementia, accounting possibly for half the

cases, and it may be the fourth or fifth most common cause of death in the United States.⁴ In a typical patient with Alzheimer's disease the personality and insight are preserved in the early stages but there is loss of memory and intelligence.⁵ The onset is insidious.

Cerebrovascular disease, and a combination of this and Alzheimer's disease, is thought to account for most of the rest of the cases of dementia. The clinical course is different and is said to show an abrupt onset, remissions and exacerbations, and focal signs. But the presentation is seldom so clear cut.⁴ The term "multi-infarct dementia" is preferred to indicate the association with hypertension or extracerebral vascular disease, or both, rather than "atherosclerosis," which implies disease of the small intracerebral vessels.⁶ Many other less common causes of dementia have been quoted, including alcoholism, Huntington's chorea, depression, normal pressure hydrocephalus, hypothyroidism, anaemia, liver disease, colloid cyst, and intracranial tumours.⁷⁻⁸

Clearly, it may be difficult to differentiate those possible causes clinically. Nevertheless, an accurate diagnosis is essential, both for managing the patient and for informing the family, as this may have serious implications for them and economic consequences for the State. Computed tomography of the head may be regarded as one part of the diagnostic range, particularly for younger patients and for those with a recent onset of dementia. At Frenchay Hospital we have been accepting such patients from hospital consultants in the south-west region since our scanner was installed in 1974. Almost 10% of new patients for computed tomography are referred with a provisional diagnosis of dementia.

Methods

The clinical features and brain scan findings were reviewed in 500 consecutive patients referred to this neuroradiological unit for investigation of dementia over 18 months. Patients with predominantly psychotic features were excluded. We accept requests for

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brain scans from a wide range of clinical specialists but not from general practitioners. The unit serves a population of around two and a quarter million people.

The clinical features relating to each patient were extracted from letters of referral, or hospital notes, or both. The following data, when available, were coded for each patient: age, sex, type of referring specialist, features of dementia, associated physical features, results of computed tomography, and value of contrast enhancement (if given). Table I summarises the coding system. Because of the nature of this study no data were available on other possible causes of dementia that were not shown by computed tomography—for instance, hypothyroidism and anaemia. Patients were grouped according to

TABLE I—Data gathered on each patient and codes used to record this

Name and No	Sex	Age	Referring physician (code)	Dementia (code)	Other features (code)	Computed tomographic findings (code)	Contrast enhanced (code)
1 Psychiatry	2 Neurology	3 Neurosurgery	4 General physician	5 Geriatrics	6 Other	1 Intellectual	5 Speech disorder
2 Memory and learning	3 Confusion	4 Orientation	1 Dementia only	2 Dementia and headache	3 Dementia and focal neurological signs	4 Dementia and extra pyramidal signs	5 Dementia and "vascular" episodes
6 Dementia and papilloedema	7 Dementia and ataxia	8 Dementia and seizures	1 Normal	2 Mild	3 Moderate	4 Severe	5 Hydrocephalus
6 Tumour—specify	7 Old	8 Recent	9 Multiple	10 Unilateral	11 Bilateral	1 No	2 No additional information
3 Additional information							

age as follows: 54 years and under, 55-64, 65-74, 75 and over. A simple classification of dementia was used (table I). The coding was chosen to match the observations by the specialists in referral letters or hospital notes. Many patients had no symptoms or signs other than deteriorating mental function, but others showed various physical features and were coded as in table I. Any patient might have several codes in each group.

Results of computed tomography—A simple system was used to code various findings, including a broad range of cerebral atrophy, hydrocephalus, tumours, subdural haematomas, and infarcts.

Contrast enhancement—Some patients were given contrast medium, and the value of this technique was recorded.

All scans were performed on an EMI 1007 brain scanner using 210 mm wedges. Most patients had four or five scan pairs, each slice being nominally 10 mm thick and orientated at 15° to the base line. A Gamma 11 computer (Digital Equipment Corporation PDP 11/34), installed in this department for dynamic isotope scanning, was separately programmed to store and analyse the data for this study.

Results

AGE, SEX, AND REFERRAL

The entire series of 500 patients was analysed by the four age groups. The sex ratio was analysed and the full data for age and sex distribution are given in table II, together with the source of referral.

