

observed hemosiderin staining of the subarachnoid space suggestive of subclinical hemorrhage. There are parallels to this phenomenon in the vascular and thoracic surgical literature. Aortic dissecting aneurysms have been documented as enlarging, despite lack of angiographic filling. Presumably, continued deposition of thrombotic material within the lumen of the aneurysm is responsible for its further enlargement. Indeed, flow into the aneurysm is minimal, below the resolution of conventional angiography. Given that the natural history of a totally thrombosed aneurysm is to shrink, resolve, or calcify, the authors rightly recommend vigilant follow-up for those lesions found to persist on subsequent

studies. Certainly, those patients who demonstrate symptomatic enlargement without angiographic filling should be considered for operative obliteration to obviate further enlargement or possibly distal embolization. Conceivably, radiolabeled blood products such as platelets, red cells, or fibrinogen may be helpful in documenting flow into an aneurysm. Turnover of any of these products documented by the appropriate scanning techniques might suggest that thrombosis is not complete.

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Chronic Encapsulated Intracerebral Hematoma Associated with Cavernous Angioma: A Case Report

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The case of a patient with a chronic encapsulated intracerebral hematoma associated with a cavernous angioma is reported. In spite of a huge space-occupying lesion in the frontal lobe, our patient showed no focal neurological deficit. The clinical picture suggested a slowly growing brain tumor. Intraoperative findings revealed a liquefied intracerebral hematoma with a thick capsule. Pathological investigations demonstrated a capsule rich in neovascularity with a cavernous angioma attached to it. It is presumed that initial bleeding from the cavernous angioma encouraged capsule formation similar to the membrane of chronic subdural hematomas and that repeated bleeding from the capillaries of the capsule allowed the expansion of the hematoma. (*Neurosurgery* 26:700-702, 1990)

Key words: Cavernous angioma, Chronic encapsulated hematoma, Intracerebral hematoma

INTRODUCTION

Intracerebral hematomas that expand gradually like a brain tumor and produce chronic or slowly progressive neurological deficits have been reported in the literature. Histologically, such hematomas are characterized by a thick fibrous capsule with rich neovascularity. We present a patient with a chronic encapsulated intracerebral hematoma associated with a cavernous angioma.

CASE REPORT

A 14-year-old boy exhibited increasing headache and vomiting over a 2-week period. He was referred to our hospital on July 6, 1985, because of a presumptive diagnosis of brain tumor made by a computed tomographic (CT) scan performed at a local hospital. He denied any history of hypertension, head injury, or coagulopathy.

Physical examination at admission disclosed nothing abnormal. Neurological examination revealed no abnormality, except for blurred margins on the temporal side of the optic discs bilaterally. All laboratory data including blood coagulation studies were within normal range.

Plain skull x-ray films also disclosed nothing abnormal. A huge isodense area associated with an eccentric high-density area adjacent to the inner table of the skull was seen in the

right frontal lobe on the plain CT scan. This was accompanied by a severe mass effect to the surrounding structures and perifocal low attenuation (Fig. 1A). A marginal zone of this isodense area was partially enhanced by the contrast material (Fig. 1B). An angiogram of the right common carotid artery demonstrated a large avascular lesion in the frontal lobe without any abnormal vessels.

A craniotomy was carried out on July 19, 1985. There was no adhesion between the dura and the cortex. The cortex revealed a brownish yellow discoloration. A small amount of dark brownish fluid similar to the content of chronic subdural hematomas was aspirated. The thick capsule just beneath the cortex was incised. The cavity was filled with fluid containing clots and was irrigated repeatedly with saline. The inside of the capsule was carefully explored. A small lobulated mass was found on the wall, which corresponded to the high-density area shown in the CT scan. There was no abnormal vessel such as a feeding artery or a draining vein. The capsule was removed totally, including the mass.

