Lesson of the Week

Rapid resolution of signs of primary intracerebral haemorrhage in computed tomograms of the brain

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Computed tomography of the brain plays an important part in the management of stroke, both in those cases in which diagnosis is uncertain[1] and to differentiate haemorrhagic from ischaemic stroke. Although deciding which type of stroke is present may not affect the management of many patients, it is important if treatment with anticoagulants or carotid surgery is being considered. When such treatment is being considered it is vital that patients with primary intracerebral haemorrhage are correctly identified as anticoagulation is dangerous and carotid endarterectomy inappropriate. It may also be advisable to exclude primary intracerebral haemorrhage before treatment with aspirin is started, although whether aspirin predisposes to extension of the stroke or recurrence in cases of primary intracerebral haemorrhage is not known.

Differentiating between haemorrhagic and ischaemic stroke on clinical grounds is not sufficiently reliable to allow these management decisions to be made[2] and is not improved by examination of the cerebrospinal fluid.[3] Clinical scoring systems, such as the recently described Allen score,[3] improve the reliability of the clinical assessment but are insensitive in identifying mild strokes due to small intracerebral haemorrhages. It is the patients with mild strokes, however, who have most to gain from secondary preventive measures.

In the weeks after a primary intracerebral haemorrhage the high density area on computed tomography is replaced by a smaller isodense or hypodense area. Sometimes these changes occur rapidly and may make primary intracerebral haemorrhage indistinguishable from infarction if tomography is delayed. If this fact is not considered then inappropriate treatment may be given. Little has been written about the clinical implications of these rapid changes after primary intracerebral haemorrhage: several of the major medical and neurological textbooks do not mention the problem.[4-7]

We present five cases of primary intracerebral haemorrhage that show how easy it is to misdiagnose the type of stroke if the timing of computed tomography is not taken into account.

In cases of primary intracerebral haemorrhage the characteristic high density lesion seen on computed tomography may resolve rapidly, so that within two weeks of stroke the radiological appearance may resemble that of cerebral infarction. If treatment with anticoagulants, carotid endarterectomy, or aspirin is being considered computed tomography should be performed within two weeks of the stroke. Failing this, the radiologist should be alerted to the delay to avoid misleading reports such as "consistent with cerebral infarct" being issued when the computed tomogram is equally consistent with primary intracerebral haemorrhage

Methods

The Oxfordshire community stroke project has been described in detail elsewhere.[8] All patients with first ever stroke and transient ischaemic attack in a well defined population of about 105 000 were seen prospectively by a study neurologist. About 90% underwent computed tomography or necropsy, and all survivors are being followed up.

Computed tomography was performed as soon as possible after the stroke, although some delay was inevitable because nearly half the patients remained at home.[9] Between November 1981 and February 1984 tomography was performed with a modified EMI 1007 scanner with a 160×160 matrix. Subsequently it was performed with a Siemens Somotom DR1 scanner, which has greatly improved resolution and allows the maximum densities and the volumes of the high density lesions to be measured with a pen guided cursor. One patient reported on here (case 5) was scanned with the older machine, and therefore these measurements were not available.

When computed tomography showed a small or moderately sized primary intracerebral haemorrhage attempts were made to rescan the patient at frequent intervals to document the resolution of the haematoma. Many patients in the project were old and frail, and often it was not possible to obtain serial tomograms. In several, however, later tomograms were obtained, five of which showed rapid resolution of the haematoma. It is these five that form the basis of this report.

The later tomograms were reported by a neuroradiologist (AM), who did not know the clinical details other than that the patient had had a clinically obvious stroke. Reporting was done without the earlier tomograms showing the primary intracerebral haemorrhage and without any information about their timing. The Allen score[3] was calculated in each case, and the probability that the stroke was ischaemic was derived from this.

Case reports

Case 1—An 82 year old man experienced a sudden right hemiparesis with vomiting but no headache or drowsiness. There was no history of hypertension, but he had suffered two minor brain stem infarctions two years before, which had been confirmed by computed tomography within six days of the events. He had been taking aspirin intermittently since then. On examination he had a bruit audible in the right supraclavicular fossa and a moderate right hemiparesis with no dysphasia. His Allen score was −5·7, indicating a 98% probability of cerebral infarction. Computed tomography

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eight days after this stroke showed a haemorrhage in the left basal ganglia. When repeated 21 days after the stroke it showed only an area of low density.

