PAPERS AND SHORT REPORTS

Value of computed tomography in patients with stroke: Oxfordshire Community Stroke Project

PETER SANDERCOCK, ANDREW MOLYNEUX, CHARLES WARLOW

Abstract

The usefulness of computed tomography (CT) was assessed in 325 consecutive patients with a "clinically definite first stroke" from a community stroke register. CT detected five "non-stroke" lesions (two cerebral gliomas, one cerebral metastasis, and two subdural haematomas), a frequency of 1.5%. Five patients were identified with cerebellar haemorrhage, but only one survived long enough to have a CT scan. CT was useful in excluding intracranial haemorrhage as the cause of the stroke in four patients receiving anticoagulants and seven receiving antiplatelet treatment; it showed intracranial haemorrhage in one patient taking aspirin. Forty six patients were in atrial fibrillation at the time of their stroke; four had intracranial haemorrhages and three had haemorrhagic cerebral infarcts. Nineteen patients with presumed ischaemic minor stroke were considered suitable for carotid endarterectomy; CT showed small haemorrhages in two.

The CT scan provides very useful information in a minority (up to 28%) of patients with first stroke, who can be selected on quite simple criteria: (a) doubt (usually because of an inadequate history) whether the patient has stroke or a treatable intracranial lesion; (b) the possibility of cerebellar haemorrhage or infarction; (c) the exclusion of intracranial haemorrhage in patients who either are already taking or likely to need anti-haemostatic drugs or are being considered for carotid endarterectomy; (d) if the patient deteriorates in a fashion atypical of stroke.

Introduction

There are several possible reasons for performing computed tomography (CT) of the head in patients with acute stroke—namely, to establish the diagnosis; to identify types of stroke amenable to surgery; to exclude intracranial haemorrhage; and in spontaneous subarachnoid haemorrhage (see Appendix). Scanning *all* patients with stroke, however, would be prohibitively expensive; some 96 000 patients have their first stroke in England and Wales every year,¹ which would require the equivalent of about 18 CT scanners at a capital cost of about £6.7m, each working 200 hours a month at a yearly cost of about £1.35m. Although such a policy might be the ideal, it should be far more economical and practical—and perhaps very little less effective—to select for scanning only those patients whose management is likely to be influenced by the investigation. Any selection criteria would best be derived from a sample of all patients with stroke, and not just those referred for hospital admission. The Oxfordshire Community Stroke Project¹ provides such a sample and, since most of the patients have had a CT scan, we are in a good position to define criteria which identify the patients most likely to benefit from it. In this paper we consider the possibilities listed above except spontaneous subarachnoid haemorrhage.

Patients and methods

The Oxfordshire Community Stroke Project is a register of all new cases of stroke and transient ischaemic attack occurring in a defined population of about 103 000 residents of Oxfordshire. The study population is not a random sample but is similar in age and sex distribution to the population of Oxfordshire.¹ Ascertainment of all new cases of stroke in this population is complete, and therefore cases of stroke in the study population are likely to be an unbiased sample of all strokes in Oxfordshire.¹

Stroke is defined as acute loss of focal and at times global (applied to patients in deep coma and to those with subarachnoid haemorrhage) cerebral function, the symptoms lasting more than 24 hours or leading to death, and with no apparent cause other than that of vascular origin.²

Methods of identification and assessment of patients and definitions of the different pathological types of stroke are described elsewhere.¹ The patients were assessed by a study neurologist as soon as possible after the onset. Details of the clinical history were confirmed by

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interview with a witness if possible (often by telephone). Any medical history was later verified by examining all available medical records, including the general practitioners' notes.

We diagnosed stroke in two stages: we made a first, *clinical* diagnosis before the results of investigations were known and then a second, *final* diagnosis once investigations were complete. The clinical diagnosis was either "clinically definite stroke" or "clinically not stroke." The final diagnosis was either "first stroke" or "not first stroke."

We made every effort to perform a CT scan, primarily to determine the pathological type of stroke, in cases of "clinically definite first stroke" and to obtain a necropsy in fatal cases.

