

# Sporadic meningioangiomas with and without meningioma: analysis of clinical differences and risk factors for poor seizure outcomes

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## Abstract

**Background** Meningioangiomas (MA) is a rare cerebral lesion. Sporadic MA occasionally combines with meningioma (MA-M). The aim of the present study was to clarify whether MA-M and pure MA have clinical differences and to determine risk factors for unsatisfactory seizure outcomes in sporadic MA.

**Methods** We reported 14 sporadic MA cases in our center and conducted a literature review. We compared the demographic, clinical, imaging, electrophysiological and pathological features and surgical outcomes. Logistic regression analysis was performed to evaluate the risk factors for poor seizure outcomes.

**Results** MA-M cases showed a more prominent male predilection (4.2 times vs. 1.6 times,  $p=0.04$ ), a shorter duration of symptoms ( $2.8\pm 0.8$  years vs.  $5.2\pm 0.6$  years,  $p=0.02$ ), and a lower seizure incidence (53.6 % vs. 89.3 %,  $p<0.001$ ) as

compared to pure MA. A gyriform alteration on imaging was exclusively associated with pure MA. The Ki-67 was higher in the meningioma component than in the MA component in MA-M ( $1.2\pm 0.3$  % vs.  $6.1\pm 1.1$  %,  $p<0.001$ ). Lesions located in the temporal lobe predicted poor seizure outcomes ( $p=0.02$ ,  $OR=4.4$ , 95 % confidence interval, 1.24–15.89).

**Conclusion** Clinical differences may be caused by the different biological natures. MA-M seems to be a neoplastic lesion, while pure MA seems to be a non-neoplastic lesion. Long-term follow-up is required for MA-M. Because the coexistence of hippocampal sclerosis may explain the poor seizure outcomes of MA located in the temporal lobe, it is important to identify underlying hippocampal sclerosis and to perform complete resection.

**Keywords** Meningioangiomas · Meningioma · Seizures · Gyriform

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## Introduction

MA is a rare cerebral lesion characterized by a plaque-like or nodular mass within the leptomeninges and the underlying cerebral cortex. The main histopathological features of MA are leptomeningeal meningotheelial proliferation and meningovascular proliferation [1]. MA can occur sporadically or in association with neurofibromatosis type two (NF2). Sporadic MA clinically presents intractable seizures or vague neurological complaints (headache, nausea). Occasionally, sporadic MA combines with a neoplasm, most commonly meningioma (MA-M) [2]. MA is usually considered a benign, non-neoplastic lesion, while genetic and molecular studies have suggested that MA-M may be a neoplasm [3–5]. Given the

neoplastic nature of MA-M, it is reasonable to assume that the clinical features of MA-M may be different from those of pure MA. However, studies undertaking comprehensive comparisons of MA-M and pure MA are almost entirely lacking. In this article, we analyzed 14 sporadic MA cases collected in our center and 160 cases reported previously [1, 3–79]. We compared the demographic, clinical, electrophysiological, imaging and pathological features, as well as the surgical outcomes, between the MA-M and pure MA groups. Clinical features that influenced seizure outcomes were also assessed.

## Materials and methods

### Patients

A total of 14 sporadic MA patients (7 MA-M cases and 7 pure MA cases) were treated in the neurosurgery department of our center between 2009 and 2013. All of the patients were initially operated on in our center. The information, including the types and duration of the seizures, was obtained from the patients' charts and the database. Each patient had at least one preoperative brain magnetic resonance imaging (MRI) and long-term video electroencephalography (EEG). The preoperative evaluation was conducted by a senior neurologist and senior neurosurgeon. All patients underwent surgery by three senior neurosurgeons in our center. Seizure and MA outcomes were followed up. All protocols were reviewed

and approved by the institutional review board of Beijing Tian Tan Hospital.

### Literature analysis

We conducted a literature search of the Embase and PubMed databases until 2013, using the following subject headings (MeSH) and keywords: meningoangiomas and meningoangiomas. After reviewing abstracts, MA-M cases and pure MA cases were retrieved. Case selection was based on the full text, and all duplicate cases were discarded. A comparison concerning demographic features, clinical presentation, location of MA, imaging, EEG, immunohistochemistry and surgical outcomes was performed between MA-M cases and pure MA cases. Whether the patient had meningioma or not, age, sex, symptom duration and MA location were evaluated for their influences on seizure outcomes.

