

East Asia Friendship Workshop

Histopathological study of five cases with sporadic meningioangiomas

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We report five cases of sporadic meningioangiomas, three males and two females, ranging in age from 12 to 36 years at diagnosis. The lesion was found incidentally by MRI after a head trauma in one case; the other four subjects had a seizure disorders, which improved following surgical resection of the cortical lesions. Grossly, the lesionectomy specimens were of a whitish color and firm consistency. Histological examination revealed that the lesions were confined to the cortex with focal involvement of the overlying leptomeninges, and revealed unifying features of meningioangiomas, such as proliferating microvessels with perivascular cuffs of spindle-cell proliferation within the cortex. Two cases had numerous calcifications; one was associated with a prominent fibro-calcifying component. Immunostaining results were variable among the cases. Only vimentin was consistently positive. Some of the spindle cells were weak positive for EMA in two cases. Immunoreactions with anti-CD34 detected within the cytoplasm of the spindle cells were observed in three of the five cases. The Ki-67 proliferation index of all the cases was very low, less than 0.1%. Neurofibrillary tangles were identified in only one of the five cases using the Bodian and immunostaining methods. These findings indicate that meningioangiomas lesions show a wide range of clinicopathological features, making diagnosis difficult. A histopathological spectrum and differential diagnoses were discussed with a review of the literature. Since this lesion is a distinct clinicopathological entity and hamartomatous in nature, it is important to make a correct diagnosis in order to avoid further aggressive treatment.

Key words: hamartomatous lesion, histopathology, meningioangiomas, neurofibrillary tangles, seizures.

INTRODUCTION

Meningioangiomas (MA) is a rare, benign, meningiovascular hamartomatous condition affecting the cerebral cortex and leptomeninges. Approximately 90 cases of MA have been reported since the first description in 1915.¹ The majority of the cases are sporadic but the association of this lesion with familial neurofibromatosis type 2 is well known.^{2–8} MA usually appears in young patients. Focal seizures and/or headaches are the most common presenting symptoms. A few cases with neurofibromatosis may be asymptomatic, and have been found incidentally after a head trauma or at autopsy. Histopathologically, the condition is characterized by cortical meningiovascular proliferation, perivascular spindle-cell proliferation through Virchow-Robin spaces, fibrosis, hyalinization, calcification and even osseous metaplasia. The pathogenesis of MA is still obscure. Previous reports have suggested that the origin of MA lesions is most probably a hamartomatous proliferation of meningotheial cells.^{9–11} In this study we present the histopathological features of five cases without stigmata of neurofibromatosis type 2. The histopathological spectrum and differential diagnoses are discussed with a review of the literature.

MATERIALS AND METHODS

We reviewed the neuropathological records at our department to find all cases of MA that were recorded from 2000 to 2004. Five cases were identified, three patients are male and two are female, ranging in age from 12 to 36, mean 21.6. One, whose lesion was found incidentally after a head trauma, was asymptomatic. Four had histories of seizures. No stigmata of neurofibromatosis type 2, including bilateral acoustic Schwannomas or masses of the spinal cord,

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Received 25 July 2005; revised and accepted 31 August 2005.

Table 1 Clinical summary of five cases with MA

Case	Age/ sex	Duration of illness (years)	Clinical presentation	Neurological examination	Location	Clinical diagnosis	Surgical treatment
1	26/M	–	Asymptomatic	Normal	Right temporal lobe	Calcific lesion	Gross total resection
2	15/F	3	Refractory seizures	Normal	Right occipital lobe	AVM	Gross total resection
3	36/F	0.5	Seizures	Normal	Left frontal lobe	Low grade glioma	Gross total resection
4	19/M	0.5	Seizures	Normal	Left parietal lobe	CA	Gross total resection
5	12/M	7	Refractory seizures	Normal	Left frontal lobe	Low grade glioma	Gross total resection

AVM, arteriovenous malformation; CA, cavernous angioma.

were found using MRI or family history in any of the cases. General physical and neurological examinations were normal. Follow-up duration ranged from 10 to 48 months. In one case (patient 1) follow-up was not possible after surgery. Detailed clinical information is summarized in Table 1.