On histopathological examination, the capsule wall was found to be fibrous connective tissue with rich neovascularity. Deposits of hemosiderin were also seen in the capsule wall. These findings were similar to those of the outer membrane of chronic subdural hematomas (Fig. 2A). The specimen taken from the lobulated mass showed various sized vascular channels filled with numerous red blood cells. No intervening

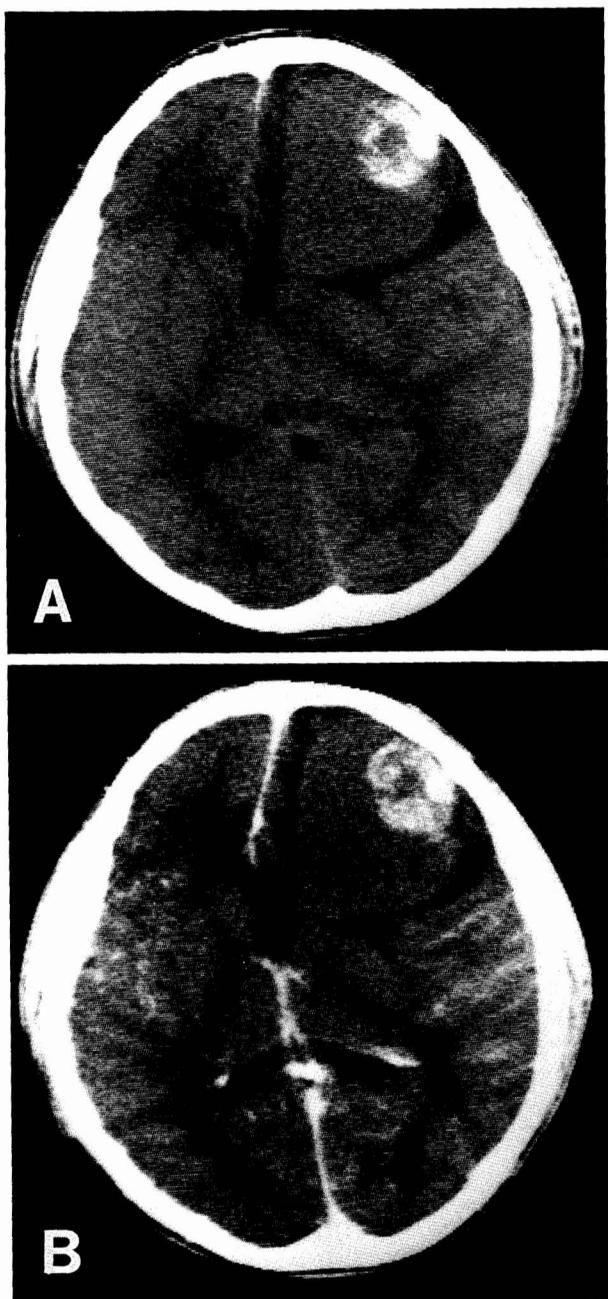


FIG. 1. *A*, plain CT scan revealing a huge isodense area containing a high-density mass 2 cm in diameter in the right frontal lobe. *B*, the margin of the isodense area is partially enhanced.

neural tissue was found. The histological features were consistent with a cavernous angioma (Fig. 2*B*).

His headache disappeared immediately after surgery, and the patient was discharged after an uneventful recovery. A postoperative CT scan revealed a huge low-density area in the right frontal lobe; however, there was neither a mass effect nor perifocal low attenuation.

DISCUSSION

The clinical course resulting from cavernous angioma is usually acute or subacute, and initial symptoms are commonly epileptic fits or intracerebral hemorrhage (11). Intra-

cerebral hemorrhage may present as a rapidly progressive, fulminating disease, sudden in onset, with the outcome being death or profound neurological deficit (6).

We have experienced an unusual case of intracerebral hematoma associated with cavernous angioma. Our patient showed only slowly progressive headache, and there was no acute episode suggesting intracerebral hemorrhage. In addition, he had no focal neurological deficit such as hemiparesis, despite the presence of a huge space-occupying lesion in the right frontal lobe. The clinical picture suggested neoplastic growth. Intraoperative findings, however, revealed a intracerebral hematoma with a liquefied brownish content similar to that of chronic subdural hematomas. How do we explain this unusual clinical course?

