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## CT Patterns in Histopathologically Complex Cavernous Hemangiomas

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Cavernous hemangiomas are relatively uncommon vascular malformations that may involve any part of the central nervous system. In some cases they are clinically quiescent and are detected at autopsy or, by chance, on a computed tomographic (CT) scan. Others cause seizures, headaches, intracranial hemorrhage, or focal neurologic deficits [1–4]. Most cases are diagnosed during the third to sixth decades of life. Identification of these malformations is clinically important because, when accessible, they can be extirpated surgically and are potentially curable [1, 2, 4]. Cavernous hemangiomas are more precisely defined by CT than by skull films, radionuclide scans, or cerebral angiograms [5-12]. CT studies of cavernous hemangiomas usually define a well demarcated, hyperdense lesion that frequently contains calcifications. There is no significant mass effect and usually only a mild degree of contrast enhancement. Hypodense areas may sometimes be observed within the lesion because of cystic components [12, 13]. Such a CT feature is nonspecific and may be seen in a variety of intracranial pathologic conditions including various forms of vascular malformations, granulomas, astrocytomas, oligodendrogliomas, craniopharyngiomas, and pineal region neoplasms [14-22]. We describe 10 histologically verified cavernous hemangiomas in which detailed CT studies were correlated with histologic findings.

#### **Subjects and Methods**

Five women and four men aged 22–54 years were studied with CT before and after intravenous injection of 300 ml of Reno-M-DIP (42 g l). Selective transfemoral cerebral angiography was performed in seven patients with magnification and subtraction technique. In five cases prolonged contrast injections (15 ml of Conray 60 delivered in 3 sec) were performed. In the other two cases, 10 ml of Conray 60 was injected at a rate of 7 ml/sec. In five patients, direct surgery was performed, while in the other four patients, CT-guided brain biopsies were performed. Tissue was fixed in 10% neutral buffered formalin. Paraffinembedded sections were stained with hematoxylin and eosin. Each specimen was carefully examined in the usual fashion by a neuropathologist who had no knowledge of the CT findings.

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TABLE 1	1:	Findings	in	Patients	with	Histolog	ically	Complex	Cavernous	Hemangiomas
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Case No.	Site	СТ	Histopathologic Diagnosis	Hemosiderin	Specimen
1	L basal ganglia	Moderate enhancement; calcification	Cavernous hemangioma and AVM	+	Biopsy
	R frontal L occipital*	Mild enhancement	Cavernous hemangioma	0	Biopsy
2	L parietal	Moderate enhancement; calcification	Cavernous hemangioma	0	Biopsy
3	Vermis	Mild enhancement	Cavernous hemangioma	0	Surgery
4	L centrum semiovale	Moderate enhancement; calcification	Cavernous hemangioma	+	Biopsy (mild hemorrhage)
5	. Medulla oblongata	Hematoma†	Cavernous hemangioma and AVM	+	Surgery
6	R thalamus	Moderate enhancement	Cavernous hemangioma	0	Biopsy (massive
7	L middle fossa	Marked enhancement	Cavernous hemangioma	0	Surgery
8	R frontal	Mild enhancement	Cavernous hemangioma and AVM	+	Surgery
9	. R frontal	Resolving hematoma with ring and tubular enhancement†	Cavernous hemangioma; venous malformation; telangiectasia	+	Surgery

Note.-L = left; R = right; AVM = arteriovenous malformation.

\* Not biopsied.

† Enhancement could not be appreciated within the lesion itself because of hemorrhage.



Fig. 1.—Postcontrast CT scan. Two cavernous hemangiomas. Lesion adjacent to left lateral ventricle is moderately enhanced and reveals areas of calcification (*curved arrow*). Other lesion is only minimally enhanced with no apparent calcification (*straight arrow*). Histologically both lesions contain calcification.



