Intracranial arteriovenous malformations

Observations after experience with computerised tomography

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SYNOPSIS Thirty-six patients with angiographically confirmed intracranial arteriovenous malformations have had computerised tomographic scans performed as part of their investigation. This study demonstrates the incidence of haematoma formation after haemorrhage, the frequency of calcification not visible on plain radiographs, and describes the possible causes for a complicating hydrocephalus. Further information has been gained from the intravenous injection of sodium iothalamate (Conray 420), with comparison of the scans taken before and after the injection.

Intracranial arteriovenous malformations (AVM) diagnosed at this hospital account for 12% of all cases presenting for investigation of subarachnoid haemorrhage. A total of 565 patients were seen between 1954 and 1968 and detailed reports on the presentation, mode of progression, and surgical treatment of this condition have been published from here and other units (Paterson and McKissock, 1956; Forster *et al.*, 1972). Diagnosis in the past has depended on cerebral angiography with further information from necropsy studies. However, the introduction of computerised tomography (CT scanning) has given us a non-invasive technique with which we can not only diagnose these lesions but also learn more about their pathophysiology.

METHODS

The present study concerns the CT scans on all patients with cerebral AVMs investigated between February 1974 (when the 160×160 matrix became available) and February 1976. It has been our custom to submit all patients presenting with a suspected intracerebral haematoma to CT scanning before angiography. Patients presenting for reasons other than haemorrhage were usually referred for exclusion of a cerebral tumour and they too had CT scanning performed as part of their routine neuroradiological investigation. One third of the patients studied also received 60 ml intravenous sodium iothalamate (Con-

ray 420). This was given slowly over five minutes and the patients reported no ill effects except for brief sensations of nausea and flushing. All the patients described in this study had the diagnosis of a cerebral AVM confirmed by angiography.

RESULTS (Table 1)

LOCATION

Twenty-eight AVMs were situated above the tentorium and eight were in the posterior fossa.

PRESENTATION

Twenty-eight patients presented with intracranial haemorrhage, five with epilepsy alone and two because of symptoms suggestive of a progressive hydrocephalus. One patient (in whom a brain stem angioma had been diagnosed after a haemorrhage many years previously) was referred for further investigations because he was developing symptoms and signs of a progressive brain stem disturbance in the absence of a further bleed.

CT SCAN FINDINGS

Haematoma Twenty-four of the 28 patients presenting with symptoms and signs suggestive of intracranial haemorrhage showed evidence on their CT scans of an intracerebral haematoma (distortion of the local anatomy by a mass whose density was in the appropriate EMI unit range). Two patients (numbers 6 and 18) were found at angiography to have berry

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TABLE 1
DETAILS OF CT SCAN FINDINGS IN PATIENTS WITH CEREBRAL AVMS

Patient	Age (yr)	Site of AVM	Presentation	Haematoma	Pre-Conray density change		Ventricular distortion*	Hydrocephalus	Conray given	Change with Conray
					High*	Low*				
1	33	Th	Ha	Yes	—	—	—	Yes	No	—
2	39	Th	Ha	Yes	—	—	—	Yes	Yes	Yes
3	55	P	Ha	Yes	—	—	—	No	Yes	Yes
4	11	Th	Ha	Yes	—	—	—	No	No	—
5	7	P	Ha	Yes	—	—	—	No	No	—
6†	51	T	Ha	No	Yes	No	Yes	Yes	No	—
7	11	C	Ha	Yes	—	—	—	No	No	—
8	49	P	Ha	Yes	—	—	—	No	No	—
9	35	CN	Ha	Yes	—	—	—	No	No	—
10	19	C	Ha	Yes	—	—	—	Yes	No	—
11	14	P	Ha	Yes	—	—	—	Yes	Yes	Yes
12	42	C	Ha	Yes	—	—	—	Yes	No	—
13	19	O	Ha	Yes	—	—	—	No	Yes	Yes
14	40	BS	Ha	Yes	—	—	—	No	Yes	No
15	18	Th	Ha	Yes	—	—	—	No	No	—
16	57	Th	Ha	Yes	—	—	—	No	No	—
17	20	Th	Ha	Yes	—	—	—	Yes	No	—
18†	52	T	Ha	No	No	No	No	No	No	—
19	14	F	Ha	Yes	—	—	—	No	No	—
20	31	O	Ha	Yes	—	—	—	No	No	—
21	8	F	Ha	Yes	—	—	—	No	No	—
22	51	T	E	No	No	Yes	Yes	No	Yes	No
23	25	F	E	No	No	Yes	No	No	Yes	Yes
24	17	BS	Hy	No	No	No	Yes	Yes	No	—
25	54	O	E	No	Yes	No	No	No	Yes	Yes
26	54	P	Ha	Yes	—	—	—	No	No	—
27	27	BS	PD	No	Yes	No	No	No	Yes	Yes
28	54	P	Ha	Yes	—	—	—	No	No	—
29	54	C	Hy	No	Yes	No	Yes	Yes	Yes	Yes
30	60	C	Ha	No	No	No	No	No	Yes	Yes
31	27	P	Ha	Yes	—	—	—	No	No	—
32	4	P	Ha	Yes	—	—	—	No	No	—
33	33	P	E	No	Yes	Yes	Yes	No	Yes	Yes
34	52	O	Ha	Yes	—	—	—	No	No	—
35	16	P	Ha	No	No	No	No	No	No	—
36	62	P	E	No	Yes	No	Yes	No	No	—

