Computed Tomography in Patients Presenting with Lacunar Syndromes


SUMMARY Of 312 stroke patients who had a CT scan, 37 had presented clinically with a lacunar syndrome. In 18 of the 37, lacunar sized infarcts were demonstrated on the scan, 13 had normal scans and in 6 large infarcts were found. Of these 6, 5 had a pure motor hemiplegia and one a pure sensory stroke. Clinical evidence and angiography revealed a potential and treatable source of emboli in both the lacunar sized and the large infarcts. Two conclusions are drawn: a clinical lacunar syndrome may be associated with a large infarct; demonstration of a lacunar infarct on CT scan does not exclude the need for angiography in appropriate cases to discover a possible source of emboli.

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Methods

Among 312 stroke patients who had been studied by CT scanning at the National Hospital, London, on EMI 1010 or 5005 machines, there were 37 who presented with one of 4 syndromes known to be associated with lacunar infarction. All the patients underwent detailed neurological examination during the acute stage of their stroke and fitted into one of the 4 following groups;

1. Pure motor hemiplegia (involving face, arm and leg);
2. Pure sensory stroke involving the face, arm and leg;
3. The dysarthria-clumsy hand syndrome;
4. Homolateral ataxia with crural paresis.

In deciding that a patient was hypertensive, the patient’s history and blood pressure readings were considered. When these were not available, the presence of hypertension was judged by serial readings after the acute stage. Initial admission blood pressure readings were not used.

The CT scans of these patients, which were done at various times after the onset of symptoms, were reviewed and the anatomical localization and estimated size of the lesions were recorded.

The size of the lesion was estimated by measuring the longest dimension and the greatest dimension at right angles to it and multiplying them. This was corrected for the method of display, i.e. polaroid, transparency, etc, and multiplied by the depth of the lesion calculated by the number and depth of contiguous sections in which it was seen. This gave, in fact, the volume of the cuboid containing the lesion and was thus the maximum possible size of the lesion having the dimensions measured. Most infarcts probably occupy approximately half this total volume and so an arbitrary estimate of the actual size was obtained by dividing the size of the cuboid by two. Despite the difficulties in obtaining an accurate assessment of the size of an irregular lesion involving several sections, it was considered better to use such a measured approximation than merely to grade by eye. The site of lesion was classified as being superficial or...
deep and according to the distribution of the supplying artery.

Results

Out of 312 patients with stroke who were investigated by CT scan, 37 were identified as having a clinical presentation compatible with a known lacunar syndrome. Twenty-six patients had a pure motor hemiplegia, 4 a pure sensory stroke, 6 a dysarthria-clumsy hand syndrome and one a crural paralysis with ataxia.

Twenty-nine were male, 8 female, (mean ages 58.5 years and 45.5 years respectively). Ten patients were normotensive (8 male, 2 female). Twenty-seven had elevated blood pressure with systolic over 150 or diastolic over 110 mm Hg (23 of these were males). Three patients were diabetic and 5 had elevated blood lipids.

Each of the patients had at least one scan. Thirteen had normal scans; 2 others had a normal scan within 24 hours of the acute episode, but an abnormal scan after one week. Among the patients with abnormal scans, 18 had small deep lesions of less than 4 ml estimated volume. Fisher initially limited the term lacune to lesions of less than 15 mm diameter which by our method of calculation gives a volume of 1.7 ml; 15 of our 18 patients were within this limit. However, Fisher extended the concept to include what he termed giant lacunes, the largest having a volume of less than 3 ml and one of 3.8 ml. Twelve of our patients had a single small lesion and 5 had at least 2; one had both a small and a large lesion. Six patients had large infarctions only, ranging in size from 5.8 to 23.0 ml.

Pure Motor Hemiplegia

The 26 patients with pure motor deficit had no impaired visual field defect or speech difficulties on examination. In 14 patients, there was hemiplegia, involving face, arm and leg, while in 12 patients there was only a paresis of face, arm and leg, the severity of involvement often being greater in one part than another. One patient had 3 episodes of paresis of one arm separated by months but paresis of the face, arm and leg in one of these attacks.