Although cavernous angioma is rarely associated with a large cyst, this association has been described as cystic cavernous angioma (3, 7, 10). These tend to be located in the brain parenchyma. All of the patients with a cystic cavernous angioma in the cerebral parenchyma reported previously showed slowly progressive neurological deterioration without acute episodes (7, 10). The cyst was filled with xanthochromic or turbid grayish fluid in each reported case. If a mild hemorrhage were to occur in the cavity of latent cystic cavernous angioma, a condition similar to that in the present patient might be expected.

Recently, patients with intracerebral hematomas who show stable or slowly progressive neurological deficits have been reported; these hematomas may be attributed not only to

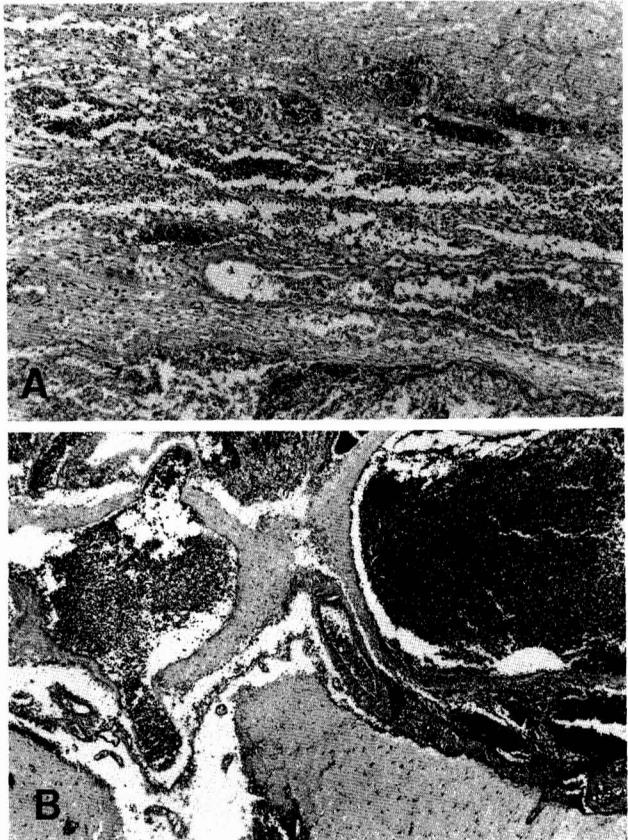


FIG. 2. *A*, photomicrograph of the capsule wall shows fibrous connective tissue with rich neovascularity (hematoxylin and eosin, $\times 33$). *B*, the specimen taken from the lobulated mass shows vascular channels of various sizes, suggesting cavernous angioma (hematoxylin and eosin, $\times 13$).

hypertension, but also to head injury, anticoagulation therapy, chemotherapy, or contraceptive medication (12). From the pathological point of view, a distinct capsule has not been described in these patients. Hirsh et al. (5) added a new type of intracerebral hematoma that had a thick, fibrous capsule. They called this unusual condition chronic encapsulated hematoma and suggested that a thick membrane arose from the fibroblasts related to the abnormal vessels demonstrated in the thick membrane itself. In the other four reported patients with thick membranes (1, 2, 8, 9), these abnormal vessels were not found in spite of a detailed histopathological investigation; however, neovascularization within the capsule wall was disclosed in each case. This is similar to the outer membrane of chronic subdural hematomas (2). Even in chronic subdural hematomas, the cause of chronically expansive growth is still unknown. Many investigators consider that recurrent hemorrhages from the outer membrane might play a major role in this growth (4). Therefore, it is possible to consider that the initial bleeding encourages capsule formation with rich neovascularity, and repeated bleeding or exudation from the capillaries of the capsule allows the expansion of the chronic intracerebral hematoma. The content of the hematoma may change over time. Takahashi et al. (8) reported a patient with a multilocular encapsulated intracerebral hematoma, which had various types of contents including solid hematoma, liquid hematoma, and xanthochromic fluid in the different parts of the capsule.

From the above discussion, the lesion in our patient is considered not to be a cystic cavernous angioma but a chronic encapsulated intracerebral hematoma because of it had a thick, fibrous membrane rich in neovascularity. It is concluded that in the chronic encapsulated intracerebral hematoma, a repeated hemorrhage from the thick membrane may play a role in its growth, and a vascular anomaly must be considered as a cause of initial hemorrhage.