Results

The study registered 736 patients between 1 November 1981 and 31 October 1983. The initial clinical diagnosis was clinically definite first stroke in 325 and not first stroke in 411 (fig). The reasons for the

came to necropsy, review of the case histories indicated that stroke was an extremely unlikely diagnosis. Thus the proportion of patients who were diagnosed as clinically not stroke who were found to have cerebral tumour or subdural haematoma was 11 out of 132 (8.3%) when only CT proved lesions were included and 16 of 132 (12.1%) when all suspected lesions were included.

Three patients who had initially been classed as clinically not first stroke were found to have vascular lesions on investigation and were reclassified as first strokes (two had cerebral infarction diagnosed by CT, and one was found to have a pontine haemorrhage at necropsy). Clinical data were incomplete in all three because of coma, confusion, or dysphasia.

CT FINDINGS IN PATIENTS WITH "CLINICALLY DEFINITE FIRST STROKE"

The table gives the results of CT and necropsy in the 325 patients who presented with a sudden focal neurological deficit and were diagnosed as clinically definite first stroke. Of these patients, 288 (89%)

Initial and final diagnoses in 736 consecutive patients registered.

latter were: previous stroke (176 cases), symptoms lasted less than 24 hours (74), non-stroke condition (132), and referred in error (29)—that is, as a result of inaccuracies in coding hospital discharge and death certificate diagnoses.

PATIENTS DIAGNOSED AS "CLINICALLY NOT STROKE"

The 132 patients classed as clinically not stroke had conditions which could be diagnosed mainly on clinical grounds. The commonest diagnoses were confusional state or coma due to a toxic or metabolic condition—for example, hypoxia, infection, electrolyte imbalance, and congestive cardiac failure. If such clinical diagnoses were supported by the results of appropriate biochemical or radiological investigation CT was not performed. In many cases several factors coexisted, and several patients had also had previous strokes. We diagnosed various neurological conditions, such as Parkinson's disease, Bell's palsy, and multiple sclerosis rather less often (a full list of the diagnoses made is available on request).

Sixteen of the 132 patients presented with progressive neurological signs and symptoms highly suggestive of a non-stroke space occupying lesion. Eleven of the 16 had a CT scan; it showed a cerebral tumour in seven and a subdural haematoma in four. After review of the histories the clinical features in all 11 cases were still not considered in any way suggestive of clinically definite stroke. Five of the 16 patients with suspected non-stroke lesions did not have CT. All five had histologically proved disseminated malignancy from primary tumours outside the central nervous system and developed slowly progressive focal neurological symptoms and signs suggestive of cerebral metastases. Although none of these five had a CT scan or

were seen by a study neurologist, and for 233 (72%) a witness account was obtained. Of the 323 patients with a final diagnosis of first stroke, 13 (4%) had a CT scan within one day, 44 (14%) within three days, 111 (34%) within seven days, 166 (51%) within two weeks, and 202 (63%) within three weeks of the onset of the stroke.

Results of CT scan and necropsy in 325 cases with an initial diagnosis of "clinically definite first stroke"

No (%) of cases		
Cerebral infarction		242 (74.5)
Diagnosed by CT scan { CT normal/atrophy CT focal infarction*	106 119	225
Diagnosed at necropsy		17
Intracranial haemorrhage		46 (14.1)
Primary intracerebral haemorrhage:		
Diagnosed by CT scan	16	27
Diagnosed at necropsy†	11	
Subarachnoid haemorrhage:		
Diagnosed by lumbar puncture only	3	19
Diagnosed by lumbar puncture, CT scan, angiography and/or at necropsy	13	
Diagnosed at necropsy only	3	
Pathological type of stroke not known‡		32 (9.8)
Non-stroke lesion		5 (1.5)
Primary cerebral glioma	2	
Subdural haematoma	2	
Cerebral metastasis	1	
Total		325 (100.0)

*Further two cases initially diagnosed as clinically not stroke were later reclassified as first stroke after CT showed focal infarct.