### Statistical methods

Descriptive statistics were applied to analyze the distributions of demographic and clinical data. The *t* test was performed for continuous variables and the chi-square or Fisher's exact test for discrete variables. Analysis of seizure outcomes was evaluated by binary logistic regression; seizure outcomes were divided into seizure-free (Engel I) and unsatisfactory (Engel II/III/IV) groups. All of the data were analyzed using SPSS software (SPSS, version 18.0 for Windows; SPSS Inc.,

**Table 1** Summary of clinical features and surgical outcomes of 14 sporadic MA cases in our center

| Case no. | Sex/age (years old)/<br>duration of symptoms<br>(months) | Symptoms           | Location               | Histologic type<br>of meningioma | Ki-67 proliferation<br>index (%) |            | Removal | Follow-up time<br>(months)/seizure<br>outcome/MA outcome |
|----------|--|--------------------|------------------------|----------------------------------|----------------------------------|------------|---------|--|
|          |  |                    |                        |                                  | MA                               | Meningioma |         |  |
| 1        | M/30/1.5   | Headache           | Right frontal lobe     | Transitional                     | 0.8                              | 3.1        | Total   | 24/SF/NR   |
| 2        | M/32/180   | GTCS               | Right temporal lobe    | Transitional                     | 1.0                              | 5.3        | Total   | 39/SF/NR   |
| 3        | M/3/0.3  | CPS                | Corpus callosum        | Transitional                     | 2.5                              | 8.2        | Total   | 20/SF/NR   |
| 4        | M/12/24  | CPS                | Left parietal lobe     | Transitional                     | 5.0                              | 8.2        | Total   | 12/SF/NR   |
| 5        | M/23/144   | GTCS               | Right parietal lobe    | Fibrous                          | 1.3                              | 4.3        | Total   | 20/SF/NR   |
| 6        | M/13/24  | Diabetes insipidus | Third ventricle        | Fibrous                          | 2.5                              | 5.5        | Partial | dead   |
| 7        | M/23/18  | CPS                | Left temporal lobe     | Psammomatous                     | 2.0                              | 5.0        | Total   | 10/SF/NR   |
| 8        | F/10/108   | GTCS               | Right frontal lobe     | No meningioma                    | 3.2                              |            | Total   | 58/SF/NR   |
| 9        | F/25/7   | ABS+GTCS           | Right temporal lobe    | No meningioma                    | 1.1                              |            | Total   | 33/SR/NR   |
| 10       | F/26/84  | GTCS               | Right parietal lobe    | No meningioma                    | 1.5                              |            | Total   | 46/SI/NR   |
| 11       | F/5/0.6  | GTCS               | Right temporal lobe    | No meningioma                    | 2.2                              |            | Total   | 22/SF/NR   |
| 12       | M/10/0.6   | GTCS               | Right parietal lobe    | No meningioma                    | 5.0                              |            | Total   | 20/SF/NR   |
| 13       | M/3.5/10   | GTCS               | Anterior cranial fossa | No meningioma                    | 2.2                              |            | Total   | 12/SF/NR   |
| 14       | M/27/156   | GTCS               | Right temporal lobe    | No meningioma                    | 1.1                              |            | Total   | 10/SI/NR   |

ABS absence seizure, CPS complex partial seizure, GTCS generalized tonic-clonic seizure, F female, M male, NR no recurrence, SF seizure free, SI seizure improved

Chicago, IL, USA).  $p$ -values  $<0.05$  were considered statistically significant.

