

Giant Intracranial Aneurysms of the Anterior Circulation: Clinical Characteristics and Diagnosis by Computed Tomography

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The computed tomography (CT) characteristics and clinical features of giant (globoid) aneurysms of the anterior circulation are reviewed. These lesions appear on the CT scan as smoothly encapsulated ovoid masses, within which a partially patent lumen is seen after the infusion of iodinated contrast material. With careful analysis of the CT scan it may be possible to differentiate giant internal carotid artery, anterior cerebral artery, and middle cerebral artery aneurysms from other parasellar and hemispheric lesions.

Key words: CT scan, Giant (globoid) aneurysms

INTRODUCTION

With the advent of the computed tomography (CT) scan, physicians have been able to solve difficult neurological diagnostic problems with relative ease and with no untoward risk to their patients. Giant or globoid intracranial aneurysms are no exception. The site, size, and, in some instances, the exact nature of these lesions can be ascertained with the CT scan. This paper reviews the recent experience at the Massachusetts General Hospital with patients harboring giant aneurysms of the anterior circulation, emphasizing the role of computed tomography in the diagnosis of and therapy for such lesions.

MATERIALS AND METHODS

Twelve patients admitted to the Massachusetts General Hospital for the evaluation of suspected parasellar or hemispheric mass lesions between October 1974 and October 1977 were found to have giant intracerebral aneurysms. Radiographic diagnosis of these lesions, measuring 2.5 cm or more in diameter, was established by means of CT scans and cerebral angiography (7). CT scans were initially performed on the EMI (EMI Ltd., X-ray Systems Division, Hayes, Middlesex, England) modified Mark I brain scanner (160 × 160 matrix); thereafter, the EMI 1005 unit (160 × 160 matrix) was used. One scan was performed with the EMI 80 × 80 matrix scanner. Contrast-enhanced scans were performed immediately after the intravenous infusion of 300 ml of 30% meglumine diatrizoate (Reno-M-DIP; E. R. Squibb and Sons, Princeton, New Jersey).

RESULTS

The results of CT scans in the 12 patients are recorded in Table 1. Such CT scans performed after the administration of iodinated contrast material show a characteristic enhanced aneurysmal wall within which is seen a vascular lumen defined by the high density attenuation coefficients of the blood iodine pool juxtaposed to the relatively lower density attenuation coefficients of the nonenhancing intraluminal clot. Evidence of bone erosion may also be seen.

The exact mechanism of the giant aneurysmal wall enhancement is unknown. We suspect that it is due to the iodine circulating within the neovascularized wall, a result of progres-

sive intraluminal thrombosis and recanalization with reabsorption of the clot and growth of the lesion by scar formation. Intraclinoid aneurysms may have an additional pseudovasovascular derived from the lamina dura or the wall of the contiguous cavernous sinus.

CASES AND DISCUSSION

Giant aneurysms of the infraclinoid internal carotid artery

Giant internal carotid artery aneurysms usually produce neurological symptoms by compressing adjacent structures. Because of their thickened dural coverings within the cavernous sinus, infraclinoid aneurysms rarely rupture into the cerebral substance. However, rupture of an infraclinoid aneurysm may result in a carotid-cavernous sinus fistula.

In Jefferson's classical description of giant aneurysms of the internal carotid artery, he divided infraclinoid intracavernous aneurysms into three groups on the basis of their local mass effect upon the three sensory branches of the trigeminal nerve (2, 3). Anterior lesions result in pain and sensory loss in the skin supplied by the ophthalmic division, whereas centrally and caudally placed cavernous lesions cause pain and sensory loss in the distribution of the maxillary and mandibular divisions, respectively. Regardless of the anteroposterior position of the lesion, ipsilateral trochlear and oculomotor nerve palsies as well as motor trigeminal palsy are usually present. The optic nerve is usually unaffected by the infraclinoid aneurysm unless the lesion extends rostrally to compress the ipsilateral nerve and chiasm. It may also extend posterolaterally into the middle fossa to compress the greater superficial petrosal nerve, which carries special visceral afferent stimuli from the ipsilateral anterior two-thirds of the tongue (13).

Jefferson drew attention to some useful details for detecting such aneurysms on plain skull films. Widening of the superior orbital fissure with erosion of the optic strut was taken to be almost pathognomonic of this lesion (3).

The characteristic CT scan findings in four cases are recorded in Table 1. All showed erosion of the anterior clinoid process and sphenoid bone as well as an increase in absorption values of the capsule and lumen and a significant volume of extraluminal thrombosis. The following case history illustrates these points.

