

The Computerized Tomographic Appearance of Angiographically Occult Arteriovenous Malformations of the Brain

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SUMMARY: *Eight patients with a histologically proven angiographically occult arteriovenous malformation of the brain had plain and infused computed tomographic (CT) examinations. In five cases angiography revealed a hypovascular mass and in three cases the angiogram was normal. On CT examination a high density lesion (six cases) and ventricular asymmetry (five cases) were demonstrated. In three patients presenting with intracerebral hemorrhage, the high density appearance*

and ventricular compression were accounted for by the presence of hematoma. In three of five seizure patients the high density lesion was associated with calcification while ipsilateral (one case) and contralateral (one case) enlargement of the lateral ventricle was seen. In five of the eight cases the vascular nature of the lesion was suggested by vascular enhancement of the infused CT scan. Glioma was a common misdiagnosis.

RÉSUMÉ: *Chez 8 patients qui avaient une malformation artérioveineuse cérébrale silencieuse à l'angiographie, mais prouvée histologiquement nous avons fait une tomodensitométrie simple et une infusion. Dans 5 cas l'angiographie montrait alors la présence d'une masse hypovascularisée, mais dans 3 cas l'angiogramme fut normal. L'examen par tomodynamométrie révèle chez 6 cas une lésion à haute densité, et chez 5 cas une asymétrie ventriculaire. Chez 3 patients l'apparence de haute densité et la compression ventriculaire furent expliqués*

par la présence d'un hématome consécutif à une hémorragie intracérébrale. Parmi 5 patients avec crise épileptique, trois associaient la lésion à haute densité à une calcification; un examen de montra une augmentation ipsilatérale, et un autre une augmentation contralatérale du ventricule latéral. Pour 5 des 8 cas la nature vasculaire de la lésion fut suggérée par l'amélioration de l'image obtenue par l'infusion. Un diagnostic erroné commun fut celui de gliome.

INTRODUCTION

Since Bergstrand and co-workers (1936) demonstrated an arteriovenous malformation (AVM) of the brain by angiography, this procedure has been regarded as the *sine qua non* of the antemortem diagnosis of these lesions (Olivecrona and Riives, 1948; Bell et al, 1978). However, the vascular nature of AVMs is not always angiographically demonstrable (Bell et al, 1978). With the advent of computerized tomography (CT) scanning, the occurrence of these angiographically occult lesions has come to the fore (Kendall and Claveria, 1976; Bell et al, 1978; Golden and Kramer, 1978; Becker et al, 1979; Leblanc et al, 1979; Terau et al, 1979). We present the CT findings in eight cases of histologically verified AVMs where the angiograms failed to reveal a vascular lesion.

MATERIALS AND METHODS

Five males and three females between the ages of 14 and 68 years (mean age: 27.5 years) had angiographic and plain and infused CT examinations of the brain, on an EMI head scanner, for investigation of an intracerebral hemorrhage (ICH) or of a seizure disorder. The three patients with an ICH presented with hemiparesis (two cases), aphasia (one case), and severe headache (one case); while the five seizure patients presented with focal (four cases) and/or generalized seizures (four cases). The mean duration of seizure in the latter group was 8.4 years.

FINDINGS

The findings of ancillary investigations are presented in Table I.

Angiography

All the angiograms were performed

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TABLE 1
Ancillary Investigation In Eight Cases Of Angiographically Occult AVMs

Case, Age, Sex, Presentation	Skull X-ray	EEG	Brain Scan	PEG
AJ, 26, Male, ICH	Shift of the Pineal	Focal Disturbance	Focal Uptake	—
MC, 68, Male, ICH	Normal	—	Focal Uptake	—
SD, 29, Female, ICH	Normal	—	—	—
RJ, 21, Female, Sx	Normal	Focal Epileptiform Disturbance	Focal Uptake	—
DG, 17, Male, Sx	Asymmetry of the Middle Fossa	Focal Disturbance	Normal	—
MG, 19, Female, Sx	Small Left Middle Fossa	Focal Epileptiform Disturbance	Normal	Enlarged Left Lateral Ventricle
KR, 14, Male, Sx	Thick Featureless	Diffuse Disturbance	—	Enlarged Right Frontal and Left Temporal Horns
GS, 16, Male, Sx	Normal	Focal Epileptiform Disturbance	—	—

