Occult Cerebral Vascular Malformation: High-field (2.0T) MR Imaging and Comparison with CT

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〈국문초록〉

잠행성 뇌혈관기형: 고자장(2.0T) 자기공명영상 및 전산화단층촬영술과의 비교

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저자들은 잠행성 뇌혈관기형으로 진단받은 환자중 전산화단층촬영술(CT), 혈관조영술, 및 고자장(2.0T) 자기공명영상(MR) 검사를 모두 시행한 14명의 환자에서 발견된 20례의 병변을 역행적으로 분석하였다. 3명의 환자에서는 수술로 확진되었으며 나머지는 전형적 임상소견 및 특정적 방사선학적 소견으로 진단하였다.

MR로는 모든 병변을 국한성 출혈성 병소로 찾을 수 있었다. 잠행성 뇌혈관기형의 특정적 MR 소견은 T2 강조 영상에서 병변의 주변부를 따라 hemosiderin침착을 나타내는 푸에로 저강도환(hypointense rim)이 옆해 중심부에는 여러단계의 혈종이 있음을 시사하는 다양한 신호강도들을 보이는 것으로 큰 혈종이 반복되지 않은 경우에는 부분이나 종괴효과(mass effect)가 경미하였다. 이 중 2례에서는 소량의 반복 출혈로 형성되는 특이한 '포도송이' 모양을 관찰할 수 있었다. 그러나, 병변이 1 cm 이하로 작은 경우에는 원형 또는 탐양의 근절한 저강도 결절로 보였고(8례), 비전형적 소견을 보이는 경우도 2례에서 있었다. 잠행성 뇌혈관기형의 가능성을 강력히 시사하는 부수적 MR 소견으로, 비정상적혈관에 의한 신호소실(signal void)이 4례에서, 주위 뇌실질의 위축이나 백질연화(leukomalacia)가 2례에서 각각 관찰되었다. 병변의 호방부위는 대뇌피질하부나(8례) 뇌실주위(6례)였다.

CT로는 20례중 4례의 작은 뇌혈관기형을 찾지 못하였으며, 대부분의 경우 CT반으로는 뇌혈관기형의 진단을 내리는 것은 어려웠다. CT에서 발견된 16례 중, 그 정도나 모양의 차이는 있으나, 15례에서 고 밀도 음영(hyperdense)을 보이는 부위를 관찰할 수 있었다. 그 중 주변부의 고 밀도 음영은 MR에서 hemosiderin침착부위와 일치하였고, 중심부위에 국한된 고 밀도 음영은 급성혈종의 MR소견을 보였으며, CT상 저밀도 음영인 부위는 MR에서 T1 및 T2 강조영상에서 모두 고강도음영을 보여 역학된 야궁성 출혈병소임을 알 수 있었다.
Summary

We retrospectively analyzed 20 lesions of occult cerebral vascular malformation (OCVM) examined by CT, angiography and high-field (2.0T) MR in 14 patients. All lesions were angiographically occult. Diagnosis was based on the typical clinical history and characteristic radiologic findings in all patients and confirmed by surgery in 3 patients.

MR demonstrated all lesions as hemorrhagic foci. The most common and typical MR finding of OCVM was circumscribed thick hypointense rim on T2-weighted images (T2-WIs) representing hemosiderin deposit with various central intensities suggesting the presence of hematomas in different stages (11 lesions). Among these, the recurrent hemorrhage in small amount produced the characteristic "a bunch of grape" appearance. But, small OCVM less than 1 cm in diameter was identified as a small homogeneous hypointense nodule on T2-WIs with sharp (6 lesions) or fade-out (1 lesion) border. There were 2 lesions showing atypical findings such as dense hemosiderin rim with central lacuna or bilobed subacute hematoma. As the associated MR findings which strongly suggest the diagnosis of OCVM signal void due to feeding or draining vessels was found in 4, lesions and the surrounding focal cortical atrophy or leukomalacia in 2 lesions. The predilection site of OCVM was subcortical (8 lesions) and periventricular (6 lesions) location.

CT depicted 16 of the 20 lesions, but missed 4 lesions of small OCVM. CT alone did not permit definitive diagnosis of OCVM in most cases. Fifteen lesions demonstrated on CT contained somewhat hyperdense area in central or peripheral location. Peripheral hyperdense area corresponded to the dense hemosiderin deposit on MR and central one to the hematoma formed by recent hemorrhage. Low density area on CT usually corresponded to liquefied subacute hematoma on MR.