TABLE 1
The CT Characteristics of Giant Anterior Circulation Aneurysms

Case	Location of Aneurysm	Increase in Peak Absorption Values with Contrast Material		Amount of Intraluminal Thrombosis (%)	Bone Abnormalities/Miscellaneous
		Capsule	Lumen		
1	Intracavernous carotid	10	20	35	Ipsilateral anterior clinoid, medial sphenoid, and sella erosion
2	Intracavernous carotid	5	15	80	Anterior clinoid process and sphenoid bone erosion; 2-mm proptosis
3	Intracavernous carotid	15	17	75	Anterior clinoid process, sella floor and medial sphenoid bone erosion; 4-mm proptosis
4	Intracavernous carotid	18	22	80	Anterior clinoid process and ipsilateral sphenoid bone erosion
5	Supraclinoid carotid	11	19	0	Posterior clinoid process erosion; obliterated surrounding cisterns
6	Supraclinoid carotid	20	25	0	No bone abnormalities; enlargement surrounding subarachnoid cisterns
7	Supraclinoid carotid	13	18	20	Undercutting of anterior clinoids and sella floor erosion, left greater than right
8	Supraclinoid carotid	4	10	50	No bone changes; atrophy of surrounding brain tissue
9	Supraclinoid carotid	10	25	75	No bone changes; calcification of posterior capsule
10	Middle cerebral bifurcation	29*	45*	80	No bone changes; subacute subdural hematoma with massive subfalcine herniation
11	Middle cerebral bifurcation	8	20	20	No bone abnormalities
12	Anterior cerebral	7	17	70	Right posterior clinoid and dorsum sella erosion

* Contrast-enhanced scan performed after 74.0 g of iodine instead of 42.3 g of iodine.

Case 2. In March 1973, this 67-year-old woman presented with painful diplopia on full upward gaze. This was thought to be due to a left superior rectus palsy. Plain films of the skull and optic canals were normal. Two months later an ipsilateral abducens palsy was detected. "Bubbling in the left ear" with persistent diplopia on upward gaze and findings of left-sided proptosis of 2.5 mm and an early left afferent pupillary defect prompted a second set of skull films, including special views of the superior orbital fissure and optic canals, which were described as normal. A Tensilon (edrophonium chloride; Roche Laboratories, Nutley, New Jersey) test was negative. Thyroxine assays were within normal limits.

In September 1974 "prickling" dysesthesia in the skin and mucosa of the ipsilateral mandibular branch of the trigeminal nerve resulted in the patient's transfer to the Massachusetts General Hospital. Visual acuity (V/A) was 20/25 OU; color vision and perimetry testing were normal.

Multidirectional thin section laminography (polytomography) of the optic canals documented a left parasellar curvilinear calcification consistent with an infraclinoid carotid artery aneurysm within the left cavernous sinus. A plain and contrast-enhanced CT scan performed in October 1974 on the Mark I EMI (80 × 80 matrix) scanner revealed a left parasellar enhancing curvilinear partially calcified rim measuring up to 50 EMI units, within which were areas of lesser nonenhancing density, ranging between 30 and 47 EMI units. There was no displacement of the ventricular system. An angiogram confirmed the CT diagnosis but underestimated the true size of the lesion. An operation was recommended but was refused by the patient. A subsequent CT scan in June 1977 again documented the left parasellar curvilinear mass. The ringlike capsule, which measured nearly 4 cm in diameter, enhanced but not as prominently as did the small nonthrombosed lumen. About 85% of the aneurysm seemed to have thrombosed spontaneously. Erosion of the superior orbital fissure, the left

anterior clinoid process, and the ipsilateral aspect of the sella turcica was also noted on this scan (Fig. 1).

Giant aneurysms of the supraclinoid internal carotid artery

When the giant aneurysm arises from the supraclinoid carotid, central scotoma with rapidly failing vision in the ipsilateral eye usually occurs. Occasionally, pressure exerted from below on the crossing optic fibers (from the nasal portion of the contralateral retina in the chiasm) at the so-called loop of Wilbrand and Saenger causes an homonymous hemianopic field defect (12). The temporal defect in the opposite eye is first noted as a superior quadrantic defect.

The results of CT scanning in five patients are recorded in Table 1. The characteristic changes expected after contrast enhancement were seen, but the volume of extraluminal clot was less in this group. Bone changes were seen in some cases. An illustrative case report follows.

Case 5. A 69-year-old retired woman presented in June 1972 with painless blurred vision of 6 months duration in the left eye ("when looking at colored lights"). V/A was 20/20 OD and 20/80 OS corrected. Visual field testing documented a superior temporal quadrantic defect OD and a complete nasal hemianoptic defect OS with a dense central scotoma. Extraocular motions, sensation in the trigeminal distribution (skin and mucosa), motor trigeminal function, taste, and smell were all intact. A left supraclinoid giant aneurysm was documented by angiography on October 25, 1972. Left common carotid ligation was not performed because of a previous left hemispheric stroke. Since that time the patient's visual acuity and field defects have not changed.

A CT scan in July 1976, without and with CE, revealed that the lesion extended to the left of the sella inferiorly and to the right of the sella superiorly, with enhancement of the surrounding cisterns. No bony abnormalities were noted. The smooth