Legend: AVM: Arteriovenous Malformation; EEG: Electroencephalogram; PEG: Pneumoencephalogram; ICH: Intracerebral Hemorrhage; Sx: Seizure Disorder.

TABLE 2
Angiographic, CT and Histological Findings In Eight Cases Of Angiographically Occult AVMs

Case Presentation	Angiogram	Plain CT	Infused CT (Suggested Diagnosis)	Pathology
AJ ICH	Avascular Mass.	High Density Lesion; Rim of Edema; Compressed Ipsilateral Lateral Ventricle.	Vascular Enhancement	Small Partially Thrombosed AVM with Hemosiderin and Reactive Changes.
MC ICH	Hypovascular Mass.	Mixed Density Lesion; Rim of Edema; Compressed Ipsilateral Lateral Ventricle.	Vascular Enhancement. (Glioma)	Small Partially Thrombosed AVM with recent Hemorrhage
SD ICH	Avascular Mass.	High Density Lesion; Compressed Ipsilateral Lateral Ventricle.	Vascular Enhancement.	Small Partially Thrombosed AVM with recent Hemorrhage
RJ Sx	Avascular Mass.	High Density Lesion.	No Enhancement. (Hamartoma vs Granuloma vs Glioma)	Large Thrombosed and Calcified AVM with Hemosiderin and Gliosis.
DG Sx	Space occupying Lesion; no AVM demonstrated.	High Density Lesion; Calcification.	No Enhancement.	Small Partially Thrombosed AVM with Psammoma Bodies.
MG Sx	Normal	High Density Lesion; Enlarged Ipsilateral Lateral Ventricle.	Vascular Enhancement. Calcification. (Glioma)	Small AVM with Hemosiderin Neuronal Loss, Gliosis and Partial Thrombosis.
KR Sx	Large Posterior Communicating Artery. No AVM demonstrated.	High Density Lesion; Enlarged Contralateral Lateral Ventricle.	Vascular Enhancement.	Large Thrombosed AVM with Hemosiderin.
GS Sx	Normal	Normal	No Enhancement	Small AVM with Gliosis.

Legend: AVM: Arteriovenous Malformation; ICH: Intracerebral Hemorrhage; Sx: Seizure Disorder; CT: Computerized Tomography.

at four films per second. The findings on angiography are presented in Table 2. Five examinations including three in the ICH group demonstrated a hypovascular mass. Two examinations were entirely normal, while another, a three cc angiogram performed for speech amytal testing as part of a seizure work-up (Wada and Rasmussen, 1960), showed only a large posterior communicating artery.

CT Scanning

The findings on plain and infused CT scanning are presented in Table 2 and summarized in Table 3. One scan in a seizure patient was normal. A high density lesion was seen in six cases and a mixed density lesion in another. A mass effect was seen as compression of the ipsilateral lateral ventricle in the three cases comprising the ICH group. Two of these lesions were surrounded by a low density area interpreted as a rim of edema. Ventricular asymmetry was present in five cases, from compression of the ipsilateral lateral ventricle in the three ICH, and from enlargement of the ipsilateral lateral ventricle in one seizure patient and enlargement of the contralateral lateral ventricle in another. In five cases further information, indicating an abnormal vasculature by vascular enhancement, was obtained after the intravenous injection of contrast material (Huckman, 1975); while in three seizure patients enhancement did not occur.