The resected tissues were fixed in 10% neutral formalin solution. Paraffin-embedded 6- μ m thick sections were then stained with HE, Klüver-Barrera (KB), reticulum fiber and Bodian methods. Immunohistochemical examinations were performed using the standard streptavidin technique with appropriate positive and negative controls. The following antibodies were employed: epithelial membrane antigen (EMA, M0613, DAKO, Glostrup, Denmark, 1:100), Vimentin (M0725, DAKO, 1:100), CD34 Class II (M7165, DAKO, 1:50), S-100 protein (Z0311, DAKO, 1:100), Smooth Muscle Actin (SMA, M0851, DAKO, 1:200), Cytokeratin (M3515, DAKO, 1:100), Glial Fibrillary Acidic Protein (GFAP, M0761, DAKO, 1:100), Synaptophysin (A0010, DAKO, 1:200), Neurofilament Protein (M0762, DAKO, 1:100) Ubiquitin (Z0458, DAKO, 1:100), Tau (A0024, DAKO, 1:100) and Ki-67 (M7240, DAKO, 1:50).

RESULTS

Clinical summary

Case 1, a 26-year-old man with an unremarkable medical history, was admitted to our hospital in March 2000 due to an incidental head trauma. Head computed tomography (CT) scan revealed a well-demarcated calcific lesion in the right temporal lobe with surrounding low density. Clinically, calcific lesion was suspected. Intraoperatively, the leptomeninges and subarachnoid space overlying the right temporal region contained a 2.5-cm spherical calcification filling a sulcus and compressing the cortical surface. The calcific lesion and surrounding infiltration were totally removed. Unfortunately, after the operation its follow-up was not possible.

Case 2 was a 15-year-old girl with a history of complex partial seizures for 3 years who was admitted to our hospi-

tal in August 2000 as a result of progressively intractable complex partial seizures with secondary generalization. She also suffered from headache and vomiting. Head CT scan revealed a low-density lesion in the right occipital lobe. On MRI, the cortical lesion was hypointense on T1-weighted images and showed low signal on T2-weighted images with surrounding hyperintense lesion. Radiologically, arteriovenous malformation was suspected. The lesion and adjacent cortex were resected. The postoperative course was uneventful and no neurological deficits were noted. A 4-year follow-up did not reveal any recurrence of the lesion.

Case 3, a 36-year-old woman with an unremarkable medical history, was admitted to our hospital in August 2001 due to generalized seizures, which had developed 6 months previously. Brain MRI revealed a left frontal mass lesion whose signal was hypointense on T1-weighted images and hyperintense on T2-weighted images, with surrounding increased T2 signal. A low-grade glioma was suspected. She underwent left frontal craniotomy and lesionectomy. The lesion appeared gray, firm, and well demarcated. Gross total removal of the lesion was achieved. The postoperative course was uneventful and no neurological deficits were noted. A 3-year follow-up did not reveal any recurrence of the lesion.

Case 4 was that of a 19-year-old man with an unremarkable medical history who was admitted to our hospital in June 2004 due to generalized seizures, which had developed 6 months previously. Brain CT revealed a 2.0-cm well-demarcated ovoid mass without peritumoral edema in the left superficial gyrus of the parietal lobe (Fig. 1). Clinically, cavernous angioma was suspected. Intraoperatively, the mass was a well-demarcated, gray and stony, hard, globular lesion. Gross total removal of the lesion was achieved. During the 10-month follow-up, there was no recurrence of seizure or tumor.

Case 5, a 12-year-old boy with progressively intractable complex partial seizures since the age of 5, was admitted to our hospital in July 2004 as a result of headache and vomiting. There was no history of perinatal problems, fever-related seizures or developmental delay. A brain MRI revealed a cystic lesion with increased cortical signal in the