The clinical course was benign in most patients with pure motor hemiplegia, 18 making almost complete recovery. Twelve patients had more than one distinct episode. The tendency was for less recovery with each succeeding stroke. One patient had a subsequent brain stem stroke to which he succumbed.

Ten of the patients had an appropriate lacunar lesion on the scan (fig. 1), 10 had normal scans but 5 had

FIGURE 1A and B  Contiguous CT sections after intravenous contrast medium showing a large lacunar infarct involving posterior limb of left internal capsule and extending into lentiform nucleus and corona radiata in a hypertensive man with pure right hemiplegia which recovered completely.
large superficial infarcts at sites appropriate to the clinical deficit (fig. 2). These results are recorded in the table.

Pure Sensory Stroke

Four patients had pure sensory stroke. All had sensory symptoms involving the left face, arm and leg without motor findings, visual field abnormality or speech disturbance. One had a normal scan, one diffuse cerebral atrophy, one showed bilateral lacunar infarcts, and one had an infarct of 5.8 mls which is larger than the accepted range for lacunes.

Dysarthria-Clumsy Hand Syndrome

Six patients had symptoms compatible with the dysarthria-clumsy hand syndrome. Five had involvement of the left hand, one the right. One patient had a motor hemiplegia involving the other side with complete recovery 20 years previously. The scans in all six patients showed small deep lesions in the capsular region; one had, in addition, an equivocal lesion in the pons. In 2 patients, 4 distinct lesions were seen.

Homolateral Ataxia with Crural Paresis

One patient, a 60 year old hypertensive man, had a crural paralysis with mild ataxia of the same side. His scan was normal.

In summary of 37 patients presenting with a clinical syndrome usually associated with a lacunar infarct, 18 (48%) had the lesion confirmed by CT scan. Thirteen patients had normal scans but these may have included lacunes which were too small to be identified. However, there were 6 examples of large superficial lesions accounting for the neurological deficit; 5 occurring in the pure motor hemiplegia group, and one in the pure sensory deficit group.

The sites of lesions considered as lacunes were all localized to the internal capsule or adjacent lentiform nucleus or corona radiata (fig. 3). Five lesions were considered to be in the genu, 3 in the posterior limb and 3 in adjacent trigone area. Five lesions were

<table>
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<th>TABLE CT Scan Results</th>
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<tr>
<td>Number of patients</td>
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<tr>
<td>i Pure motor hemiplegia</td>
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<td>ii Pure sensory stroke</td>
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<tr>
<td>iii Clumsy hand-dysarthria</td>
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<tr>
<td>iv Crural paresis</td>
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FIGURE 3A and B CT scan showing lacunar infarct in posterior limb of left internal capsule extending into lentiform nucleus in a hypertensive man with pure right motor hemiplegia which recovered. There are also lacunes in the right thalamus and an ill-defined area of low density in the right frontal lobe which had not caused clinical disturbance. (B) Coronal section of the brain showing lacunes in the right thalamus and left internal capsule and lentiform nucleus corresponding with CT scan. The patient had died from a subsequent cerebral hemorrhage which is also seen. The left and right hemispheres are respectively on the left and right of the picture.

mainly situated in the anterior limb, globus pallidus or putamen. In 2 of the lesions in the capsular region, the site could not be identified more specifically. Lesions were not identified with certainty in the brain stem but 2 patients with capsular lesions also had cerebellar infarcts.

In view of the fact that clinical syndromes usually associated with lacunes were also shown in some instances to be caused by large superficial lesions, the histories of the patients were re-examined in the 3 groups; those with normal scans, those with scans showing lacunes and those with large infarctions. The patients with large lesions had a lower incidence of hypertension with only 3 of 6 having elevated blood pressure compared with 14 of 18 patients with small lesions and 12 of 13 with normal scans. There were no significant differences in sex distribution but those with large lesions were older (mean age 64 years compared with 56 for those with lacunes and 58 for those with normal scans).