†One patient initially diagnosed as clinically not stroke was later found to have pontine haemorrhage at necropsy.

‡No CT scan or necropsy performed.

Non-stroke lesions

Only five patients (1.5%) had a non-stroke lesion detected by CT. The 95% confidence intervals of this point estimate were 0.2-2.8%. Three patients (one with cerebral glioma, one with cerebral metastasis, and one with subdural haematoma) were able to give a clear account of a sudden onset, though one patient was mildly dysphasic. The clinical details of the two others are given below.

Case 1 was an 86 year old woman with longstanding severe dementia who had been chairbound with arthritis for four years. She was sitting in a chair at 11 am when, according to her daughter, she suddenly slumped to one side. Her condition worsened over the next 24 hours, and she became doubly incontinent and hemiplegic. She was admitted to hospital. A study neurologist examined her on the third day of the illness, when she was lying with her eyes open, making unintelligible noises. She had a left facial weakness and a left hemiplegia. CT showed bilateral subdural haematomas. These were not drained because of her age and very poor general condition.

Case 2 was an 86 year old woman who lived in an old people's home. She had had several episodes of paranoid depression and had mild dementia. According to the staff she was found one morning "stiff all down the left side," having been well the previous day. On admission to hospital she was drowsy, the eyes were deviated to the right, there was a possible left homonymous hemianopia and a left hemiplegia. The CT scan showed a right frontal tumour. The matron of the home was interviewed and said that the patient's mental condition had deteriorated rapidly over the six weeks before the "stroke" but she had not noticed any weakness of her left arm or leg. The patient was given dexamethasone for a short while without any effect and died three weeks later. Necropsy showed a right frontal glioma.

The discovery of these five non-stroke lesions led to a change in management in three cases; surgical drainage was performed for one of the subdural haematomas, and two patients with gliomas were given dexamethasone. The first patient survived, and the two others died.

Non-stroke lesions in patients with "progressing stroke"

Of the 325 patients with clinically definite first stroke, 203 had a neurological deficit which was either present on waking or reached its maximum within one hour. Four of the 203 (2%) had non-stroke lesions. Seventy seven patients had an acute neurological deficit which took more than one hour, but less than seven days, to reach its maximum. Only one of the 77 (1.3%) proved to have a non-stroke lesion. The remaining 45 patients, most of whom had very mild strokes and were referred some time after the event, could not recall the exact mode of onset. Patients with clinically definite first stroke whose deficit progressed were no more likely to have a non-stroke lesion than patients whose deficit reached its maximum in less than an hour.

Cerebellar and other surgically treatable forms of stroke

Five of the 28 primary intracerebral haemorrhages were cerebellar, but unfortunately only one of these was detected in life by CT. Of the four patients whose haemorrhages were detected at necropsy, one was dead on arrival in the casualty department, and although the three others survived at least eight hours from onset, none was fit to be transferred from our main stroke admitting hospital (John Radcliffe II) to the Radcliffe Infirmary for a CT scan. In addition, there were five patients with cerebellar infarction, none of whom developed obstructive hydrocephalus or any other indication for neurosurgical treatment. None of the haemorrhages in other sites was treated surgically.

Exclusion of intracranial haemorrhage

Antithrombotic drugs—Sixteen patients were receiving anti-thrombotic drugs (five anticoagulants, 11 antiplatelet agents) at the time of their first stroke, of whom one (taking aspirin) had an intracranial haemorrhage. CT excluded haemorrhage in four patients receiving anticoagulants and seven taking antiplatelet agents. Haemorrhage could not be excluded in four who did not have CT or necropsy performed.

Atrial fibrillation—Forty six patients were in atrial fibrillation at the time of their first stroke. Four had primary intracerebral haemorrhages (two shown by CT and two found at necropsy) and three had haemorrhagic cerebral infarcts (one shown by CT and two found at necropsy). Intracranial haemorrhage and haemorrhagic

infarction could not be excluded in 11 who did not have either CT or necropsy performed. The indiscriminate use of anticoagulants would therefore have been definitely unsafe in at least 7 (15%) and possibly unsafe in 18 (39%) of the patients in atrial fibrillation at the time of their first stroke.

