Evaluation of computed tomography in vascular lesions of the vertebrobasilar territory

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SUMMARY Forty-nine patients with clinical features of vascular disease in the vertebrobasilar territory were examined to evaluate the diagnostic performance of computed tomography. It provided useful information in the diagnosis of acute vascular lesions in patients in whom a definite episode had occurred not longer than 2 weeks before computed tomography was undertaken. It was of value in both haemorrhagic and thromboembolic groups but in many patients angiography was required to make a substantive diagnosis. It was of less value in patients with vague and ill defined symptoms and of no value in transient ischaemia.

In the cerebrum vascular abnormalities such as infarcts^{1,2} haemorrhages³⁻⁶ and aneurysms⁷ are usually well demonstrated by computed tomography (CT) although considerable disability may occasionally be accompanied by a normal computed tomogram. On the other hand vascular disease of the vertebrobasilar territory is frequently difficult to assess not only because of the vague symptomatology but because the posterior fossa remains a difficult area to visualise, small lesions being easily missed. In addition longstanding vascular disease may result in a scan demonstrating only cerebellar and brain stem atrophy.⁸ Since the clinical diagnosis of a vascular lesion involving the vertebrobasilar territory is not infrequently made, we considered it would be of value to assess the accuracy of computed tomography in making a definitive diagnosis, in detecting other changes which although not directly related were possibly of a vascular nature and in addition, in assessing the need for other radiological investigations to confirm the clinical diagnosis.

Methods

Details of patients with a clinical diagnosis of vascular disease related to the vertebrobasilar territory obtained from the computer register at the National Hospital for Nervous Diseases,

between early 1974 and mid 1976. There were 49 patients, 29 males and 20 females. The notes, CT scans and angiograms of all patients were examined to assess the correlations between clinical diagnosis, CT and other radiological findings. In many cases a definitive diagnosis was never completely established.

The length of time between onset of symptoms and CT was divided into four groups: -

- 1 One to two days (ten patients)
- 2 Three to six days (five patients)
- 3 One to two weeks (eight patients)
- 4 Greater than four weeks (26 patients)

Results

Group One: (ten patients) Eight of the CT scans were abnormal and two were normal. Both patients with normal CT scans had a definite history of a brain stem vascular episode of gradual onset with a previous history of purely transient ischaemic attacks in the vertebrobasilar territory consisting of vertigo, nausea and vomiting, ataxia and long tract signs. In both patients the clinical features of the most recent attack were also transient although one lasted 24 hours. Angiography was not performed on either patient but air encephalography demonstrated cortical atrophy in one.

Of the eight patients with abnormal CT there was precise correlation between CT and the clinical features in four. In one patient, scanned two days after a subarachnoid haemorrhage, the

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Accepted 24 September 1979

basilar tip aneurysm responsible was demonstrated both before and after intravenous contrast enhancement. There was no blood in the basal cisterns. In a second patient with a basilar tip aneurysm subarachnoid haemorrhage was demonstrated on computed tomography, but the aneurysm was only diagnosed at vertebral angiography. A third patient had a history of a stroke in evolution over two days and CT showed low attenuation in the cerebellum and brain stem with enhancement after the injection of contrast. In a fourth case, with a sudden onset of symptoms, the appearances (a low attenuation area) were those of an infarct.

In the other four patients correlation between the clinical course and the CT scan was poor. Atrophy of the cerebellum and cerebral cortex was present in two, one of whom died (further reference will be made to this patient later). There was atrophy of the cerebral cortex alone in one and in the fourth patient, with quadriplegia and expressive dysphasia ("locked-in syndrome"), computed tomography showed slight dilatation of the lateral ventricles without abnormality in the posterior fossa. This patient was only scanned once and the ventricular dilatation may have represented early hydrocephalus perhaps due to a cerebellar infarct that had not been shown by CT. Two patients in this group died; one following a subarachnoid haemorrhage and the other, (previously referred to) after extensive infarction in the right middle cerebral and vertebrobasilar territory revealed only at post mortem. The scan showing atrophy only had been undertaken within 12 hours of the onset of the clinical symptoms, but 36 hours before death.