Discussion

The clinical diagnosis of a lacunar syndrome entails the recognition of one of the 4 known syndromes mentioned above in a patient who has a marked tendency to recover. The absence of hypertension in any patient does not exclude the diagnosis. The majority of our 37 patients had pure motor hemiplegia and 5 of the 6 large infarcts were in this group. The diagnosis of a lacunar infarction on the basis of pure motor hemiplegia obviously entails exclusion of any dysphasic speech disturbance and, in minimally affected cases, this may be difficult to distinguish from non-dysphasic speech difficulties. It is also necessary to exclude patients with even minor sensory disturbance. The care with which these exclusions are made will obviously depend on the individual clinician; the examinations in each patient in this series were performed by a competent neurologist and the findings at the time of examination were consistent with the lacunar syndrome. We excluded all patients with any speech disturbance, for the above reasons.

Of 37 patients who presented with a clinical picture compatible with lacunar infarction, 18 had the lesions verified by CT scan. There are a number of possible reasons for the 13 with normal scans.

Campbell et al.10 have noted that early scans may be negative with a peak detection rate between the eighth
and eleventh day. This is unlikely to be the explanation in our patients in that all of the normal scans were carried out at least 5 days after the acute episode. Two patients who had had normal scans within 24 hours of their acute episode had lacunar lesions demonstrated in the second week. Alcala et al.14 have studied possible reasons for normal scans in the presence of infarction. The most obvious explanation is that the lesion is too small to be picked up by the scan, particularly if it is in the brain stem.

It should be noted that almost half the normal scans occurred in patients with a paresis of one limb or the face and minimal involvement of the other parts. This might suggest that the smallest lacunes may produce incomplete syndromes. Fisher* has stated that an infarct of restricted size in the capsule may cause a pure motor monoplegia. However, he also points out that a monoplegia is more likely to result from a small cortical lesion. One would have expected in the latter case that the lesion would have been detected by the scan.

A third explanation for normal scans is that the lesions are in the brain stem which is a less favorable area for demonstration of lesions by scan. However, there were no particular features pointing to the brain stem as the site of the pathology in our patients.

It is evident from postmortem studies that patients may have multiple lacunes with little clinical evidence of neurological deficit. The lesions seen on CT scan are not necessarily the only lacunes present and exact clinico-pathological correlation is therefore difficult. In general, however, patients with pure motor hemiplegia with positive scans had lesions appropriate to the clinical picture. The highest incidence of positive scans was found in patients with dysarthria-clumsy hand syndrome, where all of the patients had lacunar lesions in the contralateral internal capsule. Fisher, however, incriminated a lesion in the pons as responsible for this syndrome. One of our patients had a possible pontine infarct as well and two others had unilateral or bilateral deep cerebellar infarcts, one having a total of 5 lacunar infarcts. Pontine infarcts could have been present in other patients since they are difficult to demonstrate and, therefore, are never excluded by scans.

The presence of 6 large infarcts — 5 of them with pure motor hemiplegia and the sixth with a pure sensory stroke — suggests that the clinical diagnosis of a lacunar syndrome is not always accurate. The 5 patients with pure motor hemiplegia were all males over 62 with a right-sided lesion. Three of them were normotensive and the other 2 were adequately controlled on antihypertensive agents. There was otherwise nothing remarkable about their histories. The patient who had a large lesion causing a pure sensory stroke was a normotensive female of 26 on oral contraceptives.

The size of lesions demonstrated by CT scan is similar to those described by Fisher* some of which were 25 × 25 × 10 mm when measured at autopsy; these he refers to as large lacunes. In an earlier paper, Fisher1 suggested that the nature of the lesion, rather than its size, should be the criterion for the term lacune. However, he referred to lesions larger than 1 ml as giant lacunes. The pathogenesis of these lesions appears to be similar to that of the smaller lesions and involves atheroma or a degeneration termed lipohyalanosis of the small penetrating thalamostriate arteries. Hughes,14 however, suggested that they are due to kinking of these small vessels and Ross Runsett15 has demonstrated small aneurysms in relation to them; Cole and Yates14 have shown that many are due to small hemorrhages, probably from these same aneurysms. Prineas and Marshall,16 as well as Fisher, have emphasized the relationship to hypertension.

While many of our patients were hypertensive, several were not. Lesions with the same appearance on scan could have been due to emboli, although a source of emboli was not obvious. Only one patient had a cardiac arrhythmia, one had a history of myocardial infarction, and a further 2 had angiographic abnormalities of middle cerebral artery, delayed flow, and occlusion respectively. Both the patient with a cardiac arrhythmia and the patient with delayed flow in the middle cerebral artery territory had lacunar infarcts.