## Results

### Patients

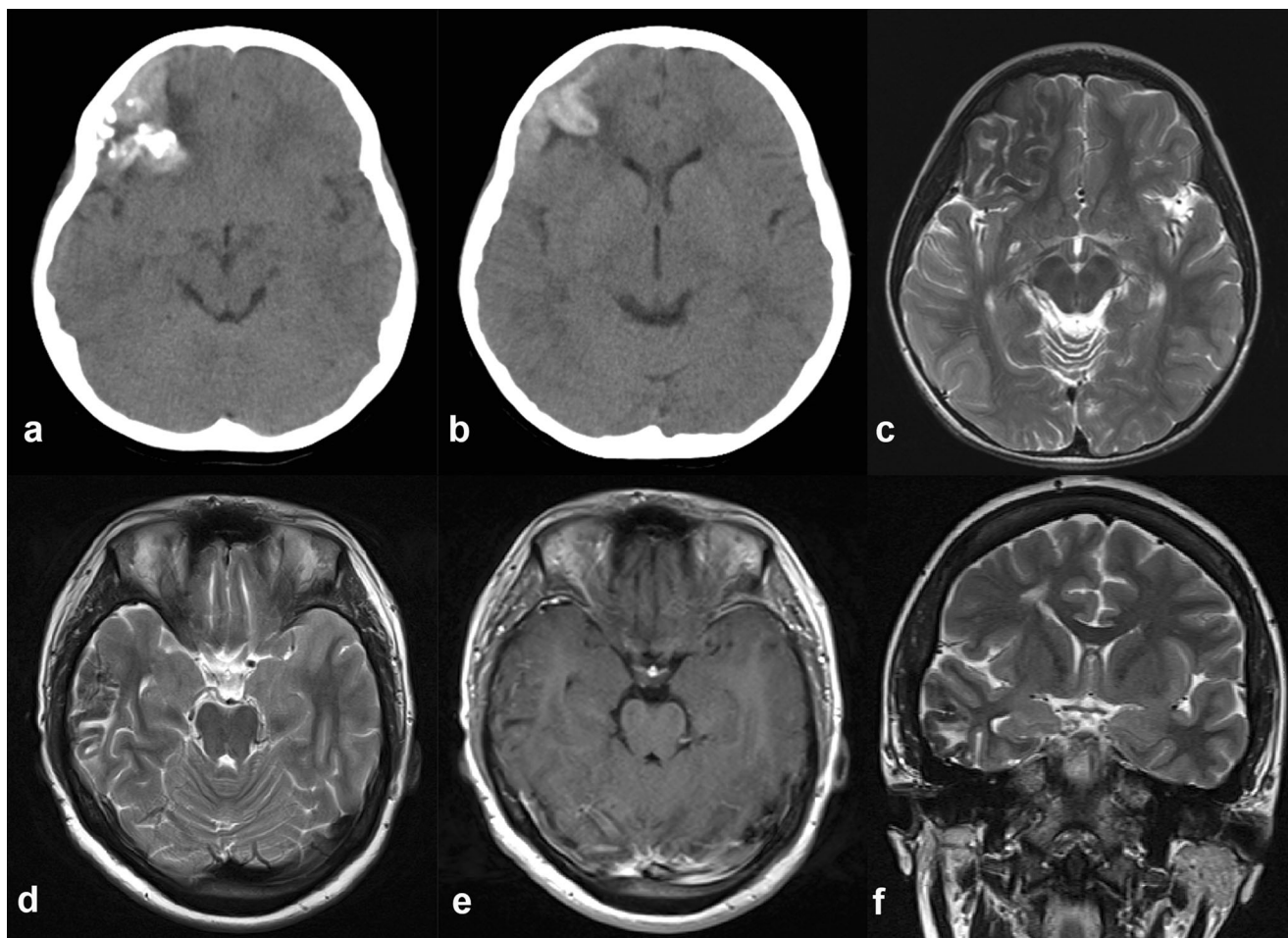
Our records yielded 14 patients, including 7 patients with pure MA and 7 patients with MA-M (Table 1). The imaging characteristics were quite variable and nonspecific. Gyriform alterations on computed tomography (CT) and MRI were found in cases 8 and 9 (Fig. 1). During surgery, MA presented diverse textures, colors and blood supplies. Most of the lesions were located in the cortex, but some were found in the white matter (case 12), third ventricle (case 6) and corpus callosum (case 3). The electrocorticography (ECoG) data showed that spikes and sharp waves originated from MA lesions in seven

patients. In the other five patients, epileptic discharges were recorded within MA lesions and the adjacent cortex. Ten of the 12 patients with seizures were seizure free postoperatively. No tumor recurrence occurred.