In the eight cases with abnormal CT, five showed clinical features of sudden onset and in seven some of the neurological disability was permanent.

Group Two: (five patients) All five patients in this group had abnormal computed tomograms. High density lesions consistent with haemorrhage were shown in the cerebellum in three, all with symptoms of sudden onset, blood being also present in the brainstem in one of them and subsequently confirmed by angiography. Contrast was not given to a second patient later shown by angiography to have an angioma. The third patient, a child of seven was admitted comatose and died shortly afterwards. Necropsy was not undertaken.

Neither of the other two patients had CT changes in the posterior fossa. One was shown to have a small basilar tip aneurysm by angiography

not visible on an unenhanced scan. The basal cisterns were normal, the subarachnoid haemorrhage having occurred six days before scanning. The other patient, with a progressive five day history of vertebrobasilar insufficiency (VBI) showed only cortical atrophy on the scan which was undertaken two days before his sudden deterioration, terminal collapse and subsequent death. A post mortem examination revealed basilar artery occlusion.

Group Three: (eight patients) All patients were scanned between seven and 14 days after the episode. There were seven abnormal scans. Three had high density lesions in the posterior fossa. The only patient in this group with a sudden onset of symptoms suffered a brain stem haemorrhage diagnosed clinically and on CT. One had a stuttering but progressive history of dysarthria, ataxia and diplopia over six days; CT demonstrated a basilar aneurysm without evidence of haemorrhage. The third demonstrated an enhancing mass in the left cerebellar hemisphere diagnosed at angiography as an angioma.

Two patients had low attenuation lesions in the cerebellum. One presented with a history suggestive of a cerebellar and brain stem infarct while the other, with symptoms pointing to supratentorial as well as infratentorial vascular disease, showed cortical atrophy as well. In the remaining two patients the abnormal scans demonstrated cerebral atrophy only. Both these patients had long histories, one of dementia and Parkinson's disease and the other of a previous head injury, but are included here because they presented with more acute episodes of ataxia and vertigo. The last patient in this group had a normal scan after three transient ischaemic episodes related to the posterior fossa over a two week period.

Group Four: (26 patients) Except for three patients scanned at four, five and six weeks after the ictus, all scans were undertaken at least eight weeks after the onset of the symptoms. Sixteen patients had abnormal and 10 normal scans. The patients with abnormal scans will be described first. Two had high density lesions in the preponine cistern; angiography confirmed a basilar artery aneurysm in one and a calcified basilar artery occlusion in the other. Both cases had long histories, the patient with a basilar aneurysm having had transient ischaemic symptoms since a head injury six weeks previously, while the patient with basilar artery occlusion had had an acute episode without progression one year previously. Two patients had low attenuation lesions

consistent with infarcts in the vertebrobasilar territory. Two patients had atrophy of the cerebellar hemispheres. Seven patients had cerebral atrophy, one had a cerebral cortical infarct and two appeared to have hydrocephalus. Five of these patients with cerebral disease had ischaemic symptoms, three being transient. The other five had vague symptoms. Most of the ten patients with normal CT scans (cases 40 to 49) presented with a history of transient ischaemia. None of these had other investigations and their diagnoses remain unconfirmed.

Discussion

The purpose of this paper has been to assess retrospectively the diagnostic performance of computed tomography in those with a confirmed or presumed diagnosis of a vascular lesion in the posterior fossa. Although a number of patients were included in whom the clinical diagnosis was equivocal and whose scans were normal the majority fell into two main groups, those with haemorrhage and those with thromboembolic disease. They are discussed together because in practice the presenting features are often similar.

Initial assessment of this diverse group of patients separated them almost equally into two groups, those (23 patients) in whom the scan was undertaken early, between one and 14 days after the ictus and those (26 patients) where the scan was undertaken more than 1 month after the onset of the symptoms.

We divided the patients scanned within the first two weeks into three subgroups since we thought it possible that thromboembolic lesions scanned within two days of the ictus might not yet be demonstrated on CT while in those patients with subarachnoid haemorrhage a transiently abnormal scan might already have returned to normal by the time the scan was undertaken. No difference was found, however, between these three subgroups. Twenty of these scans were abnormal and in two of the three normal scans, the symptoms were of transient ischaemia.