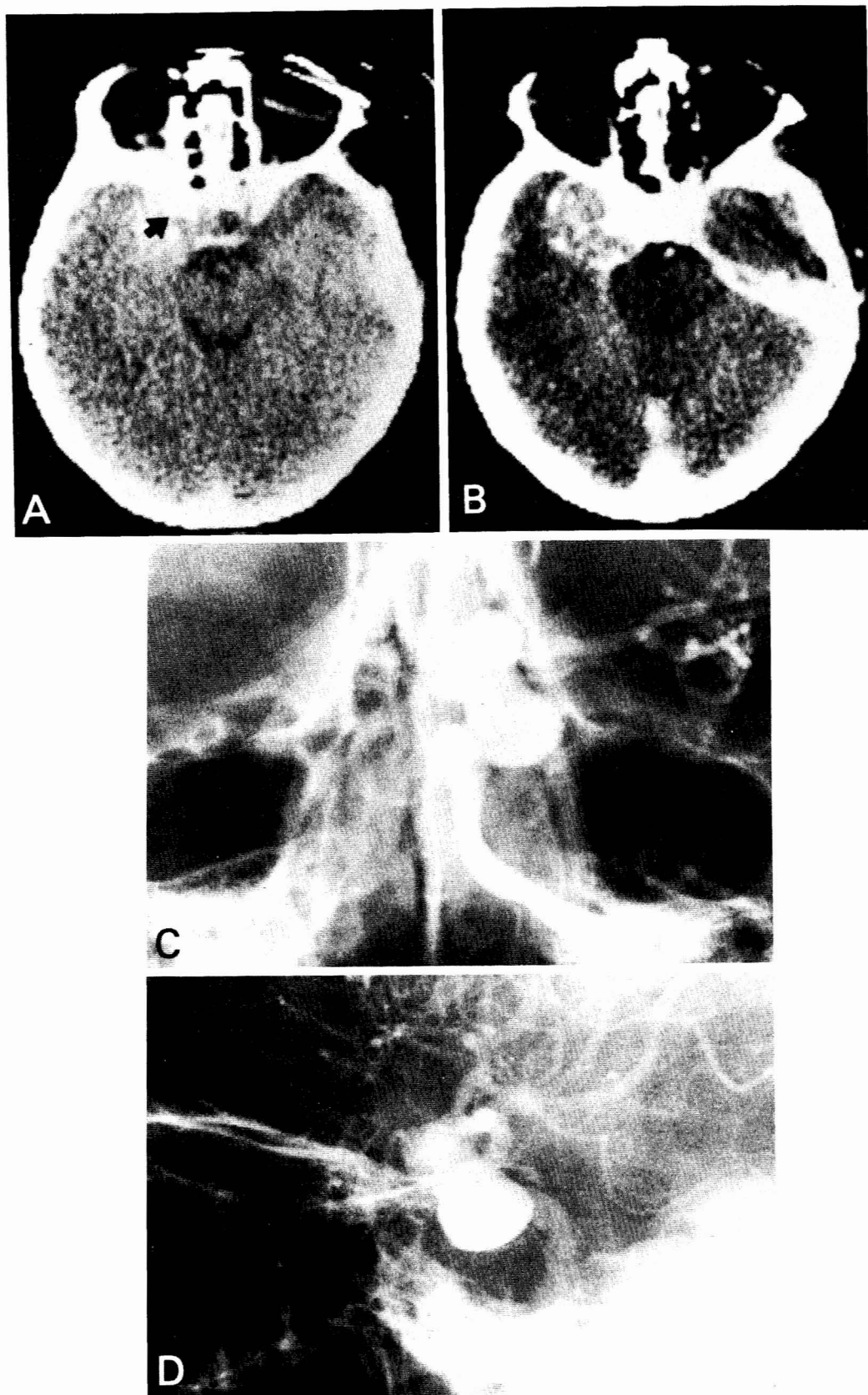


FIG. 1. Case 2. *A*. CT scan, plain study. A heterogeneous area of elevated absorption values is noted in the left parasellar region. The bony abnormalities are not well seen on the settings used for this illustration. *Arrow*, erosion of the left anterior clinoid process. *B*. after intravenous contrast infusion, there is enhancement of the capsule of the aneurysm. A smaller area of homogeneous contrast enhancement in the immediate left parasellar region, which represents iodinated contrast material within the patent lumen of the cavernous carotid aneurysm, is shown. The more lateral thrombosed portion of the aneurysm shows slightly elevated absorption values but no contrast enhancement. *C*. left carotid angiogram, Caldwell view. The nonthrombosed sac of the aneurysm in the left cavernous region is shown to be taking origin from the internal carotid artery. *D*. left carotid angiogram, lateral view. This shows the nonthrombosed portion of the aneurysm, which correlates with the parasellar contrast enhancement seen on the CT scan, but underestimates the total size of the aneurysm.