Carotid endarterectomy—Nineteen patients with presumed ischaemic minor strokes who had recovered sufficiently to be considered as potential candidates for carotid endarterectomy had a CT scan before carotid angiography. Two of the 19 had small haemorrhages in the basal ganglia and therefore, as a result of the CT scan findings, did not undergo carotid angiography.

Discussion

The value of CT in patients with "stroke" depends on the exact definition of the term, the group of patients studied, and the experience of the person making the clinical diagnosis. The definition of stroke that we used² requires that any focal neurological deficit should be of "acute" onset. It is relatively easy to detect a focal neurological deficit but it may be difficult to know whether or not the onset was sudden, particularly if the patient is aphasic, confused, or comatose; if the deficit is progressing; or if no history at all is available. A good history and a witness's account of the event are crucial and may provide enough information to resolve any uncertainties. Allen found that in his hospital based series of 174 patients diagnosed as "stroke" by the admitting physician there were nine (5%) with non-stroke cerebral lesions.³ In eight of these the clinical history was "incomplete" because the patients were either comatose or dysphasic. The frequency of non-stroke lesions was much higher in the patients with incomplete histories (eight out of 60; 13.3%) than in those with complete histories (one of 114; 0.9%). In our series patients were classed as "clinically definite first stroke" or "not first stroke" on clinical grounds, before the result of the CT scan or necropsy was available. A witness was interviewed in 72% of cases. Of the 325 patients with clinically definite first stroke, only five (1.5%) were found by CT to have non-stroke lesions; in three of these the history was "incomplete," in one because of dysphasia and in the two others because of dementia. Our sample size was sufficient to say that, given similar conditions to our study, no more than about 3% of patients with clinically definite first stroke diagnosed by a competent neurologically trained physician are likely to harbour non-stroke lesions.

Patients with progressive neurological deficits are more difficult to assess and have been said to be more likely to have a non-stroke lesion. Weisberg and Nice reported that CT detected non-stroke lesions in 13 out of 30 consecutive patients admitted to their hospital with progressing stroke (a focal neurological deficit which progressed over two to 72 hours from onset) as compared with six out of 60 patients in whom the deficit reached its maximum in less than two hours.⁴ Of the 325 patients in our series with a clinically definite first stroke, 77 had a deficit which either progressed or fluctuated for more than one hour and less than seven days after its onset, of whom only one (1.3%) had a non-stroke lesion.

It is worth considering how the estimate of the proportion of patients who had space occupying lesions might have been altered by adding the cases of cerebral tumour and subdural haematoma found in patients diagnosed as clinically not stroke. We did not perform CT in every case so diagnosed as this was usually not necessary or appropriate—for example, in patients with proved diabetic hyperglycaemia whose non-focal neurological dysfunction (coma or confusion) cleared with correction of the metabolic disturbance. Similarly, we did not perform CT in patients with focal signs due to conditions which could be diagnosed on clinical grounds alone, such as Bell's palsy. There were, however, 16 patients in this group who had symptoms and signs suggestive of a non-stroke intracranial lesion, which was confirmed by CT in 11. Possibly the remaining five, who did not have CT scans, in fact had strokes (though this seemed highly unlikely from the clinical history). Since none had

clinically definite stroke, however, the final diagnosis in these cases is not relevant to the usefulness of CT in patients with clinically definite stroke.

The overall frequency of non-stroke lesions (1.5%) in patients with clinically definite first stroke from our community based series was much lower than that reported from the hospital based series of Kinkel and Jacobs (3.7%),⁵ Allen (5%),³ and Weisberg and Nice (17.7%),⁴ though it was higher than the frequency reported by Twomey (0.4%).⁶ Although Twomey found four patients with cerebral tumours (he did not include subdural haematomas) out of 1009 patients admitted to the department of geriatric medicine at Northwick Park Hospital with "vascular stroke," only 196 (19.4%) either had a CT scan or came to necropsy. It is therefore quite likely that not all tumours were detected. Two factors may account for the higher frequency reported by the first three studies. Series which include patients in whom the diagnosis of stroke is uncertain, or are based on poor clinical data, are likely to report a higher frequency of non-stroke lesions; only Allen described the "quality" of the clinical history on which the diagnosis of stroke had been based. Furthermore, patients admitted to hospital with "stroke" may not be typical of cases of stroke in general, since they may be admitted precisely because they have unusual clinical features such as raised intracranial pressure. The frequency of cerebral tumour and subdural haematoma is therefore likely to be higher in a hospital based series of patients with clinically definite stroke than in an unselected community sample.