Introduction

Extensive experience with CT has led to the recognition of criteria for the diagnosis of occult cerebral vascular malformation (OCVM). The presence of hyperdense calcified lesion with little mass effect or enhancement suggests the diagnosis of OCVM. However, preoperative discrimination from tumors or granulomas remains problematic.

Recently, the characteristic sequential MR appearance of intracranial hemorrhage (ICH) at high-field (1.5 T) MR has been established, which enabled specific detection of hemorrhage and relatively accurate estimation of its age. Thereafter, Gomori et al reported the characteristic appearance of OCVM on high-field (1.5 T) MR images and its superiority to low-field MR or CT for OCVM.

We report 14 cases of OCVM examined by a combination of CT, angiography, and high-field (2.0 T) MR studies. Retrospective analysis of MR appearance of OCVM and comparative evaluation of OCVM subcomponents between MR and CT images were performed.

Materials and Methods

Fourteen patients of OCVM, 8 men and 6 omen, ranging in age from 9 to 52 years, were examined by MR in Seoul National University Hospital for the last one year. They presented with various neurologic symptoms (seizure for many years in four patients, episodic focal neurologic deficit with or without vomiting in four, wax-and-wane neurologic deficit in two, long-term focal neurologic deficit with slow progression in two, gradual focal neurologic deficit in one and chronic headache only in one). All five patients of supratentorial
OCVM presented with seizure or chronic headache, and those of infratentorial lesion with focal neurologic deficit.

Diagnosis of OCVM was established on the basis of characteristic clinical findings and typical radiographic manifestations in all patients and confirmed by surgical biopsy in three patients.

MR imaging was done on a 2.0T superconducting unit (Spectro-20000, Seoul, Korea) using 2-D FT spin-echo (SE) pulse sequences. T1-weighted images (T1-WIs) were obtained with repetition times (TR) of 500-600 msec and echo times (TE) of 30 msec. Balanced and T2-weighted images (T2-WIs) were obtained with TR of 2800-3000 msec and TE of 30 msec and 80 msec, respectively. Acquisition matrix was 256 X 256, and field of view (FOV) was 25 cm. The slice thickness and gap were 5-8 mm and 2 mm, respectively. Number of excitation (average) was 2-4 in T1-WIs and one in T2-WIs. Routine imaging plane was axial, but coronal and/or sagittal images were additionally obtained in some selected patients. Pre- and post-enhanced CT was performed with third generation scanners within 2 weeks before MR imaging in 12 patients. In remaining 2 patients CT was performed 8 months and 1 month before MR imaging in respect.

The CT and MR images were compared with emphasis on not only the detectability and characterization of the lesion but also correlation between MR and CT appearance of its subcomponents.

**Result**

Fourteen patients had 20 OCVM's, one patient having 7 lesions. Ten lesions were supratentorial and the other ten were infratentorial in location. The supratentorial lesions tended to be subcortical (8/10 lesions) or periventricular (2/10 lesions) distribution and the infratentorial lesions were observed at cerebellum (4 lesions) and brain stem from midbrain to medulla oblongata (6 lesions). Six lesions of infratentorial OCVM also occurred around the fourth ventricle. The size of OCVM was less than 3 cm in diameter in 18 lesions, but in remaining 2 lesions, they were as large as 4 cm.

Ten lesions could be seen equally well on CT and MR images, 6 were more conspicuous on MR, and 4 were detected only on MR.

MR detected all lesions and demonstrated its hemorrhagic nature. MR findings of OCVM were summarized in Table 1. Circumscribed thick hypointense rim on T2-WIs representing dense hemosiderin deposit with various internal signals on both T1- and T2-WIs suggesting central hematoma of different stages (9 lesions) was the most common and typical feature (Fig. 1, 2). Small OCVM without recent hemorrhage (7 lesions) was demonstrated as homogeneously hypointense nodule on T2-WIs with sharp or fade-out margin (Fig. 3). In two lesions, large eccentric hematoma displaced the nidus of OCVM to the periphery, which was markedly hypointense on T2-WIs with various internal signal (Fig. 4). Atypical findings such as thick hemosiderin rim with central lacuna and bilobed subacute hematoma were found in one lesion in each (Fig. 5). Recurrent hemorrhage in small amount made the characteristic “a bunch of grape” appearance in two lesions (Fig. 1, 2) and signal void due to feeding or draining vessels were identified in 4 lesions (Fig. 4, 5). Layering phenomenon of blood sediment was observed in 2 lesions (Fig. 1, 5). In 18 lesions, the surrounding edema was absent or minimal. But, considerable edema was associated in 2 lesions with large hematoma due to recent hemorrhage (Fig. 4). Mass effect was also absent or minimal in most of the lesions, but, was observed in strategic location (around fourth ventricle) (Fig. 6) or around the large hematoma (Fig. 4). On the contrary, focal cortical atrophy or leukomalacia was associated at adjacent brain parenchyme in 2 lesions. Multiple UBO’s (unknown bright objects) at periventricular white
Table 1. Summary of 20 Occult Cerebral Vascular Malformations in 14 Patients