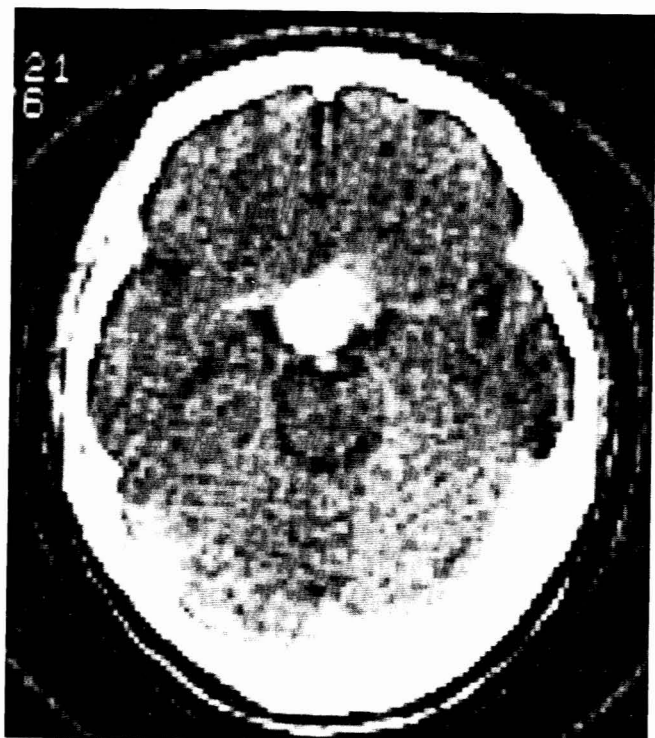


FIG. 2. Case 5. CT scan after intravenous infusion of contrast material. The area of homogeneous contrast enhancement representing circulating iodine within the lumen of the aneurysm extends farther to the right side as well as anteriorly in the suprasellar region. There is no distinction in the absorption values between the contrast material within the lumen of the aneurysm and the capsule as seen on this illustration.

capsule enhanced from a plain peak value of 30 EMI units to a peak of 50 EMI units after the intravenous infusion contrast material, and the blood pool within the lesion enhanced to a peak value of 55 EMI units (Fig. 2).

Occasionally supraclinoid aneurysms may present as an intrasellar mass lesion. For an aneurysm to be mistaken for a pituitary tumor or intrasellar craniopharyngioma, it must deform the sella turcica, compress the optic chiasm in such a way as to cause a bitemporal field defect, and sometimes produce progressive hypopituitarism without revealing itself on x-ray films by an aneurysmal type of calcification in its wall or clinically with episodes of subarachnoid hemorrhage or painful ocular palsies. Kümmell first recognized this rare entity more than 60 years ago (4). In their review of this subject in 1961, White and Ballantine stated that slightly greater than 1 of every 100 anterior circulation aneurysms treated at the Neurosurgical Service of the Massachusetts General Hospital masqueraded as a pituitary adenoma or craniopharyngioma with suprasellar extension (14). Because of this, arteriography became a routine procedure before an operative intervention in this region. We now believe that the CT scan may obviate the need for routine angiography in the preoperative evaluation of patients with suspected pituitary adenomas and a symmetrically expanded sella turcica. Patients with a parasellar mass or an asymmetrically expanded sella turcica still require cerebral angiography to help make the correct diagnosis and guide appropriate treatment. Before we adopted this policy, an error in diagnosis was made, as illustrated by the following case.

Case 6. A 50-year-old woman was referred in December 1972 for evaluation of an asymptomatic enlargement of the sella turcica. Endocrinological evaluation was normal except

for a history of galactorrhea while she was on an antidepressant medication in prior years. Neuro-ophthalmological evaluation was normal. Polytomography of the sella turcica revealed erosion of the lamina dura, greater on the left side of the sella floor. The sella volume was 1620 cu mm. The optic canals and superior orbital fissures were normal. A pneumoencephalogram did not reveal suprasellar extension, and in December 1972 she underwent a course of radiation therapy.

One year later she was readmitted with the complaint of painless "blurred vision in the periphery." Examination revealed a bitemporal hemianoptic defect with a central scotoma OS; V/A was 20/40 OD in the nasal field; V/A in the nasal field OS was finger counting at 1 foot. There was temporal pallor in the left optic disc. Extraocular motions were full. Motor and sensory trigeminal functions were normal. A diagnosis of chiasmal compression within the left anterior notch was made. At that time endocrinological evaluation disclosed a borderline low T-4 and a depressed a.m. serum cortisol level (1 mg/100 ml).

A CT scan performed in January 1977 revealed slight homogenous enhancement of a high absorption abnormality in the sella and suprasellar regions. There was erosion of the tuberculum sellae and the anterior clinoids as well as extension of the lesion with downward sloping of the sella floor toward the left parasellar region. An unusually smooth left parasellar margin of the lesion was noted on axial and coronal sections of a CT scan made after the intrathecal injection of metrizamide, the significance of which was not appreciated. The suprasellar cisterns were effaced, but the 3rd ventricle was not displaced. The lateral ventricular size was also normal. A CT diagnosis of chromophobe adenoma was made (Fig. 3).

Trans-sphenoidal decompression was aborted when the surgeon aspirated arterial blood from the lesion before opening the lamina dura of the sella floor. An intraoperative arteriogram confirmed the presence of a giant intrasellar aneurysm arising from the left supraclinoid carotid artery. Subsequently the patient underwent left internal carotid artery occlusion with a Crutchfield clamp, and successful thrombosis of the aneurysm was confirmed by subsequent angiography and CT scanning (by loss of contrast enhancement in the previously nonthrombosed lumen).

Errors in the radiographic diagnosis of this and one other case to be discussed later (Case 12) occurred because of our lack of appreciation of the asymmetrical erosion of the sella on plain films and tomography. In Case 6 our analysis of the CT scan photos sent from another hospital was incomplete because we were unable to evaluate fully the CT gray scale display. In Case 6 the smooth outer border of the lesion, representing the margin of the aneurysmal sac, and the blood-iodine absorption values, also suggesting possible aneurysm, were not appreciated. In Case 12 the small nonthrombosed lumen, which enhanced with the use of intravenous contrast material, went unnoticed.