RESULTS OF COMPUTED TOMOGRAPHY

Table III shows the findings according to age and sex and the incidence of the various diagnoses in each age group. Eighty-two patients (16%) had normal scans. The degree of atrophy was simply recorded as mild, moderate, or severe according to generally accepted

TABLE II—Distribution of patients by age and sex for each referring specialty

Specialty	Age (years)					Sex		Total No
	< 54	55-64	65-74	≥ 75	Mean age	M	F	
Psychiatry	17	47	79	67	69	77	133	210
Neurology	32	36	39	8	60	66	49	115
General physician	4	23	42	16	68	49	36	85
Geriatrics	2	3	19	37	78	24	32	56
Neurosurgery	2	3	7	3	66	9	6	15
Others	2	3	13	1	68	11	8	19
Total	57	112	199	132	68	236	264	500

TABLE III—Computed tomography findings, showing incidence of each condition, with age and sex distribution

	No (%) of patients	M/F ratio	Age (years)				
			Mean	< 54	55-64	65-74	≥ 75
Normal	82 (16.4)	37/45	57.1	32	27	16	7
Atrophy:							
Mild	122 (24.4)	50/72	68.2	11	30	57	24
Moderate	68 (13.6)	31/37	72.1	2	11	32	23
Severe	2 (0.4)	0/2	69.0	0	0	2	0
Infarct:							
Old	28 (5.6)	14/14	70.5	2	7	8	11
Recent	19 (3.8)	10/9	68.4	1	5	7	6
Multiple	127 (25.4)	66/61	71.7	4	21	52	50
Tumour	42 (8.4)	23/19	66.3	4	11	20	7
Hydrocephalus	6 (1.2)	2/4	63.6	1	0	3	2
Subdural haematoma	4 (0.8)	3/1	77.0	0	0	2	2
Total	500 (100)	236/264	67	57	112	199	132

criteria. The appearances of multiple infarction fell into three broad groups which showed some overlap: (1) typical infarcts (51 patients); (2) "holes" (41 patients); (3) periventricular lucencies (35 patients) (figure).

Six of the 42 patients with tumours had hydrocephalus. Eighteen had moderate to high grade gliomas; seven of these were in the corpus callosum (figure). Seventeen patients had metastatic deposits (one in the corpus callosum). Two had astrocytomas, and there was one case each of colloid cyst, craniopharyngioma, meningioma, ependymoma, and pineal tumour.

Of the four patients with tumours (mean age 73) who were alive and well at the latest follow-up, two had metastatic deposits, of whom one had surgery, and two had benign tumours that were treated symptomatically. The average duration of survival from diagnosis for these four patients was 21 months (range 10-25).

The other 38 patients with tumours (mean age 65.5) have since died. Many had a worthwhile initial response to treatment. Seven had no treatment (mean age 71.7) and survived for an average of three months. Twenty-nine patients were treated with steroids (mean age 64.2) and also survived for an average of three months. Six of the 38 patients (mean age 63.5) had surgery and survived for an average of 5.5 months. One patient aged 73 had radiotherapy and survived for 14 months. Several patients had more than one form of treatment.

A comparison of the duration of survival of deceased patients who had tumours with age failed to show a consistent pattern, possibly because the numbers in some age groups were small: aged under 54, 3.5 months (four patients); aged 55-64, 2.5 months (11 patients); aged 65-74, four months (19 patients); aged 75 and over, one month (four patients); all deceased patients, four months (38 patients).

Apart from the six patients with hydrocephalus and a tumour, six others had hydrocephalus. One, aged 76, had hydrocephalus and basilar invagination and aqueduct stenosis; he was much improved after the insertion of a shunt. Three patients (aged 35, 68, and 75) had low pressure hydrocephalus confirmed by other tests and improved after the insertion of a shunt. (The patient aged 75 has since died from other causes.) A further patient aged 69 had low pressure hydrocephalus confirmed but was not improved by the insertion of a shunt. One patient aged 71 had probable low pressure hydrocephalus but was not fit for further evaluation or treatment.

Four patients had chronic subdural haematoma: unilateral in three and bilateral in one. Three patients (aged 74, 75, and 87) showed appreciable improvement after removal of the clot and are still alive and well. The other patient (aged 72) improved initially but died four days later.