### Illustrative cases

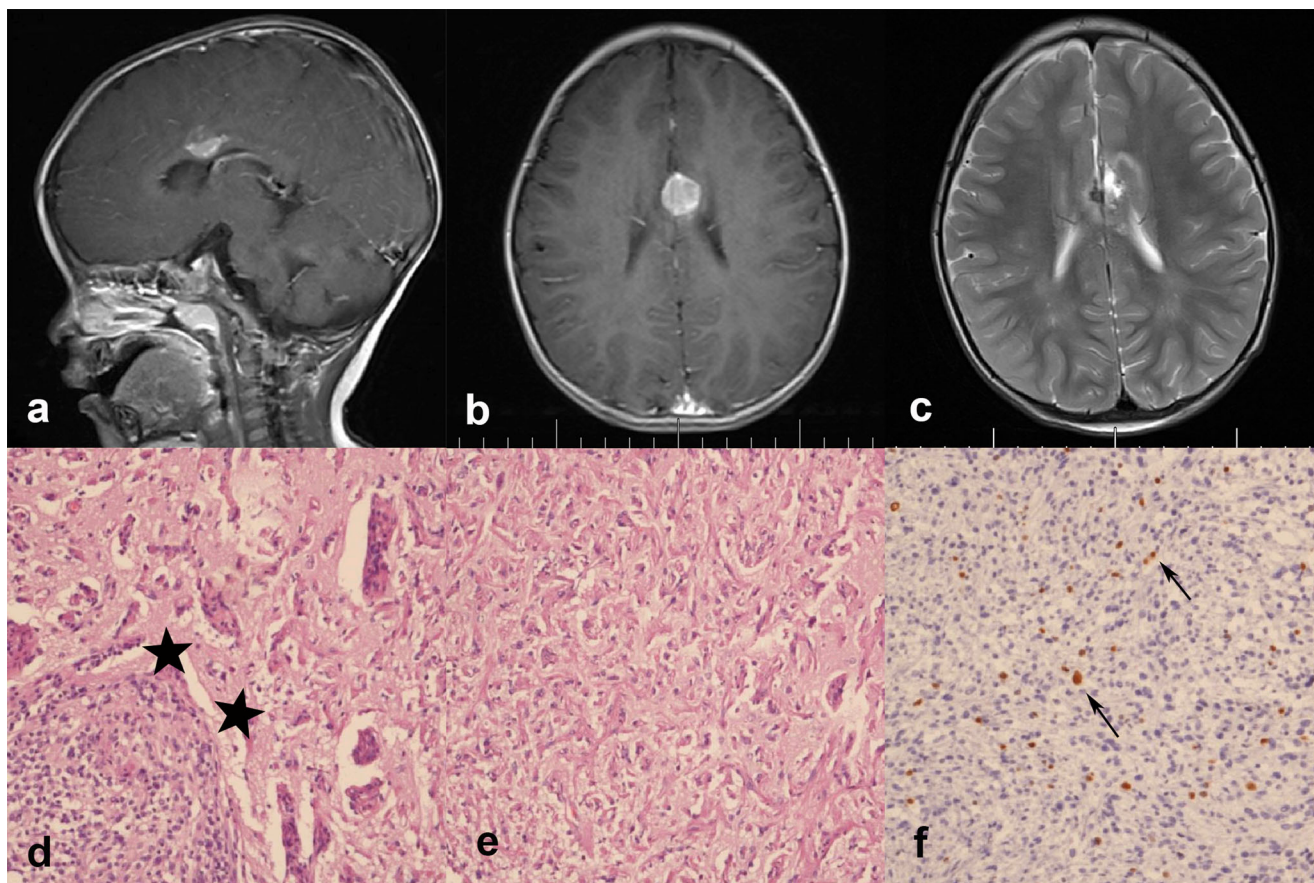
#### Case 3

*MA-M patient with the lesion located in the corpus callosum* A 3-year-old boy suffered from complex partial seizures for a month. There was no history of perinatal problems, febrile seizures or developmental delay. MRI revealed an intracranial mass measuring  $2 \times 1.5 \times 1.5$  cm located in the corpus callosum. The lesion was hypointense on T1-weighted imaging (T1WI) and hyperintense on T2-weighted imaging (T2WI), with obvious homogeneous enhancement (Fig. 2a and b). A soft yellow mass was completely removed, and at



**Fig. 1** **a** Case 8: Axial CT showing a hyperdense lesion located in the right frontal lobe with calcification. **b** Case 8: Axial CT revealing a hyperdense mass with gyriform alteration. **c** Case 8: Axial T2-weighted image demonstrating a gyriform hypointense lesion in the right frontal lobe. **d** Case 9: Axial T2-weighted image presenting a gyriform lesion in

the right temporal lobe. **e** Case 9: Axial T1-weighted imaging showing inconspicuous enhancement within the lesion. **f** Case 9: Coronal T2-weighted image demonstrating a right temporal lesion with gyriform alteration