Giant aneurysms of the middle cerebral artery

Patients harboring giant aneurysms of the middle cerebral artery frequently present with a slowly progressive contralateral hemiparesis with or without seizures due to a combination of direct brain compression secondary to mass effect and distal ischemia due to multiple episodes of intra-aneurysmal thrombosis with or without secondary distal embolization (5, 8, 11). The rapidity of evolution of the occlusive and recanalization processes determines whether distal ischemia progresses to frank cerebral infarction. This slowly progressive history is consistent, of course, with any hemispheric mass lesion. Because total or subtotal spontaneous thrombosis of giant aneurysms may occur (10), angiography may not be useful in

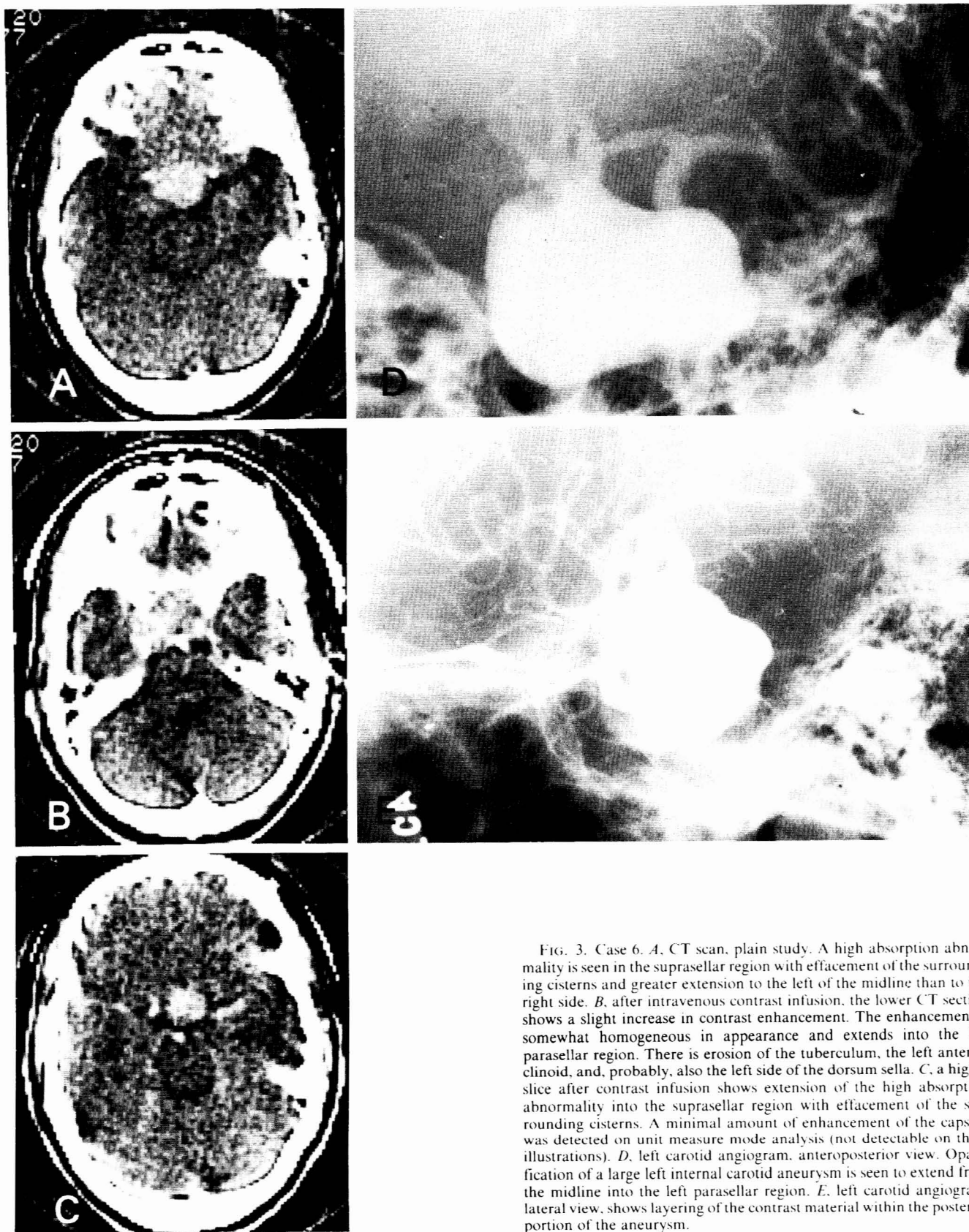


FIG. 3. Case 6. *A*, CT scan, plain study. A high absorption abnormality is seen in the suprasellar region with effacement of the surrounding cisterns and greater extension to the left of the midline than to the right side. *B*, after intravenous contrast infusion, the lower CT section shows a slight increase in contrast enhancement. The enhancement is somewhat homogeneous in appearance and extends into the left parasellar region. There is erosion of the tuberculum, the left anterior clinoid, and, probably, also the left side of the dorsum sella. *C*, a higher slice after contrast infusion shows extension of the high absorption abnormality into the suprasellar region with effacement of the surrounding cisterns. A minimal amount of enhancement of the capsule was detected on unit measure mode analysis (not detectable on these illustrations). *D*, left carotid angiogram, anteroposterior view. Opacification of a large left internal carotid aneurysm is seen to extend from the midline into the left parasellar region. *E*, left carotid angiogram, lateral view, shows layering of the contrast material within the posterior portion of the aneurysm.