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REFERENCES

1. Aoki N, Mizuguchi K: Chronic encapsulated intracerebellar hematoma in infancy: Case report. *Neurosurgery* 14:594-597, 1984.
2. Aoki N, Mizuguchi K: Expanding intracerebellar hematoma: A possible clinicopathological entity. *Neurosurgery* 18:94-96, 1986.
3. Bellotti C, Medina M, Oliveri G, Barrale S, Ettorre F: Cystic cavernous angiomas of the posterior fossa. Report of three cases. *J Neurosurg* 63:797-799, 1985.
4. Bergström M, Erickson K, Levander B, Svendsen P, Larsson S: Variation with time of the attenuation values of intracranial hematomas. *J Comput Assist Tomogr* 1:57-63, 1977.
5. Hirsh LF, Spector HB, Bogdanoff BM: Chronic encapsulated intracerebral hematoma. *Neurosurgery* 9:169-172, 1981.
6. Paillas JE, Alliez B: Surgical treatment of spontaneous intracer-

bral hemorrhage. Immediate and long-term results in 250 cases. *J Neurosurg* 39:145-151, 1973.

7. Ramina R, Ingunza W, Vonofakos D: Cystic cerebral cavernous angioma with dense calcification. Case report. *J Neurosurg* 52:259-262, 1980.
8. Takahashi N, Kikuchi H, Kobayashi K, Karasawa J: Multilocular encapsulated intracerebral hematoma [in Japanese]. *Neurol Surg* 11:739-743, 1983.
9. Terada T, Okuno T, Moriaki H, Nakai E, Komai N: Chronic encapsulated intracerebral hematoma during infancy: Case report. *Neurosurgery* 16:833-835, 1985.
10. Vaquero J, Cabezudo JM, Leunda G: Cystic cavernous haemangiomas of the brain. *Acta Neurochir (Wien)* 67:135-138, 1983.
11. Vogt K, Yaşargil MG: Cerebral cavernous haemangiomas or cavernomas. Incidence, pathology, localization, diagnosis, clinical features and treatment. Review of the literature and report of an unusual case. *Neurochirurgia* 19:59-68, 1976.
12. Yashon D, Kosnik EJ: Chronic intracerebral hematoma. *Neurosurgery* 2:103-106, 1978.

COMMENTS

Murakami et al. report an interesting case of a chronic encapsulated intraparenchymal hematoma associated with a cavernous angioma. Their discussion focuses on the pathophysiology of this lesion and draws a distinction between cystic cavernous angioma and chronic encapsulated intracerebral hematoma associated with cavernous angioma. They conclude that in their patient, the cavernous angioma probably bled initially, but that the progressive enlargement and repeated hemorrhage may have been related to the presence of the vascularized membrane. Although this interpretation may be correct, I suspect that either repeated bleeding episodes from the angioma or repeated hemorrhage into a cystic cavernous angioma could produce the same radiographic and histological situation.

This case highlights the difficulty of managing patients with non-life-threatening intracerebral hemorrhages without angiographically verified pathological findings. In this patient, the computed tomographic (CT) scan clearly demonstrated a pathological process, but this is often not the case. Magnetic resonance imaging (MRI) scans can be extremely helpful in demonstrating evidence of cavernous angioma, venous angioma, arteriovenous malformation, or neoplasms when acute CT scans are not helpful. A case can be made for surgically exploring all hematomas when there is evidence on the MRI scan of adjacent tissue not felt to be clot. If a nonsurgical course is followed, repeated angiograms and MRI scans should be considered after the hematoma has resolved to identify the occasional arteriovenous malformation rendered "occult" by a local mass effect.

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The authors have presented an extremely interesting case, which is unique, to my knowledge. Although the authors' case concerns the growth of an associated hematoma, it is well known that the cavernous angioma itself is capable of slow enlargement, perhaps by similar mechanisms. The clinical spectrum of cavernous angiomas continues to be elucidated.

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