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex</th>
<th>Symptoms Duration of Symptoms</th>
<th>Location</th>
<th>CT-MR Interval</th>
<th>Size</th>
<th>CT Findings</th>
<th>MR Finding</th>
<th>Others</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>40/M</td>
<td>Seizure, 2 years</td>
<td>Frontoparietal subcortical</td>
<td>14 days</td>
<td>4 cm</td>
<td>• Mixed density with large amorphous hypodense area</td>
<td>• Peripheral nodular enhancement</td>
<td>'Bunch of grape' appearance</td>
</tr>
<tr>
<td>2</td>
<td>44/M</td>
<td>Seizure, 3 years</td>
<td>Parietal subcortical</td>
<td>8 mos.</td>
<td>2 cm</td>
<td>• Hyperdense rim in periphery</td>
<td>• No enhancement</td>
<td>Dense hemosiderin rim with central lacuna</td>
</tr>
<tr>
<td>3</td>
<td>34/M</td>
<td>Seizure, 3 years</td>
<td>Parietal subcortical</td>
<td>1 mo.</td>
<td>1.5 cm</td>
<td>• Slightly hyperdense nodule</td>
<td>• No enhancement</td>
<td>Dense hemosiderin deposit with central multistage hematoma</td>
</tr>
<tr>
<td>4</td>
<td>24/M</td>
<td>Seizure, 5 years</td>
<td>Parietal subcortical</td>
<td>13 days</td>
<td>&lt;1 cm</td>
<td>• Slightly hyperdense nodule</td>
<td>• No enhancement</td>
<td>Rounded hemosiderin deposit</td>
</tr>
<tr>
<td>5</td>
<td>41/F</td>
<td>Headache, 3 years</td>
<td>Temporoparietal subcortical</td>
<td>1 day</td>
<td>4 cm</td>
<td>• 2 components (hyperdense and hypodense area)</td>
<td>• No enhancement</td>
<td>No enhancement</td>
</tr>
</tbody>
</table>

*Lesion 1:

5* Patient 5: Headache, 3 years

5. 42/F Facial neuralgia, 7 years

6. 32/F Hemiparesis, 6 months

7. 17/F Seizure, 5 years

8. 39/F Seizure, 2 years

9. 9/F Seizure, 2 months

10. 9/F Seizure, 2 months

11. 29/F Seizure, 20 days

12. 48/M Seizure, 4 years

13. 24/M Seizure, 1 month

14. 34/M Seizure, 1 year

MCP: Middle cerebellar peduncle

(-): No abnormality

* Case 5, 6, 8 were surgically proven.

mo: month

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matter which was usually associated in hypertensive ICH was not found at all.

CT study detected 16 of the 20 lesions. Four lesions (3 small cerebral hemispheric lesions in a patient with multiple OCVM's and a small brain stem OCVM) were not identified on CT. The CT density of the lesions was various from low to high density, but all lesions except one contained some-
what hyperdense area which was variable in density and distribution within the lesion. Patterns of these CT hyperdensity could be roughly categorized into followings: small slightly hyperdense nodule (5 lesions), large amorphous irregular high density mixed with low density (3 lesions), homogeneously hyperdense mass (2 lesions), central high density (2 lesions), hyperdense rim in periphery (2 lesions), central high density with slightly hyperdense rim (1 lesions), slightly hyperdense mass with eccentric focus of high density (1 lesions). Definite calcification was suspected in 5 lesions. One patient manifested as bilobed low density mass at midbrain which was confirmed by MR as subacute hematoma (Fig. 5). Layering phenomenon was observed at one lesion. Although OCVM itself was minimally enhanced after contrast injection, dilated enhancing vessels suggesting vascular