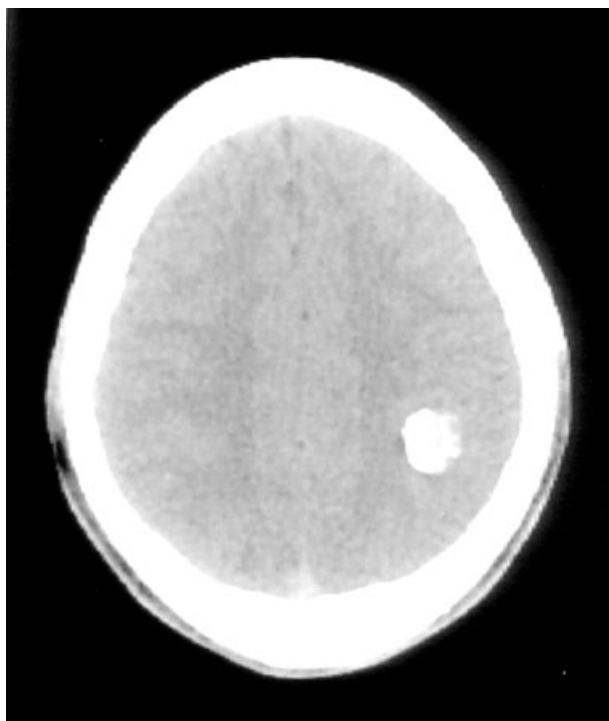


Fig. 1 Axial CT scan showing a nodular, calcific mass in the left parietal lobe.



Fig. 2 Sagittal MRI T1-weighted image showing a cystic lesion with increased cortical signal in the left frontal lobe.

Table 2 The main histopathological findings of five cases

Case	Meningiovascular proliferation	Perivascular cuffs of spindle cells proliferation	Perivascular connective tissue proliferation	Reticulum fiberde position	Calcification	Nervous tissue	
						Gliosis	NFT
1	+	+	+	+	+	+	+
2	+	+	+	+	-	+	-
3	+	+	+	+	-	+	-
4	+	+	+	+	+	+	-
5	+	+	+	+	-	+	-

NFT, neurofibrillary tangles.

left frontal lobe on T1-weighted image, interpreted as low-grade glioma or cystic encephalomalacia (Fig. 2). The patient underwent left frontal craniotomy and lesionectomy. Postoperatively, he continued to take anticonvulsant medication and has remained seizure-free with no evidence of lesion recurrence for 10 months.

Histopathological findings

All five cases showed similar histopathological features. The main histopathological findings are summarized in Table 2.

In case 1, the lesionectomy specimen contained a 3.0-cm calcification largely filling a sulcus in the leptomeninges.

The adjacent cortex was of a whitish color and firm consistency. Microscopically, the calcified leptomeningeal mass was composed of multilobulated amphophilic nodules with a concentric arrangement of acellular lamellae, some were centrally dense, hyalinized and resembled fibrocalcifying component (Fig. 3A). The adjacent cortex showed rows of proliferating oval to spindle cells with hyperchromatic nuclei, psammoma bodies, and numerous slit-like capillaries. Neither mitosis nor necrosis was found. Histopathology confirmed the diagnosis of MA.

In case 2, the surgical specimen, measuring 1.0 cm in diameter, was gray. Microscopically, the lesion was confined to the cortex with focal involvement of the overlying leptomeninges. The adherent cortex showed prominent