Th: thalamus. P: parietal. T: temporal. C: cerebellum. CN: caudate nucleus. O: occipital. BS: brain stem. F: frontal. Ha: haemorrhage. E: epilepsy. Hy: hydrocephalus. PD: progressive deficit.

*In the absence of CT scan evidence for haematoma.

†Patient also had a berry aneurysm.

aneurysms in addition to their AVMs. Both patients underwent surgery for these aneurysms, which in each case were found to have been the source of their haemorrhage. Neither of the two remaining patients underwent surgery so that the absence of haematoma has not been confirmed. Failure to detect blood clot was possibly due to technical factors in one case (patient 35) as her AVM lay close to the midline in the high parietal region.

PRE-CONRAY ABNORMALITIES IN ABSENCE OF CT SCAN EVIDENCE FOR HAEMATOMA

These were seen in eight out of a possible total of 12 patients. The most common appearance (six cases) was an area of patchy high density conforming to the

region of the angioma itself as shown by subsequent angiography. In one case (patient 33) this was superimposed on a surrounding region that was of a density lower than that of the adjacent brain. Another case (patient 22) showed only an area of low density that involved not only the region of the AVM itself but also the surrounding brain.

LOCAL VENTRICULAR DISTORTION (IN ABSENCE OF CT SCAN EVIDENCE OF HAEMATOMA)

There were six cases in this group. Two of them (patients 6 and 22) had presented with subarachnoid haemorrhage, the cause of which was noted at operation to have been a berry aneurysm but in neither case was the region of the angioma itself explored. Two

cases (patients 24 and 29) had bled five and 13 years respectively before this admission. Like the two remaining patients (numbers 33 and 36) there was nothing in their recent history, clinical findings, or results of lumbar puncture to suggest intracranial haemorrhage. None of these four cases was explored at operation.

HYDROCEPHALUS

Nine patients showed CT scan evidence of ventricular enlargement. Four patients (1, 2, 14, and 17) showed distortion of the third ventricle by their haematoma, while two (10 and 12) showed distortion of the fourth ventricle on their CT scans. Another three patients (6, 24, and 29) had hydrocephalus without CT scan evidence of haematoma.

CONRAY STUDY

Intravenous sodium iothalamate (Conray 420) was given to 12 patients, four of whom already showed evidence of haematoma on their scans. Ten scans showed an obvious change when they were repeated after the injection. These new scans exaggerated the appearance of the high density areas when they were already present (cases 25, 27, 29, and 33) and introduced them if they were not already obvious (because of haematoma in cases 2, 3, 13). In three cases the great vein of Galen could be seen on the post-Conray study and in each case it was shown at angiography to be greater than 1 cm in diameter (after allowing for magnification).

DISCUSSION

Paxton and Ambrose (1974) were the first to point out that a cerebral AVM could be visible on the CT scan. Additional examples have been given by Pressman *et al.* (1975). We have already shown in a study from this hospital (Hayward and O'Reilly, 1976) that the position of an intracerebral haematoma as demonstrated by the CT scan can be an accurate indication of the underlying pathology, but we also found that as many AVMs were missed as were predicted successfully. This inaccuracy reflects the wide distribution of the anatomical sites involved, but the presence of an intracerebral haematoma in a position not usually associated with aneurysmal rupture or primary hypertensive haemorrhage is highly suggestive of an underlying AVM, particularly when the haemorrhage has occurred in a young person. The average age at onset of symptoms in the 1976 study was 24 years and nearly 70% of the patients had had their first symptoms before they were 30. Only five patients (22, 23, 25, 33, and 36) presented during the course of this

study without either a history of past or present intracranial haemorrhage or a known diagnosis of cerebral AVM. In four it was possible for the radiologist to make a correct diagnosis on the basis of the CT scan and the short clinical history given when the investigation was requested. The other patient (case 22) was incorrectly diagnosed as a low grade glioma because the CT scan showed a large area of low density which failed to take up Conray 420 and which was compressing the adjacent ventricular system (Fig. 5). The true diagnosis was made at angiography and confirmed at operation.