Fig. 1. (case 9) (a). Axial T1-WI (TR 500 msec, TE 30 msec). There is multilocular hyperintense lesion simulating a "a bunch of grape" in left midbrain with layering phenomenon at the medial locule. The supernatant is hyperintense and the dependent layer is slightly hypointense. (b). Axial T2-WI (TR 3000 msec, TE 80 msec). There is peripheral hypointense rim of variable thickness and the central portion of each locule shows different signal intensity indicating different stages of hemorrhage. The supernatant of the medial locule remains hyperintense (free methemoglobin) and the dependent layer shows marked hypointensity suggesting the presence of deoxyhemoglobin in intact red blood cells. (C). Follow-up after 3 months, T2-WI (TR 3000 msec, TE 80 msec). Overall size of the lesion slightly decreased. The hypointense rim is thickened, and the layering phenomenon disappeared. But, considerable amount of central hematoma is still remained.
Fig. 2. (case 1) (a). Unenhanced axial CT-2 years before. There is amorphous irregular hyperdense area suggesting calcification at right frontoparietal white matter. Prominent sulci of both frontal lobes indicate focal cortical atrophy. (b). Recent unenhanced axial CT. Hyperdense mass definitely has grown, but, is not accompanied by edema or mass effect. (c). Sagittal T1-WI (TR 500 msec, TE 30 msec) shows multilocular hyperintense lesion with marked peripheral hypointensity. (d). Axial T2-WI (TR 3000 msec, TE 80 msec). Peripheral hypointensity is more prominent and extensive and the central signal intensity is complex. It is impossible to discriminate calcification from hemosiderin because dense hemosiderin deposit produces profound hypointensity on T2-WI. There is no surrounding edema.
nature were identified in 4 lesions as in MR. The eccentric hematoma in subsacute stage showed rim enhancement. Two supratentorial lesions closely mimicked calcified glioma (Fig. 2).

Correlation between CT and MR appearance of OCVM subcomponents was tried. Definite calcification seen in 5 cases on CT was not delineated on MR due to profound hypointensity caused by hemosiderin deposition (Fig. 2, 4). Peripheral hyperdense rim, eccentric hyperdense focus, and small slightly hyperdense nodule on plain CT showed marked hypointensities at the exactly same site on T2-weighted MR images, reflecting hemosiderin deposition secondary to old hemorrhage (Fig. 3, 4, 6). Central high density on plain CT was slightly hypointense on T1-WIs and markedly hypointense on T2-WIs indicating acute hematoma in intracellular deoxyhemoglobin state (case 6). Central high density in case 11 was mostly hyperintense on both T1- and T2-WIs indicating subacute hematoma in extracellular methemoglobin state, because MR images were obtained 12 days later (Fig. 6). Low density area on plain CT revealed hyperintense signal on both T1- and T2-WIs representing liquefied subacute hematoma (Fig. 5).

Fig. 3. (case 4) (a). Unenhanced axial CT. Slightly hyperdense nodular lesion at left parietal subcortical area (arrow) is accompanied by low density at adjacent white matter. This lesion showed no enhancement. (b). Axial T1-WI (TR 600 msec, TE 30 msec). The lesion is slightly hypointense (arrow) and surrounding white matter is more hypointense. (c). Axial T2-WI (TR 3000 msec, TE 80 msec) demonstrates markedly hypointense nodule suggesting dense hemosiderin deposit, surrounded by hyperintense zone indicating leukomalacia.
Discussion

Vascular malformations that cannot be seen angiographically have been termed "occult". Although the true incidence is unknown, vascular malformations are found in approximately 0.14% of the general population^{12) and the ratio of OCV-M to the classical malformation was 48:5, as detected in 48 autopsied cases by McCormick and...

![Image](attachment:image.png)

**Fig. 4.** (case ) (a). Unenhanced axial CT shows slightly hyperdense round mass at right cerebellar hemisphere. There is more hyperdense nodular lesion at the lateral periphery and considerable amount of edema and mass effect is associated. (b). Enhanced axial CT clearly demonstrates linear vascular enhancement. The mass itself is not enhanced. (c). Axial T1-WI (TR 600 msec, TE 30 msec). The lesion is homogeneously hyperintense and the 4th ventricle is compressed and displaced. (d). Axial T2-WI (TR 3000 msec, TE 80 msec). The lesion is single large hematoma showing donut-shaped peripheral hyperintensity (free methemoglobin) and marked central hypointensity (intracellular methemoglobin). Peripheral focal hyperintense area on unenhanced CT is demonstrated as marked hypointense area with central irregular hyperintensity suggesting nidus of OCV-M (arrow). Signal void in branching pattern indicating feeding or draining vessels is identified. Peripheral hypointense rim (hemosiderin deposit) is thin and even in thickness and surrounding edema is hyperintense.
Nofzinger\textsuperscript{13}). They may be discovered incidentally or the patient may experience a variety of symptoms; most commonly seizure, headache, or neurologic deficits of various types and degrees.