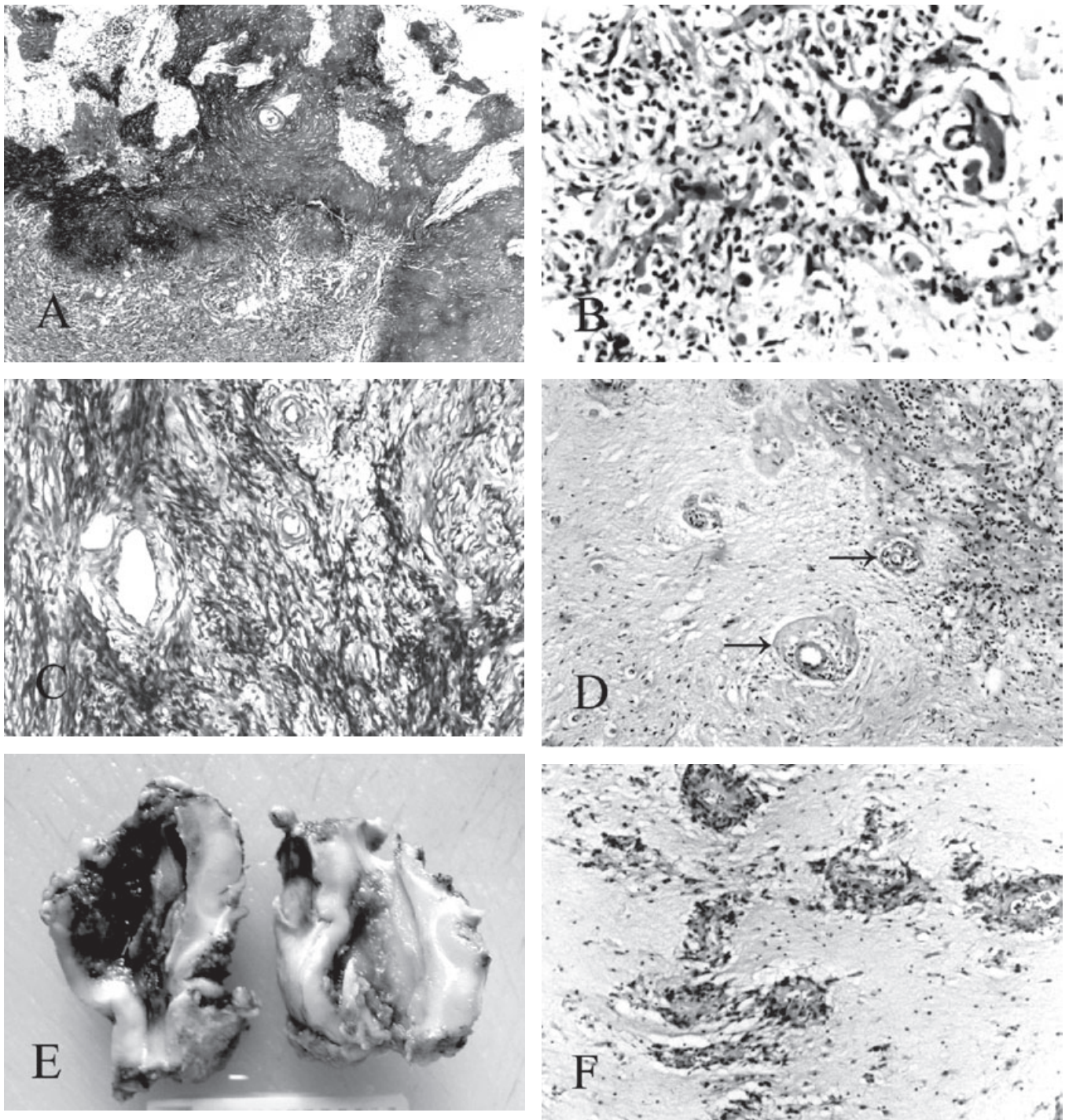


Fig. 3 Light microscopic features of the MA: (A) case 1, fibrocalcifying nodule with spindle cells proliferation in the adjacent cortex (HE, $\times 40$); (B) case 2, predominantly vascular case with numerous capillaries and venules in the cortex (HE, $\times 200$); (C) case 3, highly cellular area showing fascicular pattern; there is complete effacement of cortical architecture (HE, $\times 100$); (D) case 4, some vessels of the cortex with thickened vascular wall and minimal cuffs of spindle-cell proliferation (arrows) (HE, $\times 100$); (E) case 5, cut sections revealed a pale, rubbery and gritty intracortical lesion with cystic formation; and (F) case 5, perivascular spindle-cell proliferation presenting in the white matter (HE, $\times 100$).

proliferation of microvessels, mainly capillaries and venules (Fig. 3B). Cuffs of spindle cells surrounded them. No calcification was noted. These findings were similar to MA with a predominant vascular pattern according to the classification of Wiebe *et al.*¹²

In case 3, the lesionectomy specimen, measuring $3.5 \times 2.5 \times 1.2$ cm, was of a whitish color and firm consistency. Microscopically, the most prominent feature of the transcortical plaque was a complex of small-caliber blood vessels lined by a single layer of endothelial cells and encircled by spindle cells in cuffs as well as parallel bundles (Fig. 3C). Neither cellular atypia nor necrosis was found. Some blood vessels had fibrosed or hyalinized vascular walls. A dense reticulum and collagen fiber network was aggregated around the bigger blood vessels. No calcification was noted. These findings suggested MA with a predominant cellular pattern according to the classification of Wiebe *et al.*¹²

In case 4, the lesionectomy specimen, measuring 1.2 cm in diameter, had leptomeningeal calcific deposits and a cortical plaque. The main histopathological features of this case were similar to case 3. It was characterized by a proliferation of leptomeningeal vessels and perivascular cuffs of spindle cells within the cortex. The perivascular arrangement of spindle cells was more evident in the periphery of the lesion. Some vessels were surrounded by two or three layers of concentrically arranged spindle or oval cells (Fig. 3D). Sparse lymphocytic infiltrations were present in the leptomeninges and near some cortical vessels. Multiple calcified deposits were found in the leptomeninges and cerebral cortex. Abundant reticulum fiber with a pericellular distribution was observed. MA with a predominant cellular pattern was established in this patient.