Fig. 2.—A, Precontrast CT scan. Area of slightly increased density (arrow) with average of 51 H. Fourth ventricle is slightly deformed. B, Postcontrast. Minimal enhancement (54 H) (arrow). C, Histology. Portion of cavernous hemangioma with thick bands of interstitial collagen. Only a few viable endothelial cells remain. Vascular channels are virtually free of blood cells. Dense intraluminar structures (calcifications) (arrows) (×640).

#### Results

Eleven lesions were identified by CT in nine patients (table 1). The most common presenting complaints were headaches (seven patients), seizures (four patients), and focal neurologic deficits (four patients). After brain biopsy, one patient developed a mild bleed; another had a massive and fatal hemorrhage.

Although 11 lesions were found in our nine patients, only 10 were confirmed histologically and all contained cavernous

hemangioma. Four of the 10 lesions consisted of two or more types of cerebrovascular malformation. Three manifested a complex vascular pattern that included arteriovenous malformation (AVM) and cavernous hemangioma elements; the fourth involved a distinctly unusual combination of cavernous hemangioma, telangiectasis, and venous malformation. One patient had three discrete lesions, but only two were biopsied and are considered in this report.

Histologically, eight of the cavernous hemangiomas contained calcifications that occurred particularly within throm-



Fig. 3.—Cavernous hemangioma simulating meningioma in 51-year-old woman. Pre- (A) and post- (B and C) contrast CT scans. Markedly enhancing extraaxial lesion arising in parasellar region with some bony erosion (arrow).

Faint capillary blush was seen on angiography. **D**, Histology. Multiple thinwalled large channels lined by flattened endothelium and, in some cases, bands of collagen (×280).



Fig. 4.—A and B, Postcontrast CT scans. Intracerebral hematoma in frontal lobe with enhancing rim in periphery. B shows a few tubular, enhancing structures in upper part of hematoma (*arrow*). Anteroposterior (C) and lateral (D) carotid angiograms. Vascular channels in late venous phase converge toward central vein (*arrows*). Except for mass effect, arterial and capillary

phases of cerebral angiogram were normal (not shown). E–G, Three different parts of lesion. E, Multiple thin-walled channels without intervening neural tissue, characteristic of cavernous hemangioma (×280). F, Thick-walled vessel of AVM (×65). G, Thin-walled channels of telangiectasis are separated by neural and glial tissue (×280).

bosed lumina, but amorphous or punctate calcifications within the lesion were only seen by CT in three of the 10 lesions (fig. 1).

Microscopically, all of the complex malformations were found to contain recent hemorrhage or deposits of hemosiderin. Minimal contrast enhancement (with an increase of less than 5 H) was noted in three lesions (fig. 2), and moderate enhancement (an increase of 10-15 H) in four lesions. In one other patient, the enhancement was marked and involved a homogeneously enhancing cavernous hemangioma in the middle cranial fossa that also produced bony erosion. The CT appearance of this lesion was indistinguishable from that of a meningioma (fig. 3). Two other patients exhibited CT evidence of recent hemorrhage within the lesion. In one of these lesions enhancement was not appreciated on CT studies because of hemorrhage into the angioma, and an acute hematoma within the medulla oblongata was later evacuated. Histologic examination of the surgical specimen revealed a vascular malformation with some elements typical of cavernous hemangioma. In the other patient, ring enhancement around the periphery of a frontal hematoma indicated that the hemorrhage was in a resolving stage. Microscopically, this surgical specimen revealed cavernous hemangioma, venous angioma, and telangiectasia, all coexisting within different parts of the hematoma (fig. 4). In two other patients, the surgical specimens contained a combination of cavernous hemangioma with other types of vascular malformation, which had thickened vessel walls containing elastica as well as intervening neural tissue. Foci of hemosiderin were observed in five lesions, four of which consisted of a combination of two or more types of vascular malformations.