Of all patients with primary intracerebral haemorrhage, it is those with cerebellar haemorrhage (or massive cerebellar infarction) who are most likely to benefit from CT, since timely detection can lead to lifesaving neurosurgery to evacuate the clot or shunt any accompanying obstructive hydrocephalus.⁷⁻⁸ Hospital based series suggest⁷⁻⁹ that it may not be an uncommon problem. McKissock *et al* estimated in a necropsy series that 8% of all primary intracerebral haematomas were cerebellar¹⁰; more recent studies have shown that 9-10% of all CT proved primary intracerebral haemorrhages are cerebellar.⁹⁻¹¹ In our series only one patient had a cerebellar haemorrhage detected in life by CT. None of the five patients with cerebellar infarction developed obstructive hydrocephalus. Cerebellar stroke is therefore relatively uncommon as a cause of first stroke (3.1% of all cases), but when it does occur and the patient is fit enough to reach an appropriate hospital CT is invaluable.

The clinical differentiation of cerebral infarction from primary intracerebral haemorrhage is unreliable¹²⁻¹⁴ even when clinical scoring systems are used.³⁻¹⁵ Furthermore, there may be no blood in the cerebrospinal fluid in patients with primary intracerebral haematoma.³⁻¹⁴

The ability of the CT scan to distinguish infarction from haemorrhage declines with time after the stroke, however, since haematomas gradually become isodense—and therefore undetectable.¹¹⁻¹³⁻¹⁶ Small haematomas may become isodense within five days of the stroke.¹⁶ Were we to reclassify any patient who had a CT scan more than two weeks after the stroke as pathological type not known, then the numbers (and percentages) of patients with each type of stroke would be: cerebral infarction 156 (49.3%), primary intracerebral haemorrhage 28 (8.7%), subarachnoid haemorrhage 19 (5.9%), and not known 120 (37.2%). Plainly such calculations affect only the proportions in each of the pathological subcategories of stroke and do not affect the differentiation of "stroke" from "non-stroke" lesions by CT.

The exclusion of intracerebral haemorrhage may, however, be necessary only in patients who are already taking anti-haemostatic drugs at the time of, or will need to take them after, their stroke and in patients who are being considered for carotid endarterectomy. In our series there were 16 patients who were taking antithrombotic, and therefore potentially antihaemostatic, drugs at the time of their first stroke in whom it was important to distinguish infarction from haemorrhage so that the drugs could be reviewed, stopped, or continued. Although most

patients had cerebral infarcts, there was one who definitely had an intracranial haemorrhage and four who might have had. Of greater numerical importance were the 46 patients in atrial fibrillation, since some doctors might want to anticoagulate at least some and possibly give aspirin to others. Four of these patients had a primary intracerebral haemorrhage and three a haemorrhagic cerebral infarct, and the use of anticoagulants would therefore have been unsafe in at least 15% of patients in atrial fibrillation at the time of their stroke. CT may also be used to exclude intracranial haemorrhage in patients with presumed ischaemic minor stroke who are fit for carotid angiography and carotid endarterectomy. This is a relatively infrequent indication for CT and is likely to vary widely throughout Britain.¹⁷ In our series CT usefully excluded haemorrhage in 17 and showed it in two patients with presumed ischaemic stroke who were considered suitable for carotid endarterectomy.