Histological Examination

All cases were histologically proven according to the usual criteria (Russell and Rubinstein, 1977). Five AVMs were associated with hemosiderin deposition. Three AVMs in the seizure group were associated with calcification, while three in the ICH group showed recent hemorrhage (Table 3). All the lesions showed extensive thrombosis.

Location of the AVMs

The AVMs were supratentorial and localized as follows: left frontal region (RJ); right supplementary motor cortex (DG); left pre-central region (KR); right insular region (SD); first left temporal gyrus (MG); second left

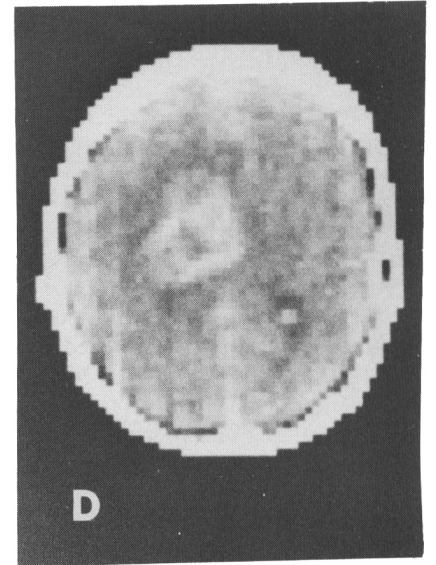
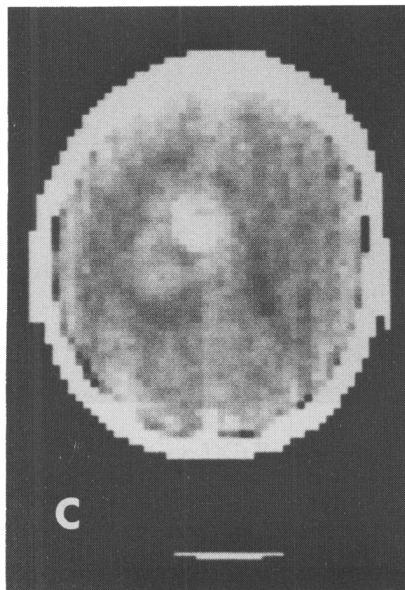
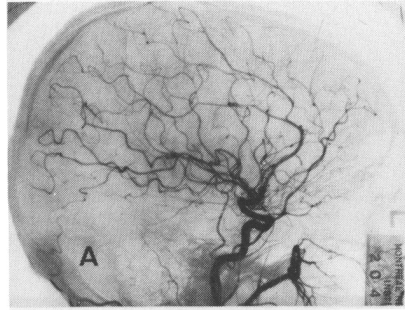


Figure 1 — (Case AJ) Left internal carotid angiograms and plain and infused CT scans of a 26 year old man presenting with the acute onset of headache and right hemiparesis. Angiography (a and b) revealed the presence of a space occupying lesion in the left frontal region but failed to demonstrate an AVM. The plain CT scan (c) shows a dense lesion surrounded by a rim of edema compressing the ipsilateral lateral ventricle, that enhanced with infusion (d). Histological examination identified a small thrombosed AVM and hemosiderin deposition (e). (x40)

temporo-occipital convolution (MC); left parietal area (AJ); first right temporal gyrus (GS).

DISCUSSION

The CT appearance of AVMs is well recognized (Pressman et al, 1975; Hayward, 1976; Terbrugge et al, 1977; Leblanc et al, 1979); and Bell (1978) has recently reviewed the pre-CT reports of angiographically occult AVMs. We have recently reported that 11% of 54 patients with an AVM seen at our institution over a five year period had angiographically occult lesions (Leblanc et al, 1979). Since then, two other cases have been encountered bringing the total to eight cases with CT correlation. This material forms the basis of this communication. In five cases, the angiogram, although abnormal, did not reflect the vascular nature of the lesion, while three studies were normal. Seven CT examinations were abnormal, showing a high or mixed density lesion in 87% of cases and ventricular asymmetry in 62%. Contrary to the angiographic appearance was suggested by the CT finding of vascular enhancement in 62% of the cases. The diagnosis of AVM, however, was not entertained because of the negative angiographic findings. Common misdiagnoses on the basis of the CT appearance were a glioma (three cases) hamartoma or granuloma.