**Fig. 2** Case 3. **a, b** Enhanced MRI showing homogeneous enhancement located in the corpus callosum. **c** Postoperative 3-month MRI (T2WI) indicating total removal of the lesion and no recurrence. **d** Transition zone between the MA component and meningioma component

(*asterisks*). Hematoxylin and eosin, original magnification  $\times 100$ . **e** The histological appearance of the MA component. Hematoxylin and eosin, original magnification  $\times 100$ . **f** Ki-67-positive cells (*arrows*) in the meningioma component, original magnification  $\times 100$

the 3-month follow-up the MRI showed no recurrence (Fig. 2c). After histopathological examination, MA-M was confirmed, and a transition zone between the MA component and meningioma component was observed (Fig. 2d and e). Ki-67 was observed in 2.5 and 8.2 % of the cells of the MA and meningioma, respectively (Fig. 2f). Preoperatively, ictal EEG showed bilateral frontal and temporal epileptic activities (Fig. 3). The patient was seizure free at last follow-up (Fig. 4).

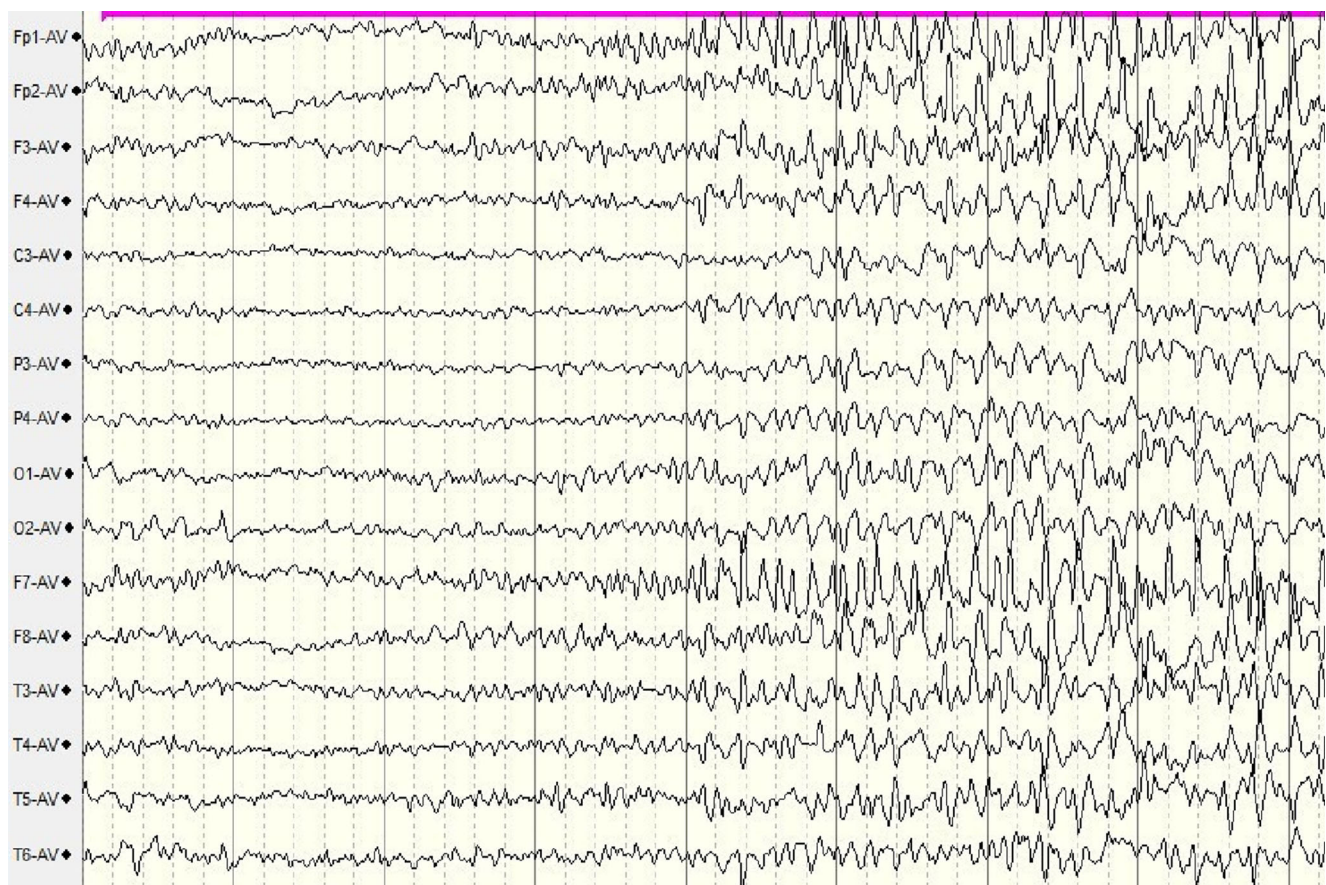
#### Case 12

**Pure MA patient with MA located in the white matter** A 10-year-old boy presented with a 20-day history of simple partial seizures. There was no history of febrile seizures. CT revealed a hypodense round mass with calcification in the right parietal lobe (Fig. 5a). MRI revealed that the lesion was located in the white matter without extension into the cortex. Ring-like enhancement was observed (Fig. 5b). The lesion was hypointense with a central cerebrospinal-like area on T1WI and hyperintense on T2WI (Fig. 5c). During surgery, a soft, lightly vascularized lesion was completely removed, and at

the 3 month follow-up the lesion presented no recurrence (Fig. 5d). Under microscopy, the specimen showed numerous small vessels ensheathed by spindle-shaped cells (Fig. 5e). The cortical parenchyma showed reactive astrocytes associated with gliosis (Fig. 5f). Ki-67 was detected in less than 5 % of the cells. Preoperatively, EEG indicated a right occipital and temporal slow wave (Fig. 6). The patient was free of seizures at last follow-up (Fig. 7).

#### Histopathology

Specimens demonstrated numerous spindle-shaped cells arranged in fascicles or in a storiform pattern surrounding the small blood vessels, with or without psammoma bodies. The nuclei of the spindle-shaped cells were normal, and no features or mitosis was found. The histological type of the meningioma component was variable (Table 1). Vim, CD34 and GFAP were positive in most cases, while other markers were expressed differently. Ki-67 was higher in meningioma components than in MA components in MA-M cases ( $2.2 \pm 0.5$  vs.  $5.6 \pm 0.7$  %,  $p < 0.001$ ).