If we are prepared to "miss" up to 2.8% of non-stroke lesions in clinically definite first stroke, and if the results of our study are generalisable to other physicians managing patients with stroke, then the proportion who require CT as an aid in their management is at the most about 28%—3.1% with cerebellar strokes, 4.9% receiving antihaemostatic drugs, 14.2% possibly needing antihaemostatic drugs for secondary stroke prevention, and 5.8% who might be suitable for carotid endarterectomy. Naturally these figures will be influenced by the perceived utility of such treatments as anticoagulants, aspirin, and carotid endarterectomy. "Missing" 2.8% of non-stroke lesions may appear worse than it really is, since many are untreatable (four out of five in our series) and most will probably declare themselves if the doctor is alert to any uncharacteristic deterioration or raised intracranial pressure developing after what appears to have been a "stroke."

At present most patients with stroke in Britain are either kept at home or admitted to district general hospitals which are unlikely to have a CT scanner. Very few are admitted direct to regional or subregional neurological centres where both the level of clinical neurological skill is highest and CT facilities are available. Fortunately, we think that the criteria for scanning patients are straightforward—namely, (a) doubt (usually because of an inadequate history) whether the patient has had a stroke or has a treatable intracranial lesion (such as a meningioma or subdural haematoma); (b) the possibility of cerebellar haemorrhage or infarction; (c) the exclusion of intracranial haemorrhage in patients who either are already taking or likely to need antihaemostatic drugs or are being considered for carotid endarterectomy; and (d) if the patient deteriorates in a fashion atypical of stroke.

We conclude that it is both unnecessary and wasteful to scan all patients with stroke, particularly if by so doing they have to be admitted or transported to hospitals many miles away, or if such a policy reduces the availability of CT to patients who need it more. We think that our criteria define a minimum standard in the management of stroke; sufficient CT facilities should be available to meet it, or better it.

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Addendum

Since this report was first prepared a further 175 cases of clinically definite first stroke have been registered. None of the patients had non-stroke lesions detected by CT or found at necropsy. The frequency of non-stroke lesions in patients with a clinically definite first stroke is therefore five out of 498 (1.0%).

Appendix

POSSIBLE INDICATIONS FOR PERFORMING CT SCAN ON PATIENT WITH STROKE

To establish diagnosis:

Differentiate "stroke" from "non-stroke" in patients with a poor history (dysphasia, confusion, coma) or a progressive deficit
Identify the pathological type of stroke (intracranial haemorrhage or cerebral infarction)

To identify surgically treatable type of stroke:

Supratentorial haematomas suitable for evacuation
Cerebellar haematomas or infarcts, with or without obstructive hydrocephalus
Arteriovenous malformations or aneurysms

To exclude intracranial haemorrhage:

Already receiving or before anticoagulant treatment
Already receiving or before antiplatelet treatment
Before angiography (for presumed ischaemic minor stroke, etc)

In spontaneous subarachnoid haemorrhage:

Confirm presence of blood
Localise any intracerebral haematoma
Identify site of bleeding
Identify source of bleeding—for example, aneurysm or arteriovenous malformation
Identify cause of deterioration: hydrocephalus, recurrent haemorrhage, cerebral infarction secondary to vasospasm

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Smoking and coronary artery disease assessed by routine coronary arteriography

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Abstract

The association between extent and duration of smoking habit and severity of coronary atheroma was examined in 387 patients undergoing routine coronary arteriography before valve replacement surgery. Total number of cigarettes smoked in life correlated significantly with

severity of coronary artery disease ($p < 0.001$) and number of coronary arteries with stenoses of 50% or more ($p < 0.001$). Severity of coronary artery disease in current smokers was similar to that in former smokers. Multiple regression analysis showed diastolic blood pressure, cigarette consumption, age, ratio of total cholesterol to high density lipoprotein cholesterol, and history of angina to be the important predictors of severity of coronary artery disease.

An estimate of the number of cigarettes smoked in life can be useful in identifying patients with coronary artery disease if used in conjunction with data on other important risk factors.

Introduction

Since White first described an association between smoking and myocardial infarction¹ numerous studies have shown the same association.²⁻⁸ Most prospective epidemiological studies with

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