The neck vessels are an alternate source of emboli and one patient had bilateral carotid atheroma and severe internal carotid stenosis with an infarct just outside the lacunar range. A further patient, however, had bilateral carotid atheroma with a lacunar sized infarct on the side opposite the hemiplegia and a symptomless larger right frontal infarct. This patient underwent endarterectomy. Several other patients were treated with either aspirin or anticoagulants. On the other hand, out of 7 patients with atheroma of neck vessels, the 4 patients in whom the atheroma was regarded as minor, all had lacunar sized infarcts or normal scans.

Four patients had carotid occlusions, all of them right-sided and on the side opposite the hemiparesis. The scans were normal in one patient, 2 had lacunar sized lesions on the same side as the occlusion and the fourth had a large infarct. All 4 were normotensive. Thus the demonstration of small deep infarcts on CT scan does not exclude the necessity for angiography.

From this study, it is apparent that patients presenting clinically with the lacunar syndrome may have large and superficial infarcts as well as small deep ones, and that both may be associated with a potential and treatable source of emboli. One benefit of CT scanning in these patients is to detect large lesions producing the clinical picture usually associated with lacunes. Though cost effectiveness of CT scanning in the practical management of cerebrovascular problems has recently been called into question,16 it clearly has a part to play in clinicoanatomical correlation in this difficult diagnostic field.

Acknowledgment

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Evidence for Greater Susceptibility of Isolated Dog Cerebral Arteries to Ca Antagonists than Peripheral Arteries
KOICHIRO SHIMIZU, M.D., TOMIO OHTA, M.D., PH.D., AND NOBORU TODA, M.D., PH.D.

SUMMARY In helically-cut strips of dog cerebral, coronary and mesenteric arteries, contracted with prostaglandin (PG) F2\alpha, or K+, the addition of verapamil caused a dose-related relaxation. Verapamil-induced relaxations were greater in cerebral than in the other arteries when contracted with PGF2\alpha, but did not significantly differ in the arteries contracted with K+. Similar results were obtained with diltiazem and nifedipine. The contractile response to PGF2\alpha was attenuated by pretreatment with verapamil, the attenuation being greater in cerebral than in mesenteric arteries. Nitroglycerin and sodium nitroprusside relaxed cerebral, coronary and mesenteric arteries contracted with PGF2\alpha to a similar extent. It may be concluded that dog cerebral arteries contracted with PGF2\alpha, one of endogenous vasospastic substances, are more susceptible to agents which interfere with the influx of Ca++ across cell membranes than coronary and mesenteric arteries; these agents may thus be of value in the treatment and prophylaxis of cerebral vasospasm.

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References

Evidence for Greater Susceptibility of Isolated Dog Cerebral Arteries to Ca Antagonists than Peripheral Arteries

ISOLATED CEREBRAL ARTERIES respond to vasoconstricting and vasodilating agents differently from peripheral arteries.1,2 Cadmium (Cd++) antagonists, including verapamil, nifedipine, diltiazem and Cd++, interfere with the influx of Ca++ across cell membranes of vascular smooth muscle,3-7 thereby resulting in vasodilatation. Contractile responses to Ca++ of cerebral arteries exposed to Ca++-free media and depolarized by K+ are attenuated to a greater extent by verapamil and Cd++ than those of coronary and mesenteric arteries.8

Nitroglycerin and sodium nitroprusside are potent vasodilators9,10 and cause rapid hypotension. These agents are also expected to release cerebroarterial spasm following subarachnoid hemorrhage.11 No information is available concerning the response of cerebroarterial smooth muscle to these vasodilators.

The present study was thus undertaken to compare quantitatively the response of isolated dog cerebral, coronary and mesenteric arteries to Ca++ antagonists, nitroglycerin and sodium nitroprusside. Verapamil prevention of drug-induced contractions was also compared in cerebral and mesenteric arteries.

Methods
Mongrel dogs of both sexes, weighing 7 to 15 kg, were anesthetized with intraperitoneal injections of sodium pentobarbital 50 mg/kg, and sacrificed by...
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