In all groups there were patients with basilar aneurysms, haemorrhages and infarcts in the vertebrobasilar territory, as well as five patients with changes outside the posterior fossa. Of these one probably was developing hydrocephalus due to a brain stem haemorrhage, while another had

an evolving but incomplete stroke at the time of his scan which was normal.

There was agreement between the clinical and CT diagnosis in 14 patients, all of whom had either posterior fossa haemorrhages or low density infarcts. The primary pathology, either angioma or aneurysm, required confirmation by angiography. In the group of patients scanned late in their disease there was considerably less correlation between the clinical features and CT. Apart from two basilar aneurysms, evidence of old infarcts in the vertebrobasilar territory in two and cerebellar atrophy in a further two, CT only gave nonspecific information.

A further evaluation of the diagnostic limitations of CT scanning was undertaken in the thromboembolic group of patients alone. If those whose clinical features were strongly suggestive of an infarct or transient ischaemia are considered (table 5) only six out of 14 (43%) clinically diagnosed infarcts or strokes in evolution and none of 17 clinically diagnosed transient ischaemic attacks demonstrated CT changes consistent with infarction in the vertebrobasilar territory. Three patients with normal scans had strokes in evolution and it is possible that a subsequent scan would have revealed an infarct in these patients. Cerebellar atrophy possibly due to previous vertebrobasilar disease was observed in one patient with a clinical infarct and one with transient ischaemia, both of whom were in group 4. There is no evidence that computed tomography provided any positive information in patients with transient ischaemic attacks alone.

Computed tomography therefore provides useful information in the diagnosis of the acute vascular lesions of the posterior fossa in those in whom a definite episode has occurred not longer than two weeks before computed tomography is undertaken. It is of value in both the haemorrhagic and thromboembolic groups, but in many of these angiography is required to make a definite diagnosis. Computed tomography is of less value in the group of patients with vague and ill defined symptoms, suggestive of vertebrobasilar disease, but without evidence of an acute episode and it is of no diagnostic value in transient ischaemia.

We wish to thank Miss Wendy Pidgeon for her secretarial help.

Table 1 Group 1—Time between ictus and CT scan 1–2 days

Case no.	Onset	Clinical diagnosis	Plain CT findings	Enhanced CT findings	Angiogram diagnosis	Final diagnosis
1	Sudden	Basilar aneurysm	Pontine & 4th ventricular haemorrhage	Enhancement of aneurysm	Aneurysm	Basilar aneurysm
2	Sudden	SAH Basilar aneurysm	SAH	No enhancement	Aneurysm	Basilar aneurysm
3	Sudden	Brainstem infarct	Low attenuation in brainstem and cerebellum	No enhancement	Not done	Brainstem infarct
4	Gradual	Posterior fossa infarct	Low attenuation in cerebellum	Enhancement	Not done	Cerebellar infarct
5	Sudden	Posterior fossa infarct	Cerebellar and cerebral atrophy	Contrast not given	Not done	Multiple infarcts (death)
6	Gradual	? Embolic	Cerebellar and cerebral atrophy	Contrast not given	Vertebral artery stenosis	Probably ischaemic
7	Gradual	? Ischaemia	Cerebral atrophy	Contrast not given	Not done	Not confirmed
8	Sudden	Brainstem infarct	? Early hydrocephalus	Contrast not given	Not done	Brainstem infarct
9	Gradual	TIA	Normal	Contrast not given	Not done	TIA
10	Gradual	TIA	Normal	Contrast not given	Not done	TIA

Table 2 Group 2—Time between ictus and CT scan 3–6 days

Case no.	Onset	Clinical diagnosis	Plain CT findings	Enhanced CT findings	Angiogram	Final diagnosis
11	Gradual over 4 days	Cerebellar haemorrhage	Cerebellar and Brainstem haemorrhage	No enhancement	Angioma left cerebellar haemorrhage	Angioma haemorrhage
12	Sudden	Posterior fossa haemorrhage	Cerebellar and brainstem haemorrhage	Contrast not given	Not done	Brainstem haemorrhage (death)
13	Sudden	Cerebellar haemorrhage	Subarachnoid and cerebellar haemorrhage	Contrast not given	Angioma of vermis	Angioma haemorrhage
14	Sudden	Subarachnoid haemorrhage	4th ventricle displaced backwards	Contrast not given	Basilar aneurysm	Basilar aneurysm
15	Gradual	Stroke in evolution	Cerebral atrophy	Contrast not given	Not done	Infarct