Altogether 153 patients were given contrast medium. In 94 of

CT scans showing multiple infarcts: (a) Bilateral infarcts in posterior cerebral area; (b) multiple "holes" in both hemispheres; (c) periventricular lesions adjacent to both lateral ventricles. Glioma: (d) Contrast enhanced scan showing glioma of anterior corpus callosum.

these the medium was given to assess further a focal lesion seen on the plain scan. This provided positive information in 52 (55.3%), including all the patients with tumours. In 59 contrast medium was unhelpful or simply provided a further basis on which to exclude lesions.

OTHER DIAGNOSES

Thirty-one patients (16 of whom were men) had a history of serious alcohol abuse (6%). Eight of these patients had infarcts; however, none had treatable structural lesions. Nineteen patients had a history of Parkinson's disease. Of these, one had a normal scan, eight had multiple infarcts, and 10 showed atrophy. None of these patients had calcification of the basal ganglia, although two without Parkinson's disease did. Three patients had a history of Huntington's chorea: one had a normal scan and two had atrophy.

DIAGNOSTIC POINTERS

We analysed in detail the different types of dementia recorded for these patients and also other physical features to find characteristics of the patients' clinical presentation that might help to indicate to the referring physician that a particular patient may have dementia associated with some localised and possibly treatable lesion. To this end the diagnoses made by computed tomography were allocated to one of two groups: potentially treatable structural lesions and untreatable lesions. A diagnosis by computed tomography of tumour, hydrocephalus, or chronic subdural haematoma was classified as a treatable lesion; old, recent, or multiple infarcts, atrophy, and normal scans were classified as untreatable. If a patient had atrophy and a treatable lesion then for the purpose of regrouping the treatable lesion took precedence.

All 500 patients were assigned to the two groups according to the principles outlined above. Fifty-two patients (10%) had a potentially

treatable structural lesion, while 448 (90%) had untreatable lesions. Using these data we reanalysed the types of dementia and other physical features to evaluate their usefulness as diagnostic pointers (table IV). Only speech disorder, headache, focal signs, and papilloedema produced a better pick up rate than scanning all 500 patients indiscriminately.

TABLE IV—Incidence of potentially treatable and untreatable structural lesions for all groups according to age, sex, type of dementia, and associated physical features

	Total No	No (%) with potentially treatable structural lesion	No (%) with untreatable lesions
Men	236	28 (11.9)	208 (88.1)
Women	264	24 (9.1)	240 (90.9)
Age			
≤54	57	5 (8.8)	52 (91.2)
55-64	112	11 (9.8)	101 (90.2)
65-74	199	26 (13.1)	173 (86.9)
≥75	132	10 (7.6)	122 (92.4)
Type of dementia			
Intellectual	245	23 (9.4)	222 (90.6)
Memory and learning	190	19 (10)	171 (90.0)
Confusion	182	27 (14.8)	155 (85.2)
Disorientation	56	8 (14.3)	48 (85.7)
Speech disorder	53	10 (18.9)	43 (81.1)
Mood change	130	11 (8.5)	119 (91.5)
Unclassified	14	1 (7.1)	13 (92.8)
Other features			
Dementia only	327	17 (5.2)	310 (94.8)
„ and headache	30	9 (30.0)	21 (70.0)
„ and focal signs	74	22 (29.7)	52 (70.3)
„ and extrapyramidal signs	21	0	21 (100)
„ and vascular episodes	20	1 (5.0)	19 (95.0)
„ and papilloedema	3	3 (100)	0
„ and ataxia	38	6 (15.8)	32 (84.2)
„ and seizures	21	3 (14.3)	18 (85.7)
Total No	500	52 (10.4)	448 (89.6)

A striking finding in this analysis, and also perhaps a compelling reason to do scanning in demented patients, was the incidence of a potentially treatable structural lesion in patients without any other symptoms or signs. Out of 327 patients with dementia only, 17 had a potentially treatable structural lesion (5%). The following features recorded on the other patients showed a useful correlation with the subsequent demonstration of a potentially treatable structural lesion.

Headache—Of the 30 patients with headaches, nine had a potentially treatable structural lesion. Of these, eight had tumours and one a subdural haematoma; (two patients had hydrocephalus associated with tumours).

Focal neuro signs—Of the 74 patients with focal neuro signs, 22 had a potentially treatable structural lesion, including 20 with a tumour and two with a subdural haematoma. A total of 39 patients with focal signs had infarcts (52.7%).