OCVM includes lesions from all four major varieties of vascular malformation (capillary telangiectasia, venous angioma, cavernous angioma, and arteriovenous malformation)\textsuperscript{5,8}). The abnormal vessels of venous angiomas and AVM are usually evident on angiograms\textsuperscript{14}), but, some may be thrombosed and angiographically occult\textsuperscript{4,15}). Capillary telangiectasia generally occurs in the pons and is seldom identifiable angiographically. Although it is incidentally found at autopsy, it can be a source of pontine hemorrhage\textsuperscript{6,16}). Cavernous angiomas are most often located supratentorially and are usually subcortical. They are multiple in 16–33\% of the patients\textsuperscript{17}). On pathological examination, they are well circumscribed, and pale areas of fibrosis and calcification may be seen in the substance of the angioma. The neighboring brain shows the yellow-brown pigmentation of hemosiderin deposits\textsuperscript{17}). There are also hemosiderin deposits within the angiomas\textsuperscript{4,17}). Above 4 types of vascular malformations can coexist in a single lesion\textsuperscript{9}).
On plain CT, OCVM tend to present as well-circumscribed hyperdense, or occasionally isodense, relatively small mass lesions, generally no more than 3 cm in diameter, often containing recognizable punctate or small dense foci indicative of calcification. After intravenous contrast enhancement, relatively mild or moderate rather than marked enhancement is commonly seen. Occasionally, small regions of intense enhancement suggest the presence of feeding or draining vessels. One-third to one-half of the lesions will demonstrate a mass effect. Edema is generally absent.\(^{18}\)

The characteristic locations of the lesions include brain stem, periventricular white matter, and gray-white matter junction.\(^{11}\) In a small number of cases, repeated hemorrhages, which may be subclinical, cause these lesions to grow (Fig. 2). CT findings of our cases also coincided with above description.

Although CT has in fact accounted for the detection of a large number of these lesions than previously possible, the above characteristics are relatively nonspecific. The same findings can be attributed to a low-grade glioma such as astrocytoma

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Fig. 6. (case 11) (a). Unenhanced axial CT shows a round mass of more hyperdense center within less hyperdense peripheral rim. Enhanced CT showed no significant enhancement. (b). Axial T1-WI (TR 600 msec, TE 30 msec) obtained 12 days later. The lesion center is mostly hyperintense with isointense area and the periphery is hypointense. (c). The lesion center is persistently hyperintense, that is, hematoma in free methemoglobin state. The peripheral hypointensity is more marked and well correlated with peripheral hyperdense rim on CT.
and oligodendroglioma, and a granuloma. In our series, case 1 and case 5 (lesion 1) closely mimicked oligodendroglioma (Fig. 2).

The advent of MR provided promising capabilities in depicting vascular structures with greater anatomical detail and in identifying hemorrhagic lesions in different stages. MR imaging at 0.35 T and 0.6 T has been reported to be less sensitive than CT in detecting OCVM, even if it provides significant improvement in the accuracy of diagnosing OCVM. Recently, Gomori et al suggested that OCVM have characteristic appearance on high-field (1.5 T) MR images and high-field MR is much more sensitive and specific than CT or low-field MR (0.12 and 0.35 T) in evaluation of OCVM. This is due to the heightened sensitivity of high-field MR imaging to the iron in hemosiderin deposits and acute hematomas (proportional to the square of the magnetic field strength), and to its greater spatial and contrast resolution. In our series, 2.0 T MR detected all lesions including 4 lesions which was missed by CT and clearly demonstrated their hemorrhagic nature. On the other hand, CT is more sensitive than MR to calcium. Consequently, the OCVM that contains only calcification without previous hemorrhage can be detected solely by CT. But, this clinical situation is very rare and we did not meet any one case like this.