Case 2—A 77 year old man experienced sudden weakness of his right arm and unsteadiness on walking. He did not have headache, vomiting, or drowsiness. The symptoms resolved within 72 hours, and when seen he had no abnormal neurological signs. He was known to be hypertensive and to have atrial fibrillation of long duration. His Allen score was −0·23, indicating a 96% probability of cerebral infarction. Aspirin was started. Computed tomography eight days after the stroke showed a haemorrhage in the left basal ganglia, and aspirin was promptly withdrawn. Computed tomography 21 days after the stroke showed only an area of low density in the left basal ganglia.

Details of lesions seen in computed tomograms in patients with primary intracerebral haemorrhage

Case No	Days after stroke	Maximum density (HU)	Volume ($\times 10^{-3}$ l)	Maximum diameter ($\times 10^{-3}$ m)	Rate of change/day: Density (HU)	Volume ($\times 10^{-3}$ l)	Diameter ($\times 10^{-3}$ m)
1	8	55	6·9	27·3	1·9	0·53	2·1
	21	30	0	0			
2	8	64	5·1	32·5	2·6	0·39	2·5
	21	30	0	0			
3	7	67	5·5	22·4	2·6	0·39	1·6
	21	30	0	0			
4	4	74	3·2	19·5	3·0	0·21	1·3
	19	29	0	0			
5	7	No measurements available					
	13						

HU=Hounsfield unit.

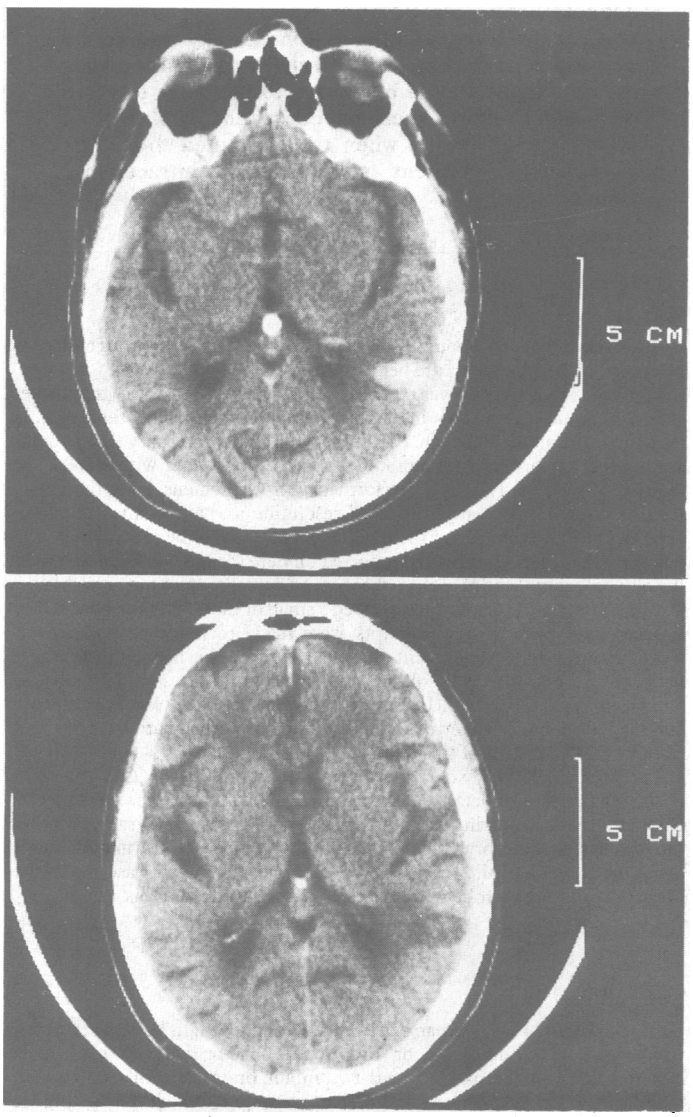

FIG 1—Case 3. Computed tomograms showing right parietal haemorrhage at seven days (top), which had resolved by 21 days, leaving a low density area (bottom). The late scan could have been interpreted as showing cerebral infarction in the distribution of a distal branch of the right middle cerebral artery.