Papilloedema—Three patients had papilloedema and tumours.

Vascular episodes correlated reasonably well with the occurrence of infarction. Of the 20 patients with this symptom, nine had evidence of infarcts, but in only two of these were they multiple.

Discussion

Computed tomography, and before that pneumoencephalography, allow us to study the morphology of the living brain. We know that ventricular size and sulcal width increase with advancing age even in normal people and that the borderline between so called cerebral atrophy and normal age changes is indistinct and therefore subject to observer bias on computed tomography. Computed tomography, as opposed to pneumoencephalography, however, allows us to examine the patient as an outpatient and assess the appearance of the living brain without upsetting the patient, so that the degree and often the cause of the atrophy and other intracranial lesions may be seen.

It is difficult to compare our results with those of Marsden and Harrison,⁷ or Smith and Kiloh.⁸ Both of these studies were done mainly in the era before computed tomography when more invasive techniques were required to exclude structural lesions. Clearly, any retrospective study like ours may

have some selection in the population under review, since the patients are referred for investigation by computed tomography. Nevertheless, we believe that our study looks at a less selected group than the previous studies because assessment by computed tomography is noninvasive, allowing investigation of a greater number and broader range of people with dementia than would have been possible using invasive techniques.

In Smith and Kiloh's series⁸ the mean age of patients was 57.7, almost 10 years lower than in our patients. In Marsden and Harrison's series⁷ the mean age was 61. The preponderance of women in the age group 75 and over in our series reflects the pattern in the general population.

The appearance of a normal looking brain at computed tomography (16% in our series) in a demented patient should prompt the search for a potentially treatable cause elsewhere—for instance, depression, hypothyroidism, anaemia—though not all demented patients have cerebral atrophy, with the corollary that not all patients with atrophy are demented.⁹⁻¹³ There is a broad relation, however, between increasing ventricular dilatation and increasing intellectual impairment.¹⁴ In our study, therefore, we adopted the simple classification for cerebral atrophy seen at computed tomography as described. Only two patients were classified as having severe atrophy (both were alcoholics) and this probably relates to our policy of preferring to accept patients with a recent onset of dementia—that is, measured in months rather than years. Our "cerebral atrophy" group is composed of patients with appearances on brain scan of atrophy but no recognisable infarcts. How many of these have Alzheimer's disease is not known, as no brain biopsy specimens were taken, but postmortem examination elsewhere of patients with dementia has shown characteristic features in half of all cases.¹⁵ Thus we assumed that most, if not all, of these patients have Alzheimer's disease.

Our analysis showed clear evidence of structural infarction in 35% of patients, and these showed typical features of old, recent, or multiple infarcts. The multiple grouping included those with appearances of typical infarcts, and of others with "holes," and of some with periventricular lesions, but there was a considerable overlap in these three groups. The last group have appearances in common with those described as watershed infarctions.¹⁶ Smith and Kiloh found that "atherosclerotic" disease was the source of dementia in 13.6% of patients,⁸ and Marsden and Harrison in 9.5%,⁷ but these figures are apparently based on arteriographic evidence in one series and on clinical rather than imaging data in the other series. The incidence of infarction in our patients agrees with postmortem studies. Clearly, structural evidence of infarction might be important in the future in relation to possible advances in prevention and treatment.

The incidence of serious alcohol abuse in Smith and Kiloh's (7%)⁸ and Marsden and Harrison's (7%)⁷ series was similar to that in ours.

In our series the higher average age of patients may have accounted for some of the difference in the incidence of potentially treatable structural lesions of 10% (Smith and Kiloh reported 7%⁸) and explain why our series contained 17 cases of metastases. The incidence of potentially treatable structural lesions in Marsden and Harrison's series was 16%, probably reflecting the selection processes in referral to such a highly specialised unit as that at Queen Square. Our experience has shown that most demented patients with primary brain tumours or metastases do not survive very long and thus such lesions might not therefore be considered as "treatable." On the other hand, some symptomatic improvement is often possible, and in any case these conditions should not go undiagnosed. Recognising hydrocephalus is important, especially as the response to treatment of normal pressure hydrocephalus in this series was encouraging. Our experience with chronic subdural haematoma was similar. Contrary to the experience of others the number of older patients who have responded well to treatment of these lesions was encouraging.