Table 3 Group 3—Time between ictus and CT scan 7–14 days

Case no.	Onset	Clinical diagnosis	Plain CT findings	Enhanced CT findings	Angiogram	Final diagnosis
16	Sudden	Pontine haemorrhage	Pontine haemorrhage	Contrast not given	Not done	Pontine haemorrhage
17	Gradual	Brainstem lesion type	High density mass	Enhanced (Vascular tumour)	Angioma	Pontine angiomatous malformation
18	Gradual	Brainstem infarct	High density lesion in brainstem	Enhanced	Aneurysm	Basilar aneurysm
19	Gradual	Ischaemia left carotid territory	Cerebellar low attenuation cerebral atrophy	Contrast not given	Not done	Cerebellar infarct
20	Gradual	Cerebellar and brainstem infarct	Cerebellar low attenuation	Contrast not given	Not done	Cerebellar infarct
21	Gradual	?Ischaemia, Parkinsonism	Cerebral atrophy	Contrast not given	Not done	Presumed TIA
22	Gradual	?Ischaemia, trauma	Cerebral atrophy	Contrast not given	Not done	Presumed TIA
23	Gradual	Equivocal	Normal	Contrast not given	Not done	Not diagnosed

Table 4 Group 4—Time between ictus and scan—greater than one month (only patients with abnormal scans included)

Case no.	Onset	Clinical diagnosis	Plain CT findings	Enhanced CT findings	Angiogram	Final diagnosis
24	Gradual	Cerebellar infarct	High density in brainstem	No enhancement	Basilar artery occlusion	Basilar artery occlusion
25	Gradual	Ischaemic	High density ? aneurysm in brainstem	Enhanced	Large basilar aneurysm	Basilar artery aneurysm
26	Sudden	Cerebellar infarct	Cerebellar low attenuation and cerebral atrophy	Contrast not given	Not done	Old cerebellar infarct
27	Gradual	Brainstem vascular lesion	Cerebellar atrophy	Contrast not given	Not done	Consistent with previous brainstem vascular lesion
28	—	TIA	Cerebellar atrophy and cerebral atrophy	Contrast not given	Not done	
29	Gradual	?Infarct	Low attenuation in occipital cortex	Contrast not given	Not done	?Old infarct
30	Sudden	Old infarct ?recent history	Cerebral atrophy and cortical infarcts	No enhancement	Not done	?Carotid territory infarcts
31	—	VBI	Cortical infarct	No enhancement	Not done	?Carotid territory infarct
32	—	TIA	Cerebral atrophy	Contrast not given	Not done	Not diagnosed
33	Gradual	Alzheimer's disease	Cerebral atrophy	Contrast not given	Not done	Not confirmed
34	—	? TIA	Cerebral atrophy	Contrast not given	Not done	Not confirmed
35	—	?	Cerebral atrophy	Contrast not given	Not done	Not established
36	—	?	Cerebral atrophy	Contrast not given	Not done	Not established
37	Gradual	VBI	Cerebral atrophy	Contrast not given	Basilar atheroma	VBI
38	—	?	Hydrocephalus	Contrast not given	Normal	Not established
39	Gradual	Brainstem disorder	Hydrocephalus	Metrizamide scan showed mixed picture	Not done	? Communicating hydrocephalus

Table 5

		Number	CT Infarct
Group 1	Clinical infarct	3	2
	Clinical ischaemia	2	—
	Stroke in evolution	1	—
Group 2	Clinical infarct	—	—
	Clinical ischaemia	2	—
	Stroke in evolution	1	—
Group 3	Clinical infarct	2	2
	Clinical ischaemia	—	—
	Stroke in evolution	1	—
Group 4	Clinical infarct	6	2
	Clinical ischaemia	12	—
	Stroke in evolution	—	—

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