Cerebral angiography showed a stain in two lesions and mass effect in three (two of these had intracerebral hematoma); one patient had radiating veins draining into a cerebral vein. In this case, these veins appeared in the late venous phase of angiography and were considered typical for venous angioma. No enlarged feeding vessels or rapid arteriovenous shunting could be observed in this group of patients.

### Discussion

There are several classifications for cerebrovascular malformations [23–26]. The most popular one is the classification proposed by Russell and Rubinstein [26], who separated cerebrovascular malformations into several groups based on their microscopic features: (1) capillary telangiectasis, (2) cavernous hemangioma, (3) AVM, and (4) venous malformation.

Histologically, cavernous hemangiomas are composed of clustered vascular channels of varying size having a wall lined only by endothelial cells, separated by penetrating tongues of connective tissue. No normal neural tissue intervenes. Some vascular channels have thicker walls containing various amounts of collagen, calcium, and hemosiderin. Cavernous hemangiomas are accompanied by a spectrum of histopathologic changes including hyaline degeneration, thrombosis, massive or multiple regions of minor focal hemorrhage, mineral deposits, and collagen. These histologic changes have a bearing on the CT appearance. Absence of intervening normal neural tissue distinguishes cavernous hemangioma from capillary telangiectasis. In some cases, however, differentiation of capillary telangiectasis and cavernous hemangiomas may be difficult histologically. Transitional forms containing elements of telangiectasis and cavernous hemangiomas or cavernous hemangiomas combined with other types of vascular malformation have been reported rarely [26–29]. Wortzman et al. [30] recently reported a case that could not be placed histologically within the Russell and Rubinstein classification. This lesion was called *dysplastic vascular malformation*.

Cavernous hemangiomas have also been reported in combination with glial neoplasms; it has been suggested that this is probably not a chance combination [31, 32]. Cavernous hemangiomas, despite their nonneoplastic and noncellular proliferative nature, may grow and damage adjacent neural tissue, resulting in gradual progression of clinical symptoms. Enlargement of the lesion is thought to be mainly from progressive dilatation of the thin-walled vascular component. This factor is to be considered in formulating a differential diagnosis when serial CT scans show an enlarging lesion.

The occurrence of multiple types of lesion in the same patient is of special interest. It is possible that examining multiple sites in surgical samples may reveal these combined lesions with a frequency greater than has been reported. Occasionally, however, a vascular malformation may be completely destroyed by hematoma, making its histologic identification as the cause of the hemorrhage difficult. Occasionally, a cavernous hemangioma, particularly in the parasellar region, may enhance markedly with an appearance similar to that seen in meningioma. Mori et al. [33] presented five cases of extradural cavernous hemangiomas in the middle cranial fossa. Two of the patients had CT scans that showed a highdensity mass with homogeneous marked enhancement. Ishikawa et al. [10] and Namaguchi et al. [34] each presented cases with similar CT findings. Cerebral angiography may not help to differentiate a middle fossa cavernous hemangioma from meningioma. Cavernous hemangiomas occurring in the middle fossa tend to occur in middle-aged women. It is very difficult to remove these tumors totally because they tend to be adherent to the cavernous sinus, and may also bleed profusely.

Recently, CT-guided stereotaxic brain biopsies have been used increasingly to establish the histologic diagnosis of many intracranial lesions. The intent is to provide proper management, including the avoidance of unnecessary radiation therapy in certain patients [35]. However, two of our four patients undergoing brain biopsy developed intracerebral hemorrhage, mild in one case but massive in the other. It is significant that a review of the CT scans in two of these patients revealed that the lesions had moderate contrast enhancement. On the other hand, both lesions in figure 1 were biopsied without complications.

Although some CT patterns found with cavernous hemangiomas are indistinguishable in appearance from those seen with other intracranial lesions, a correct preoperative diagnosis of cavernous hemangioma can often be suggested. However, our observation of other distinctive types of vascular malformation in association with cavernous hemangioma in four patients adds a further consideration in CT-pathologic correlation. Such complex malformations may be more common than has been reported heretofore.

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