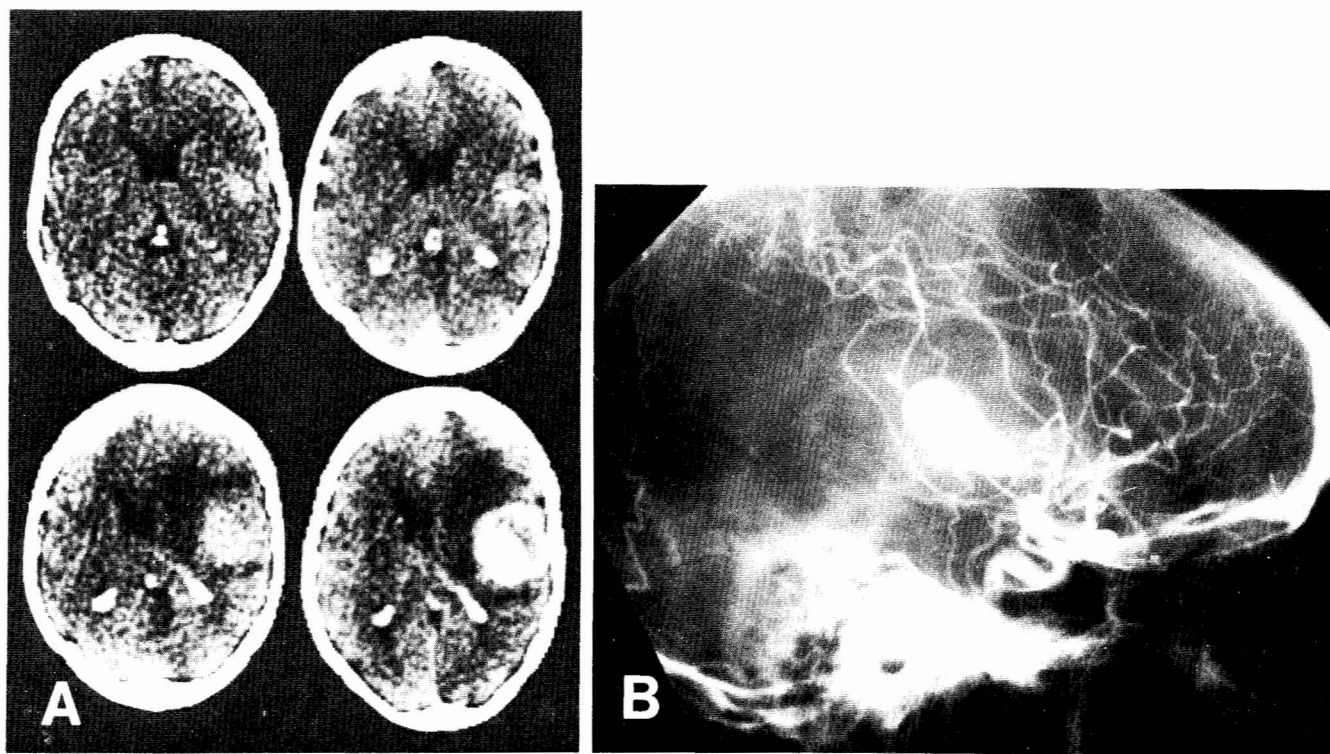


FIG. 4. Case 10. *A*, CT scans. *Top*: July 1976. The plain study (*left*) shows a small area of high absorption abnormality in proximity to the right sylvian fissure. The scan after intravenous contrast infusion (*right*) shows an area of homogeneous contrast enhancement adjacent to the slightly high absorption region. *Bottom*: April 1977. The plain study (*left*) shows a significant increase in the size of the previously seen high absorption abnormality located in the region of the right sylvian fissure and temporal lobe. An area of low absorption abnormality surrounds the region of high absorption and there is a midline shift from right to left. The scan made after intravenous contrast infusion (*right*) shows an area of homogeneous contrast enhancement within the lower medial half of the plain scan high absorption abnormality. This represents the nonthrombosed portion of the giant aneurysm. Enhancement of the capsule results in a signet ring appearance of the lesion. *B*, right carotid angiogram, lateral view. Opacification of the nonthrombosed portion of the aneurysmal sac is demonstrated, with a surrounding mass effect displacing the middle cerebral branches. The thrombosed portion of the aneurysm contributes to the over-all mass effect. The aneurysm takes origin from the middle cerebral artery in the vicinity of the sylvian fissure. Another incidental aneurysm is present on the more proximal portion of the middle cerebral artery.

discerning the exact size or even the nature of the space-occupying hemispheric lesion. Enhancement of the capsule of the aneurysm and the enhancement from the blood iodine pool within the nonthrombosed portion of the aneurysm usually help to differentiate aneurysm from tumor and abscess on the CT scan.