The presence of a high density lesion on the plain CT scan of a large proportion of patients with an AVM has been noted by all reviewers (Pressman et al, 1975; Terbrugge et al, 1977; Leblanc et al, 1979). In our patients this was accounted for by the presence of a hematoma in three cases and by calcification in three others. The nature of the high density in the other case (KR) may be explained by the presence of a large thrombosed AVM. The associated hemosiderin deposition may also be pertinent. Others have suggested that the high density appearance may reflect a relative increase in blood volume within the lesion (Pressman et al, 1975). This is probably not the case in our patient. Ventricular asymmetry was a prominent finding; it is explained by a mass effect in the ICH group. In

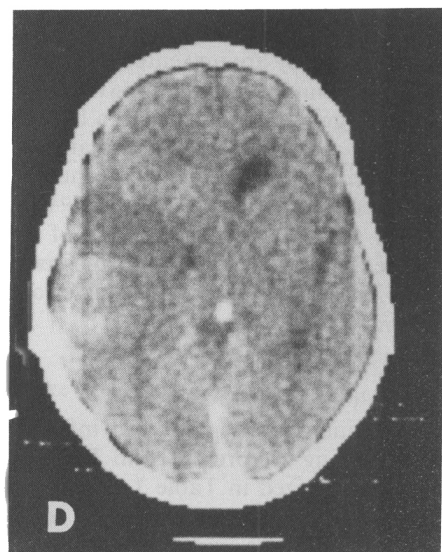
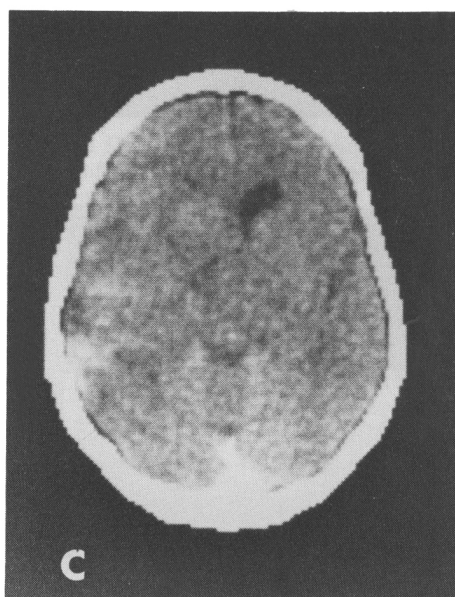
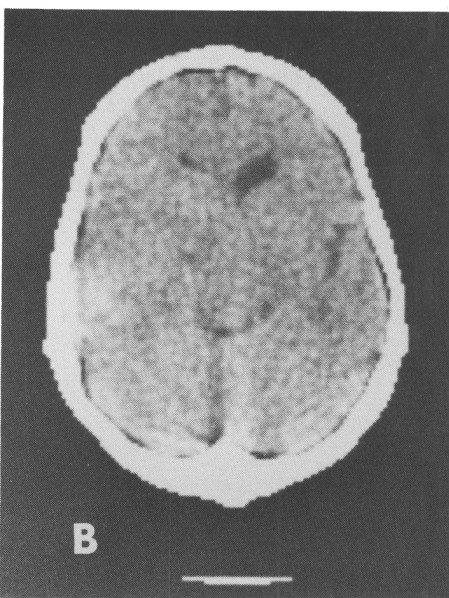
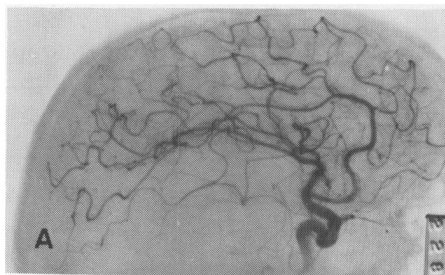