Case 3—A 66 year old left handed man suddenly developed dysphasia without weakness of his arms or legs. He did not have headache, vomiting, or drowsiness. He gave a history of hypertension. His Allen score was −5·7, indicating a 98% probability of cerebral infarction. His dysphasia recovered rapidly, and he was discharged taking aspirin. Computed tomography seven days after the stroke showed a small right parietal haemorrhage (fig 1 (top)), which had resolved 21 days later, leaving a low density area in the tomogram (fig 1 (bottom)). Aspirin was withdrawn.

Case 4—A 60 year old man woke with left sided facial weakness of an upper motor neurone type and dysarthria but no focal signs in his arms or legs. He did not have headache, vomiting, or drowsiness. He was known to be hypertensive. His Allen score was −3·4, indicating a 97% probability of cerebral infarction. Computed tomography four days after the stroke showed a haemorrhage in the right basal ganglia, which was still visible at 12 days but had resolved by 19 days, leaving only an area of low density.

Case 5—A 59 year old woman presented with sudden confusion, loss of memory, and drowsiness. She had a mild headache but no vomiting. On examination she had a spastic quadriparesis and profound loss of memory. She had been mildly hypertensive in the past but had not been treated for this. Her Allen score was +6·75, indicating an 87% probability of cerebral infarction. Computed tomography seven days after the stroke showed a left thalamic haemorrhage. When tomography was repeated 13 days after the stroke no abnormality was seen.

The table gives the maximum density, volume, and diameter of each lesion and the rates of change of the variables. Volume and diameter were assigned a value of zero at the time of the second scan even though the lesion might have disappeared before this, so the rates of change are therefore minimum estimates.

The second tomograms, performed within 21 days of the stroke, were all consistent with a diagnosis of cerebral infarction.

Discussion

Primary intracerebral haemorrhage appears as an area of well defined high density (50-80 Hounsfield units (HU)) in a computed tomogram[10] except in severe anaemia.[11] After a few days a surrounding area of low density appears, which has been attributed to oedema, clot retraction, and ischaemia of the surrounding brain.[11] After the stroke the density of the haematoma has been reported to decrease at a rate of about 1·4 HU/day (range 0·6-2·6).[10] This is probably due to breakdown of haemoglobin rather than actual resorption of the clot.[12] The extent of the high density area also decreases by an average of 0·65 mm/day (range 0·2-1·4).[10] At some stage the haematoma becomes isodense with the surrounding brain, and later it may leave an area of hypodensity. The changes recorded here, however, were much faster than this, perhaps because all the haematomas were small (table). We showed a mean decrease in

maximum density of 2·5 HU/day (range 1·9-3·0) and a decrease in diameter of 1·9 mm/day (range 1·3-2·5). The rate of change in density and extent of the primary intracerebral haemorrhage may not, however, be constant but may increase with time.