Although probably large from their beginning, giant aneurysms may grow as a result of repeated hemorrhages from tiny capsular vessels followed by propagation of thrombus and slow vascular recanalization (1, 9). Recanalized vessels probably coexist with the parent vessel until the main channel occludes as a result of the torsion and compression generated by the enlarging aneurysm. The following case history illustrates these points.

Case 10. A 62-year-old woman presented in July 1976 with transient episodes of "slurred speech" and "clumsiness" in the left hand and leg. Examination revealed a blood pressure of 270/130, dysarthria, gait dystaxia, and a clumsy left hand. Skull films were normal. A diagnosis of right pontine lacunar infarction was made, and the patient was discharged on antihypertensive medicines. Her signs resolved in 72 hours. She then had a second spell of incoherence with numbness in the left hand, and a CT scan was requested. The plain scan showed a small, low absorption abnormality near the midlateral aspect of the right sylvian fissure. The CE scan showed a homogeneous appearance of contrast enhancement with slightly irregular borders (Fig. 4A).

Anticonvulsants were administered. Over the ensuing 6 months the patient's family noted that her mentation was "slowing down," and examination confirmed a slight left hemiparesis, worse than at the time of her July hospital discharge. A repeat CT scan documented that the mass had approximately doubled in size. Homogeneous contrast enhancement with absorption values consistent with a blood iodine pool occurred, associated with a moderate region of thrombosis and slight capsular enhancement (Fig. 4A). Subsequent angiography showed a large middle cerebral artery aneurysm (Fig. 4B). An operation was recommended, and she underwent successful resection of the aneurysm with preservation of the distal flow to the right parietal cortex. Her hemiparesis and mentation have markedly improved since that time.

Giant aneurysms of the anterior cerebral artery

Giant aneurysms arising from the anterior communicating artery-anterior cerebral artery complex usually present with symptoms of progressive frontal lobe dysfunction punctuated by episodic frontal headache, seizures, and spells of lower limb weakness (1, 6). Apathy, antisocial personality changes, and urinary frequency with incontinence usually develop before the onset of gait and memory disturbances, a differential point separating the features of this pathological entity from normal pressure hydrocephalus (NPH). In NPH, changes in gait and memory usually antedate the onset of urinary incontinence, and headache is rarely a feature of this disorder. Our clinical