In case 5, The lesionectomy specimen measured $4 \times 3 \times 2$ cm. Cut sections revealed a pale, rubbery and gritty intracortical lesion with cystic formation (Fig. 3E). Microscopically, the lesion was composed of elongated spindle cells with wavy nuclei, forming moderately- to highly-cellular, focally palisading zones around fibrillar areas in which cell nuclei were scarce. Although the compact areas were mainly localized to the cortex perivascular spindle-cell proliferation was also observed in the white matter (Fig. 3F). No calcification was noted. The appearance was that of MA with a predominant cellular pattern.

On immunohistochemical analysis, the proliferating spindle-cell population expressed vimentin uniformly. Results for other markers varied: two cases (patients 4, 5) showed focal EMA positivity and three (patients 1, 3, 4) were focally positive for CD34. All were negative for cytokeration, S-100 protein, SMA, GFAP and neurofilament. The Ki-67 proliferation index for all the five cases was very low, less than 0.1%.

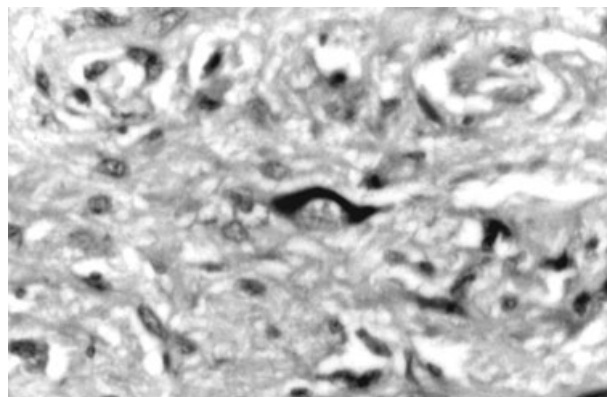


Fig. 4 Tau-positive neurofibrillary tangles within cytoplasm of neuron in the cortical lesion of case 1 (Tau-immunostain, $\times 400$).

All the cases showed gliosis and neuronal loss with residual neurons presenting with chronic non-specific degenerative changes within the lesion areas and in the neighboring cortex. Bodian staining and Ubiquitin, Tau immunostaining demonstrated neurofibrillary tangles (NFT) in the cortex adjacent to the lesion only in one case (patient 1) (Fig. 4).

DISCUSSION

Meningioangiomas is treated by surgery and, after total excision, the lesion does not regrow.¹³⁻¹⁶ Although we have described five cases of sporadic MA, presurgical diagnosis remains difficult because diagnostic tools lack specificity. CT and MRI erroneously suggested low-grade glioma, vascular malformations and calcific lesion in our cases. CT and MRI findings reported in MA are controversial; they depend on the developmental histological status of the lesion.¹⁷⁻¹⁹ Radiological appearances are mostly not specific for the diagnosis. On CT, MA is usually iso- to slightly hyperintense with or without the features of calcification, but low-density lesions and normal CT images have also been reported.^{17,18} The MRI findings are variable. Partington and Nomura have reported iso-intensity with gray matter on T1 and hypointensity on T2-weighted images.^{20,21} Thus, a wide spectrum of imaging expressions of MA often impedes the clinical diagnosis.

The histopathological features of our cases are summarized in Table 2. All our patients' MA lesions were confined to the cortex with variable involvement of the overlying leptomeninges. Although all the cases had unifying features, such as cortical vascular proliferation and perivascular cuffs of spindle-cell proliferation, each case was unique. Wiebe *et al.* described the histopathological spectrum of MA,¹² which can be broadly classified into pre-

dominantly cellular and predominantly vascular types. The predominant cellular pattern was to show moderate to high cellularity, as in our present cases (patient 3, 4, and 5). Varying architecture was noted, consisting of focal areas of storiform, rhythmic palisading and fascicular patterns. All cellular cases had lesional cells that, in some areas, appeared to emerge from a perivascular location and infiltrate the cortex. This occurred centrally within the lesions, where cellularity was most dense. Peripherally, the perivascular relationship of the cells became evident. Moreover, patient 5 showed the white matter involved. The predominantly vascular pattern, as in our patient 2, contained thick-walled, hyalinized blood vessels and increases in numbers with minimal perivascular cell proliferation. Different forms of calcification are usually encountered in MA. Our patient 1 showed the arrangement of concentric lamination of calcific concretions. Similar changes were also recorded in previous reports, termed fibro-osseous, fibrocalcifying component or focal bony metaplasia.²²⁻²⁷ So, the histopathological feature of patient 1 suggested MA with a predominant fibrocalcifying formation. Our cases indicated that the morphological changes of the MA lesions were variable. Depending on the age of the lesion, we consider that MA might be divided into three types, predominantly cellular type, vascular type and fibrocalcifying type.