Neither of the two studies quoted made any detailed attempt

to correlate clinical features to find helpful pointers to diagnosis. About 65% of our patients presented with dementia and no other recorded symptoms or signs. Nevertheless, 5% of these had a potentially treatable structural lesion. As a corollary, 30% of those with tumours also presented with dementia only. Those are two good reasons for using computed tomography. Differentiating between those with a potentially treatable structural lesion and those without was difficult. Headache, focal signs, papilloedema, and speech disorders, however, were helpful diagnostic pointers and should normally be a sound indication for a brain scan in a patient with recent onset of dementia. Contrary to the findings of Smith and Kiloh,⁸ we did not find age of help in the diagnosis. So called "vascular episodes" were not reliable for the diagnosis of multiple infarcts. Of all the patients with headache, 30% had a potentially treatable structural lesion, but 8% of the patients with normal scans also complained of headache.

Our experience with contrast enhancement suggests that its use in patients with dementia should normally be limited to cases where a focal lesion is present on the plain scan.

It is clearly impracticable to scan all patients with dementia. We think, however, that the results of this study suggest that most patients with any of the diagnostic pointers listed above, and who have been demented for more than one month and less than one year should have a brain scan. For the remainder who have no such signs the decision whether to scan is not so clear and will depend on the merits of each case and the local resources. The low cost and simplicity of computed tomography compare favourably with the cost of care in an institution.¹⁷ Accurate diagnosis may identify a few patients with treatable lesions and the benefits to all concerned may be incalculable.

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References

- 1 Roth M. Epidemiological studies. In: Katzman R, Terry RD, Bick KL, eds. *Alzheimer's disease: senile dementia and related disorders*. New York: Raven Press, 1978:337.
- 2 Terry RD. Dementia: a brief and selective review. *Arch Neurol* 1976;**33**: 1-4.
- 3 College Committee on Geriatrics of the Royal College of Physicians. Organic mental impairment in the elderly. *J R Coll Physicians Lond* 1981;**15**:141-67.
- 4 Katzman R, Karasn TB. Differential diagnosis of dementia. In: Fields WS, ed. *Neurological and sensory disorders in the elderly*. New York: Stratton Intercontinental Medical Book Corporation, 1975:103-34.
- 5 Blessed G, Tomlinson BE, Roth M. The association between quantitative measures of dementia and of senile change in the cerebral grey matter of elderly subjects. *Br J Psychiatry* 1968;**114**:797-811.
- 6 Hachinski VC, Lassen NA, Marshall J. Multi-infarct dementia. *Lancet* 1974;ii:207-9.
- 7 Marsden CD, Harrison MJG. Outcome of investigation of patients with presenile dementia. *Br Med J* 1972;ii:249-52.
- 8 Smith JS, Kiloh LG. The investigation of dementia: results in 200 consecutive admissions. *Lancet* 1981;ii:824-7.
- 9 Huckman MS, Fox J, Topel J. The validity of criteria for the evaluation of cerebral atrophy by computed tomography. *Radiology* 1975;**116**: 85-92.
- 10 Wells CE, Duncan GW. Dangers of over reliance on computerised tomography. *Am J Psychiatry* 1975;**134**:811-3.
- 11 Claveria LE, Moseley IF, Stevenson JF. The clinical significance of "cerebral atrophy" as shown by CAT. In: du Boulay GA, Moseley IF, eds. *First European seminar on CAT in clinical practice*. Berlin: Springer Verlag, 1977:213-7.
- 12 Roberts MA, Caird FI, Steven JL, Grossart KW. Computerised axial tomography and dementia in the elderly. *J Neurol Neurosurg Psychiatry* 1976;**39**:909-15.
- 13 Fox JH, Topel JL, Hackman MS. Use of computerised tomography in senile dementia. *J Neurol Neurosurg Psychiatry* 1975;**38**:948-53.
- 14 Roberts MA, Caird FI. Computerised tomography and intellectual impairment in the elderly. *J Neurol Neurosurg Psychiatry* 1976;**39**:986-9.
- 15 Anonymous. Alzheimer's disease. *Br Med J* 1980;**281**:1374.
- 16 Wodarz R. Watershed infarctions and computed tomography. A topographical study in cases with stenosis or occlusion of the carotid artery. *Neuroradiology* 1980;**19**:245-8.
- 17 Millard PH. Last scene of all. *Br Med J* 1981;**283**:1559-60.

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