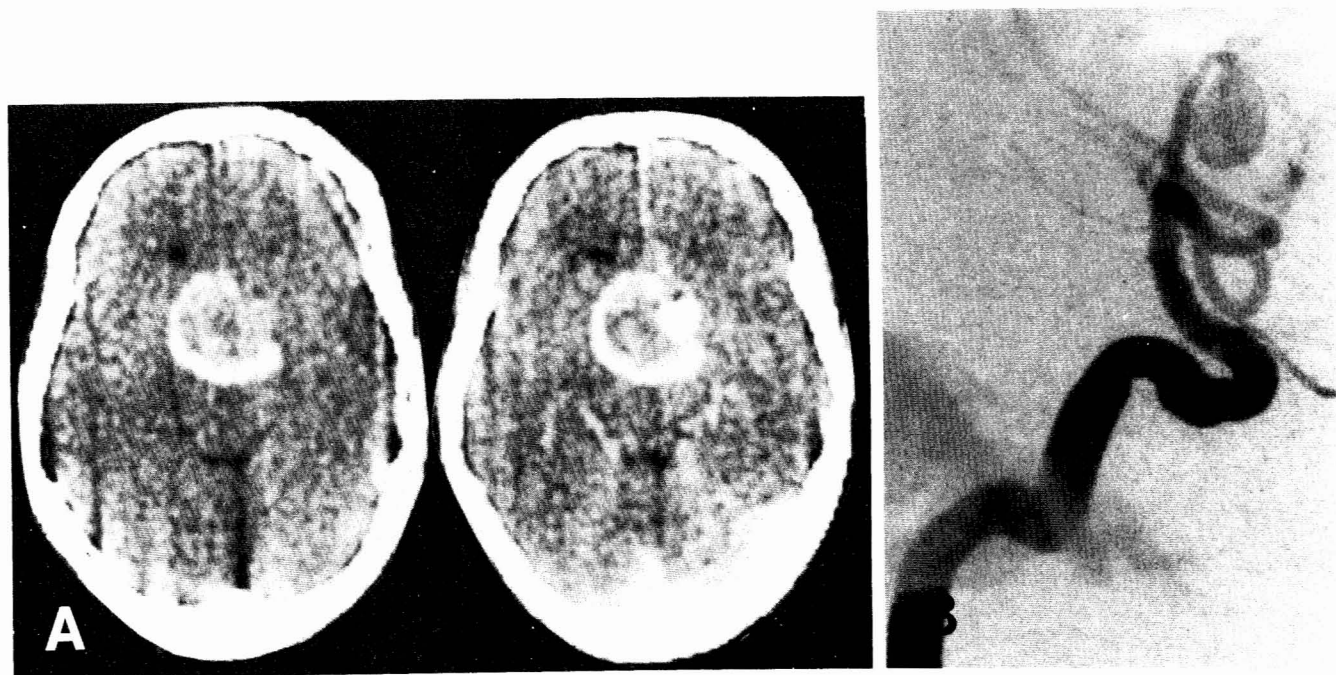


FIG. 5. Case 12. *A*, CT scan. The *left* image is without contrast material and shows a rim of high absorption values that represents calcification within the capsule of a giant aneurysm. The area within the rim of calcification also has slightly elevated absorption values. The *right* CT image was made after intravenous contrast infusion and shows moderate enhancement of the capsule of the aneurysm and a homogeneous area of enhancement on the right side of the capsule that represents the nonthrombosed lumen containing circulating iodine. *B*, subtracted lateral view of a right carotid angiogram. There is opacification of the nonthrombosed lumen of an aneurysm taking origin from the right anterior cerebral artery. The supraclinoid internal carotid artery, proximal middle cerebral artery, and horizontal portion of the anterior cerebral artery are stretched due to a mass effect from the total size of the aneurysm. The nonthrombosed sac of the aneurysm correlates well with the homogeneous area of enhancement on the CT scan.

experience has not included such a lesion. Rarely, a giant aneurysm of the anterior cerebral artery may present as a suprasellar mass lesion like a giant aneurysm of the supraclinoid carotid artery. The following case illustrates this point.

Case 12. This 54-year-old female bookkeeper first presented in the spring of 1973 with a complaint of failing vision in the left eye. The OD V/A with correction was 20/20, and she counted fingers at 1 foot OS. Visual field testing confirmed a large central scotoma with an inferior nasal quadrantic field defect OS. An OS afferent pupillary defect was suggested by a positive swinging flashlight test, and funduscopy confirmed the presence of advanced optic atrophy. Skull films, including special views of the superior orbital fissures and optic canals, were normal. A diagnosis of inflammatory optic neuritis was made, and high dose prednisone prescribed for 2 months resulted in subjective improvement in her vision.

She was lost to follow-up until May 1976 when her V/A OD was 20/400 and there was light perception only OS. A bitemporal hemianoptic defect was noted. The right pupil reacted briskly to light, and the left pupil was amaurotic. Skull films revealed erosion of the posterior clinoid process and dorsum sellae on the right. The superior orbital fissures and optic canals were normal. The CT scan demonstrated a smooth, partially calcified, encapsulated suprasellar lesion. The eccentrically placed "lumen" of the lesion, the significance of which was appreciated only after operative intervention, enhanced from 30 to 57 EMI units (peak values of the plain and CE scans), and the capsule enhanced from 38 to 45 EMI units (peak values, plain and CE scans) (Fig. 5*A*). A cystic craniopharyngioma rather than an aneurysm was suspected.

Aspiration of the thick-walled "cyst" at craniotomy resulted in samplings of arterial blood. Angiography under the same anesthetic confirmed the presence of a giant, partially throm-

bosed, saccular aneurysm with a narrow neck at the junction of the right anterior cerebral and anterior communicating arteries (Fig. 5*B*). Direct clipping of the aneurysm with resection of the aneurysm wall and retained thrombus was performed without untoward effects. Postoperative improvement in OD visual acuity was documented.

Fusiform aneurysms

Fusiform aneurysms (arteriosclerotic widened arterial channels) may be differentiated from giant globose aneurysms (congenitally large channels due to a defective internal elastic membrane) by the CT scan. The former tend to thrombose in the periphery, with a resultant patent central lumen, and have a tortuous course; the latter develop a central or eccentric thrombosis of the lumen with circumferential flow into and around the relatively round (nontortuous) aneurysm. In either case the CT scan shows the lesion to be considerably larger than the size suggested by the angiogram when there is partial or complete thrombosis (5, 9).

SUMMARY

In general, giant aneurysms are characterized by their smooth-walled, occasionally calcified capsule, the noncalcified portion of which enhances slightly from 35 to 48 EMI units (plain to CE scan peak values) after the intravenous infusion of contrast medium. Within the capsule may be a region that is eccentrically placed and that corresponds to the laminated clot if there is partial thrombosis. This area shows no contrast enhancement and may be isodense or slightly high in absorption values when compared to nearby brain. The complementary nonthrombosed region shows homogeneous contrast en-

hancement, from 40 to 60 EMI units, and corresponds to the patent lumen. The plain CT of this region may show slightly elevated absorption values due to the circulating blood volume. In addition, bony abnormalities, considered to be typical signs of giant aneurysms on plain films, may be present on the CT scan. These include asymmetrical sloping of the sella floor, erosion of the ipsilateral anterior clinoid, and destruction of the ipsilateral superior orbital fissure (orbital apex region).

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COMMENT

The authors have addressed still another diagnostic use of computed tomography. Giant intracranial aneurysms have often been difficult to assess with conventional neuroradiological procedures in terms of true size, relation to the parent artery, and amount and location of the intra-aneurysmal thrombus. CT scans have certainly proved helpful in defining some of these spatial relationships.

However, Polaroid pictures have limited value for the full evaluation of these lesions. It is well presented in the article that the analysis of peak absorption values is a critical point in evaluating these lesions.

Despite the relative ease of performing CT scans, their use does not obviate the need for angiographic evaluation of these lesions to determine the best therapeutic approach.

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