The classical high-field MR appearance of OCVM is circumscribed regions of low intensity on T1-WIs that become more hypointense and more extensive on T2-WIs with multiple areas of various signal intensity patterns within it. This feature was also one of the most common findings of OCVM in our series (Fig. 1, 2). But small OCVM without recent hemorrhage was seen as small homogeneously hypointense nodule less than 1 cm on T2-WIs with sharp or fade-out border and was as frequent as the former. Large eccentric hematoma distorted the classic pattern of OCVM (Fig. 4). Atypical findings such as thick hemosiderin rim with central lacuna, which is the finding of old hypertensive ICH, and bilobed subacute hematoma was also observed (Fig. 5). In these “non-classical” OCVM, the associated findings and location of the lesion were very helpful in differential diagnosis. That is, the signal void due to feeding or draining vessels, predilection site such as subcortical or periventricular location, and surrounding focal brain atrophy or leukomalacia strongly suggested the diagnosis of OCVM (Fig. 3, 4, 5). Additionally, we presented the characteristic “a bunch of grape” appearance of OCVM in 2 lesions, which was produced by recurrent hemorrhage in small amount (Fig. 1, 2).

In comparative study between the CT and MR appearance of OCVM subcomponents, Gomori et al reported that hyperdensity of OCVM on CT did not correspond to the more peripheral hypointensity seen on T2-weighted MR images. In our series, however, peripheral hyperdensity on plain CT corresponded exactly to the marked hypointensity on T2-weighted MR in location (Fig. 4, 6). This represent that dense hemosiderin deposit due to repeated hemorrhage contribute considerably to the peripheral high density of OCVM on plain CT. On the other hand, central higher density correlated with acute hematoma on MR (Fig. 6). Low density area within the lesions, central or eccentric, was usually manifested on MR as hyperintense area on both T1- and T2-WIs, indicating subacute hemorrhage in extracellular methemoglobin state (Fig. 5).

There are two entities that may be confused with dense hemosiderin deposits: dense calcifications and flow void (lack of signal) in vessels. All three may be hypointense on both T1-and T2-WIs. These entities can be differentiated by high-resolution, high-field spin echo MR or gradient echo technique. Calcium deposits tend to form in the center of lesions. They have a constant mild hypointensity and fixed extent on both T1-and T2-WIs and may have irregular margins. However,
hemosiderin deposits, the residue of the parenchymal reaction to hematoma, are both more hypointense and of greater extent on T2-WIs than on T1-WIs. They are on the periphery of lesions and have smoother margins. In addition, high-field MR is much more sensitive to hemosiderin than CT\textsuperscript{10,11}. Flow voids follow the branching pattern of vessels, unlike the shell-like deposits of hemosiderin in the parenchyma surrounding hematomas (Fig. 4). Flow in vessels may show paradoxical enhancement on T1-WIs or even-echo rephasing on T2-WIs\textsuperscript{14,22}. On the gradient echo technique the hemosiderin deposits appear to be more hypointense and greater in extent due to greater susceptibility, while flow in vessels shows hyperintensity\textsuperscript{23}. But, in our series, we did not obtain any image with gradient echo technique. These differences permit the differentiation of hemosiderin from calcium and flow void.

There are many conditions which must be differentiated from OCVM. Simple hematomas associated with trauma, surgery, or hypertension have a single cavity. Hemorrhagic contusions and infarctions may have multiple collections but are of the same age on high-field MR images and have other associated parenchymal findings. Cortical hemorrhagic infarcts affect the cortex, unlike the subcortical or periventricular location of OCVM. Old hematomas eventually collapse to form hemosiderin-lined clefts or nodular hemosiderin deposit. In contrast, the classical MR appearance of OCVM suggests the long-standing hemorrhagic lesion with recurrent bleeds. Actually, follow-up studies showed nonresorbable nature of OCVM with slow increase or decrease in size (Fig. 1, 2), unlike with the resorbable hematoma caused by a single large bleed. In addition, typical clinical course and CT findings reinforce the possibility of OCVM.

Hemorrhagic neoplasms can show striking similarity on MR with OCVM\textsuperscript{23}. Clinical course of the patient and known malignancy or metastasis frequently suggests the diagnosis. Radiologically, multiplicity and the presence of adjacent edema, especially if extensive, favors a neoplastic origin. In questionable cases, CT may provide further information because OCVM tend to be isodense or hyperdense on plain CT and occasionally shows calcification.

REFERENCES