The histopathological diagnosis of MA depends on its localization, size, and the chronicity of the lesion.^{11,14,22} Many cases showed proliferating perivascular cells infiltrating the cortex in association with marked cellularity and reactive gliosis. Unless the pathologist is familiar with the histopathological features of MA, these features may lead to an erroneous diagnosis. The histopathological differential diagnoses include meningiomas with cortical invasion, vascular malformation and gliomas. Meningiomas exhibit a predominance of meningothelial cells and do not have the pronounced vascularity seen in MA. An angiomatous meningioma contains numerous mature blood vessels with meningothelial cells in varying proportions, but neurons and glial cells are absent. An apparent meningothelial component may raise suspicion of an atypical or anaplastic meningioma with cortical invasion, but a lack of cellular atypia, mitotic activity, necrosis and cortical destruction favor MA. Vascular malformation tends to bleed, which is an extremely rare feature of MA since the lesion is firm due to the presence of a variety of mesenchymal and neural elements.^{28,29} The presence of small-sized vessels, their uniform distribution, lack of hemorrhage, normal intervening tissue, associated spindle-cell proliferation and superficial location should preclude diagnosis of vascular neoplasms. The elongated, spindly cells may be mistaken for astrocytes. Furthermore, prominent endothelial proliferation might result in an erroneous diagnosis of

a high-grade glioma. However, the absence of the typical 'copper wire' appearance of astrocytic fibrils and glomeruloid vessels and the presence of neurons within the lesion should preclude that diagnosis. On the other hand, we should pay great attention to recent reports concerning MA coexisting with meningioma, oligodendroglioma and vascular malformation.³⁰⁻³⁸

The origin of the MA lesion is not clear. Basically, it was considered a hamartomatous proliferation of meningothelial cells, blood vessels and fibroblasts in variable proportions.^{11,39} Foci of meningothelial hyperplasia are known to appear in the context of von Recklinghausen's disease,⁴⁰ and this could be a plausible origin for those cases of MA associated with neurofibromatosis. On the other hand, the observation of abnormal local vascularization in some cases has led several authors to regard MA simply as a vascular malformation with added meningothelial reaction.³³ However, recent immunohistochemical studies did not support a meningothelial origin for the perivascular cells, but suggest that pluripotent cells may differentiate into various cell types found in MA.^{12,15,41-43} Immunohistochemistry has limited diagnostic value as staining patterns vary among MA cases. Our immunostaining results parallel those in the literature. Only vimentin, as a non-specific marker of the mesenchymal cell was consistently positive. EMA expression, as a marker for arachnoid cap cells was noted in two of the five cases. These findings indicated that perivascular mesenchymal components, such as fibroblasts and perivascular connective tissue might be playing an important role in the pathogenesis of MA.

Neurofibrillary tangles have been described in a wide spectrum of diseases and are seen in the setting of degeneration within neuronal cytoplasm. These changes were identified in only one of our five cases. In the largest reported series of MA, Halper *et al.*¹³ described the presence of NFT within cortical neurons in five of their six reported cases. Only rarely have tangles been noted by others in the literature.^{12,41,44} Therefore, Halper and his colleagues hypothesized that the presence of NFT in MA more likely represented a secondary degenerative phenomenon rather than an intrinsic component of the lesion.¹³

Finally, the diagnosis of MA is important since it is a hamartomatous condition that presents as a mass-occupying lesion within the cortex. It should be considered in differential diagnosis of intracortical lesions, especially in children and young adults. The superficial location, perivascular cuffs of spindle-cell proliferation with variable involvement of the overlying leptomeninges and calcification, and small plump vessels, should clinch the diagnosis. It is important to make a correct diagnosis in order to avoid further aggressive treatment.

ACKNOWLEDGMENTS

The authors thank Prof Yoichi Nakazato, First Department of Pathology, School of Medicine, Gunma University, Gunma, Japan for the consultation of case 5.

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