Figure 2 — (Case MC) Left internal carotid angiogram and plain and infused CT scans of a 67 year old man presenting with aphasia. The angiogram (a) demonstrates the presence of a hypovascular mass displacing the middle cerebral artery, without demonstrating abnormal vascularity. The plain CT scan (b), done nine days after the onset of symptoms, shows a mixed density lesion of the left posterior temporal and parietal areas surrounded by a rim of edema and compressing the left lateral ventricle. The infused CT scan (c), done two days later, shows vascular enhancement of this lesion. An infused CT scan (d), performed three weeks later, shows enlargement of the lesion. Histological examination revealed a partially thrombosed AVM of the second left temporal convolution and recent hemorrhage (e). (x40)

TABLE 3
CT Findings In Eight Cases Of Angiographically Occult AVMs

CT Findings	No. of Cases
High Density Lesion with Mass Effect	2
Mixed Density Lesion with Mass Effect	1
High Density Lesion without Mass Effect	4
Rim of Edema	2
Ventricular Asymmetry	5
Normal	1
Enhancement with Infusion	5

Legend: AVM: Arteriovenous Malformation; CT: Computerized Tomography.

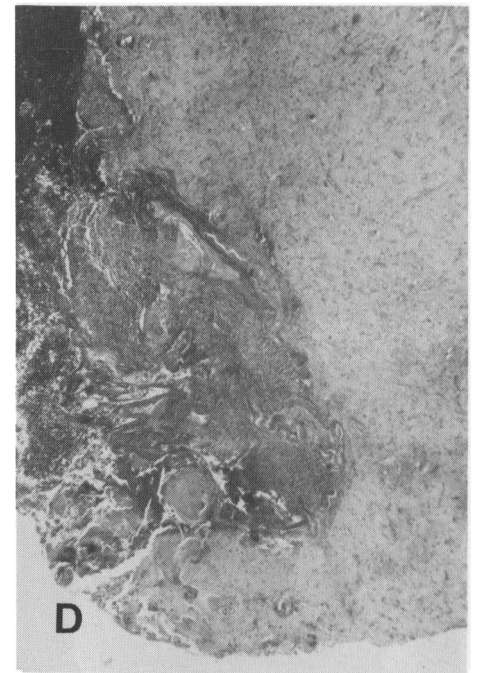
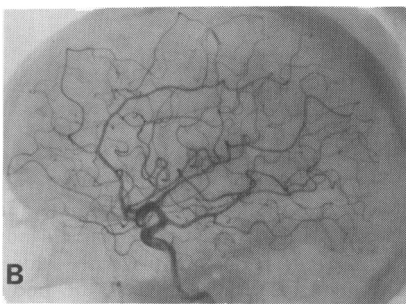
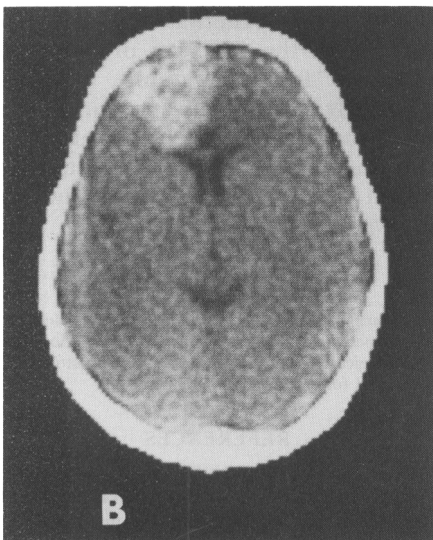
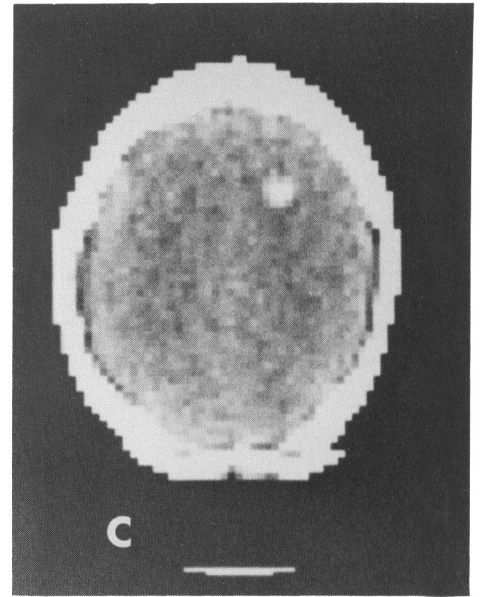
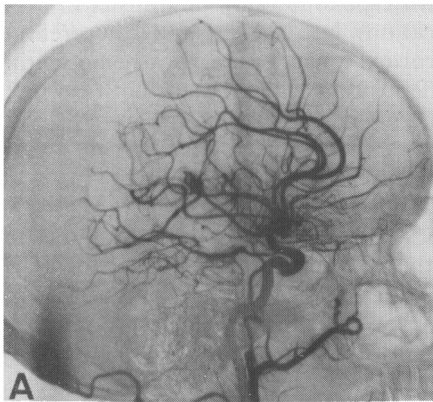