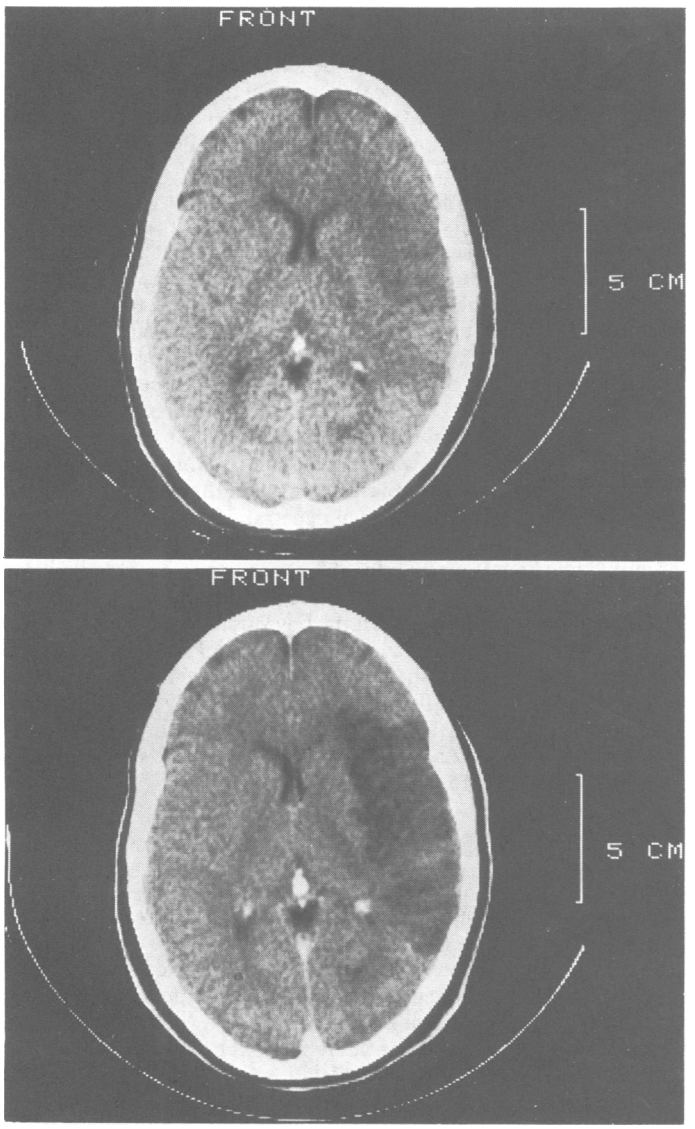

FIG 2—Computed tomogram within two days of cerebral infarction (top), which had become more obvious two weeks later (bottom). The late scan is unlikely to be confused with a resolving haemorrhage because the low density area is large and in the distribution of the right middle cerebral artery.

These cases show how the high density area evident in computed tomograms after primary intracerebral haemorrhage may become isodense or hypodense in a period as short as 13 days. When the late tomograms were shown to a neuroradiologist all were diagnosed as showing cerebral infarcts. Had it not been possible to obtain an early scan, therefore, primary intracerebral haemorrhage would not have been suspected and inappropriate treatment might have been, and in some cases was, given. The clinical features of patients with moderate or small primary intracerebral haemorrhage often give no clue to the underlying pathology. This is shown by the low Allen scores in all of these cases: four of the five scores showed a greater than 90% probability that the stroke was due to infarction.

Problems of diagnosis arise only in patients with small lesions because those with larger haemorrhages are likely to have clinical features, often related to intraventricular extension, that will alert the clinician to the diagnosis. A large haematoma will also take much longer to resolve and will therefore remain visible in computed tomograms for many weeks. If a large area of low attenuation is seen in a computed tomogram three or four weeks after a stroke it is unlikely to have been caused by primary intracerebral haemorrhage, particularly if the shape is typical of infarction in the distribution of a cerebral artery (fig 2).

Computed tomography is the most accurate tool for differentiating types of stroke in the acute phase. Magnetic resonance imaging is unlikely to replace it in this role, at least in the foreseeable future, because of its limited availability. In any case, magnetic resonance imaging is unreliable in differentiating haemorrhage from infarction immediately after stroke. After several weeks, however, the T_2 relaxation times for primary intracerebral haemorrhage decrease and may allow this to be distinguished from cerebral infarction.[13] This may be useful in the future where differentiation is essential and computed tomography is unavoidably delayed.

With the increasing availability of computed tomography in the United Kingdom, especially in district general hospitals, it will probably be used increasingly to assess patients with stroke. We thus emphasise that to exclude a primary intracerebral haemorrhage with maximum reliability computed tomography must be performed early, certainly within the first two weeks and ideally within the first seven days after the stroke. If this is impossible the radiologist should be informed of the delay so that subtle changes associated with resolving haematomas will not be missed. These changes include a persisting mass effect, an area of isodensity surrounded by hypodensity, and the shape of the hypodense area, which may be elliptical or slit like.[10] If the radiologist is aware that the tomogram was obtained late his report should not contain a phrase such as "consistent with an infarct"; "consistent with an infarct or previous primary intracerebral haemorrhage" is preferable. This may prevent the patient being given treatment that is not only inappropriate but dangerous.

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How reliable is the laboratory test for psittacosis and do false positive results occur?

Serological tests for psittacosis are reliable and are the standard method for confirming the diagnosis. False positive results are not a problem, but it should be borne in mind that most laboratories use a generic test that will not distinguish between infection with different species of chlamydia. Specific tests are available to differentiate between infection with *C psittaci* and *C trachomatis*.—HILLAS SMITH, consultant in infectious diseases, London.