The typical appearance of a cerebral AVM on the CT scan is of small discrete areas of high density as compared with the surrounding brain (Figs. 3 and 7). The cerebral tissue immediately adjacent to the angioma may, in fact, be of a lower density than normal (Fig. 2). Of the six patients demonstrating areas of increased density before the injection of Conray, only one (case 6) had calcification visible on his plain radiographs, demonstrating the increased sensitivity of the CT scan system to detection of changes in soft tissue density.

The injection of 60 ml sodium iothalamate can produce an obvious exaggeration of these initial high density changes (Figs. 1, 2, and 4). Cases 22 and 23 showed low density areas but the enhancement study failed to produce any significant change in the appearance of the former, while in the latter there appeared nearby a small area of increased density that was described by the reporting radiologist as being like a smoke ring (Fig. 6). Three out of the 12 AVMs studied after the injection of sodium iothalamate failed to show significant enhancement. As can be seen in Table 2, this did not appear to be related to the size of the lesion but more to the blood circulation time as judged by the angiogram. Thus in none of the three cases could the AVM itself be seen on an angiogram film taken later than the capillary phase. Three out of 12 cases is not a sufficiently large number from which to draw conclusions about an observation that does not appear to have an obvious theoretical basis. All the post-Conray scans were made immediately after the injection, without moving the patients from the machine but the possibility must exist that a change in head position had occurred. It would seem most likely that the degree of Conray enhancement should depend on the size of the blood pool within the lesion itself and its immediate draining veins and this would be independent of the speed of the pathological circulation.

Significant ventricular distortion due apparently to the angioma itself was seen in six cases. Intracerebral blood clot usually appears as an area of increased density on the CT scan but with the passage of time it may assume the same density as surrounding brain

FIG. 1 Case 3, who presented with intracranial haemorrhage, before (a) and after (b) the intravenous injection of Conray.

FIG. 2 Case 33, investigated after one epileptic attack—before (a) and after (b) Conray.

FIG. 3 Case 6. Calcification in the right temporal angioma.

before eventually ending up as a low density area or intracerebral cyst. In only one of these cases (patient 22, Fig. 5) was the area of the angioma itself explored

at operation and this revealed no evidence of haematoma. However, there was no evidence from the history, clinical findings, or results of lumbar puncture in these six patients to suggest recent haemorrhage from the AVM. The space occupying effect of the AVM could have been affected by previous haemorrhage in cases 24 and 29 (Fig. 7).

The true incidence and progression of hydrocephalus associated with non-traumatic intracranial haemorrhage will become apparent only as CT scanning is used more readily. With rupture of an aneurysm, hydrocephalus is usually of a communicating type and may follow impaired absorption of CSF due to blocking of the arachnoid villi (Ellington and Margolis, 1969). In this study it can be seen that all the cases of hydrocephalus had a definite degree of ventricular distortion, except one (patient 6) where the haemorrhage was subsequently found at operation to have been from a ruptured berry aneurysm. Two patients who presented with symptoms and signs of a progressive hydrocephalus, in the absence of further haemorrhage, required the insertion of a ventriculoperitoneal shunt.

Finally, it should be emphasised that the presence of signs of blood clot on the CT scan need not interfere with the ability of an underlying AVM to enhance after the injection of Conray. This extra procedure should always be considered in a case of intracerebral haemorrhage where an AVM is a clinical possibility.

FIG. 4 Case 30. Left cerebellar angioma before (a) and after (b) Conray.

FIG. 5 Case 22. Post-Conray study showing a fronto-temporal low density area with shift of the ventricular system to the left and no evidence of enhancement.

FIG. 6 Case 23. Post-Conray study. A small 'smoke ring' appearance is seen in the right posterior frontal area.

FIG. 7 Case 29. Pre-Conray study showing the patchy high density appearance of the angioma in the right cerebellar hemisphere, and the displacement of the fourth ventricle.

TABLE 2
DETAILS OF PATIENTS RECEIVING SODIUM IOTHALAMATE (CONRAY 420)

Patient	Angioma size (cm)			Site of Conray enhancement	Latest angiogram phase showing angioma
	Med/Lat	Ant/Post	Vert		
2	4.0	4.0	5.0	Angioma and great vein of Galen	Early venous
3	4.0	3.0	4.0	Angioma and great vein of Galen	Early venous
11	1.5	3.5	3.5	Great vein of Galen	Capillary
13	0.5	0.75	0.75	Angioma	Early venous
14	2.0	2.0	2.0	None	Late arterial
22	1.0	1.2	1.0	None	Late arterial
23	3.0	2.5	1.5	Angioma	Early venous
25	2.5	3.5	1.0	Angioma	Late venous
27	3.5	3.0	2.0	Angioma	Late venous
29	4.0	5.0	4.0	Angioma	Early venous
30	6.0	7.0	5.0	Angioma	Venous
33	4.5	4.0	2.5	Angioma	Early venous

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