Figure 3 — (Case RJ) Left carotid angiogram and plain CT scan of a 21 year old woman with an 18 year history of focal seizures involving the right arm and leg. The arteriogram (a) revealed the presence of a hypovascular lesion of the left frontal lobe, without mass effect or abnormal vascularity. The plain CT scan (b) demonstrated a non-enhancing hyperdense lesion of the left frontal lobe. Histological examination revealed a thrombosed and calcified AVM. (x40)

Figure 4 — (Case DG) Right internal carotid angiograms and plain CT scan of a 15 year old boy with a two and one half (2½) year history of focal seizures involving the left arm. AP (a) and lateral (b) arteriograms are normal. The plain CT scan (c) shows a hyperdense lesion of the right frontal lobe that does not enhance with infusion. Histological examination revealed the presence of a small partially thrombosed AVM of the right pre-central area with psammoma bodies (d). (x40)

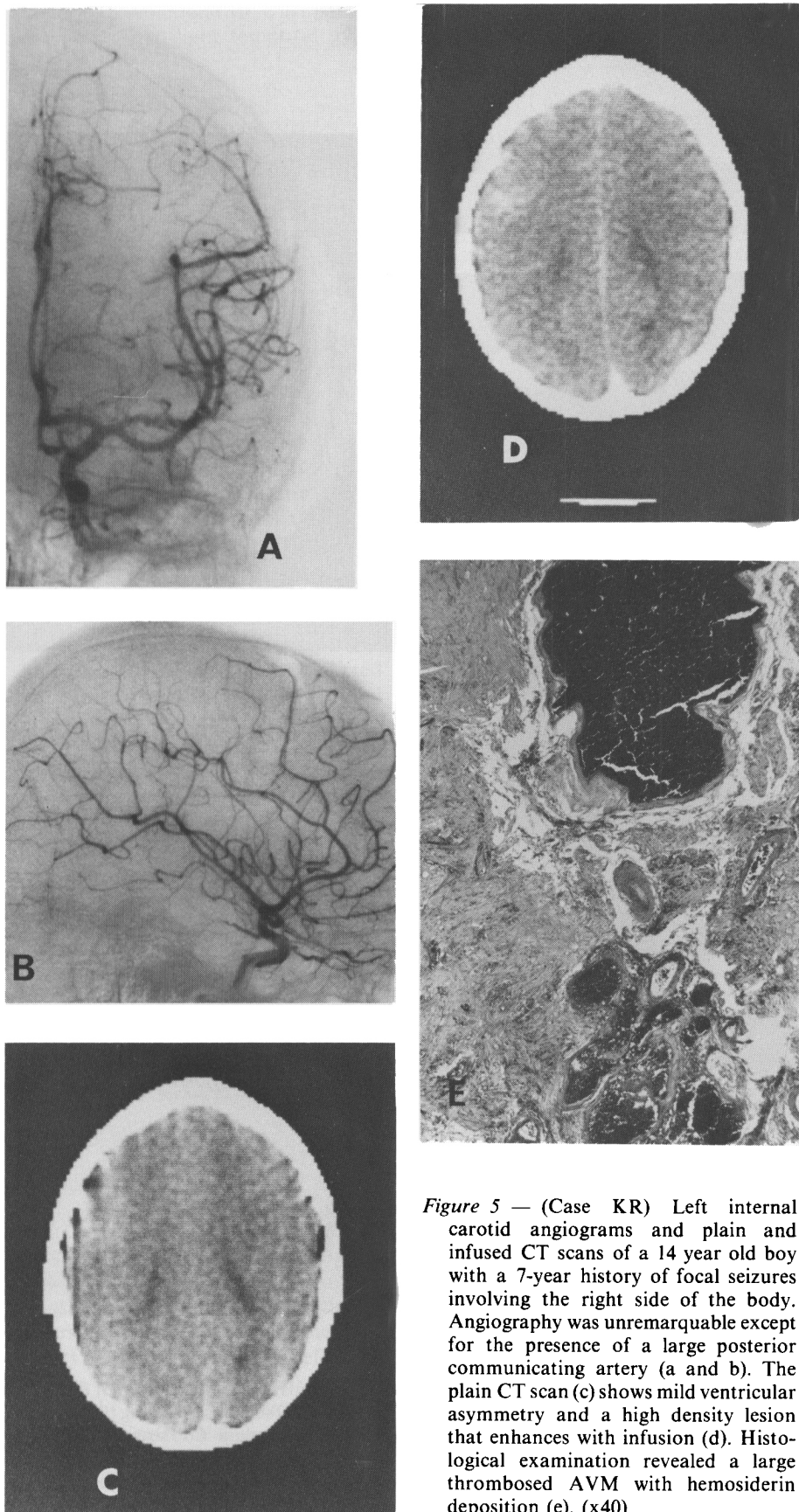


Figure 5 — (Case KR) Left internal carotid angiograms and plain and infused CT scans of a 14 year old boy with a 7-year history of focal seizures involving the right side of the body. Angiography was unremarkable except for the presence of a large posterior communicating artery (a and b). The plain CT scan (c) shows mild ventricular asymmetry and a high density lesion that enhances with infusion (d). Histological examination revealed a large thrombosed AVM with hemosiderin deposition (e). (x40)

one seizure patient there was enlargement of the ipsilateral lateral ventricle. This may reflect focal atrophy secondary to chronic ischemia from shunting of blood within the lesion as a "cerebral steal phenomenon", as demonstrated by Feindel and co-workers (Feindel et al, 1971; Feindel, 1975). The explanation of contralateral lateral ventricle enlargement in another seizure patient is not apparent. Three of the seizure patients had associated hemosiderin deposition. Recently, Sybert and Willmore (1978) have produced electrographic and clinical seizures with the iontophoretic transfer of iron cations into the rat and cat cerebral cortex. It may be that focal hemosiderin deposition contributed both to the high density appearance on the CT scan and to the development of a seizure disorder in some of our patients.

CT scanning is a useful adjunct to the investigation of patients with ICH or seizures. An underlying AVM is suspected, even when the vascular nature of the lesion is not demonstrated angiographically, when there is vascular enhancement of a high density lesion. Alternatively, a similar picture on the CT scan provides an incentive for angiography. This is especially so when a prominent or abnormal vessel is demonstrated, as was the case in 16% of patients in a recent study of the CT appearance of AVMs (Leblanc et al, 1979).

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