**Fig. 3** Ictal EEG in case 3 shows bilateral frontal and temporal epileptic activities

#### Literature review

A total of 160 sporadic MA patients (35 MA-M cases and 125 pure MA cases) were collected in 75 previous publications. Combined with our 7 MA-M cases and 7 pure MA cases, 174 patients were analyzed as follows (Fig. 8).

#### Demographic features

The mean ages at the time of diagnosis were nearly identical for MA-M and pure MA. A more obvious male predilection was found in the MA-M group. In addition, the MA-M cases showed a shorter duration of symptoms before diagnosis (Table 2).

#### Clinical presentation

As the most common symptom, seizures were found in 116 patients (79.5 %). The MA-M group presented a significantly lower incidence of seizures than the pure MA group (Table 2). There were no differences in seizure types between MA-M and pure MA cases (Table 2). Other atypical symptoms included headache, vomiting, diabetes insipidus, facial weakness, muscle atrophy and pain.

#### Location of MA

A single lesion was the predominant pattern of sporadic MA, with only six patients presenting with bilateral or multifocal lesions [24, 39, 60, 66, 69]. The frontal and temporal lobes were affected in 48 (32.2 %) and 52 patients (34.9 %), respectively. In addition to the posterior head, the cingulate gyrus, corpus callosum, third ventricle and brain stem were occasionally involved. No significant difference was found in the location between the MA-M and pure MA (Table 2).

#### Imaging

The most common finding on CT was a round, hypodense mass with varying degrees of calcification, occurring in 49 of 95 (51.6 %) patients (Table 2). On MRI, the lesions were confined to the cortex in 90 (88.2 %) of 102 patients. On T1WI, MA usually demonstrated a low- or isointense signal, occasionally with a mixed signal. On T2WI, 73.2 % lesions were hyperintense, while the others were hypointense. Gadolinium enhancement was applied in 65 patients, of whom 50 (76.9 %) showed minimal enhancement to substantial enhancement. There were no significant differences in imaging features between MA-M and pure MA (Table 2). However, A



**Fig. 4** Postoperative EEG in case 3 is normal with no epileptic activity

total of 17 patients [1, 10, 18, 32, 45, 69, 78, 80], including case 8 and case 9 in our series (Fig. 1), showed a gyriform signal pattern on either CT or MRI, which were all pure MA patients.

### EEG

Fifty-four patients underwent scalp EEG, and 41 of them (75.9 %) showed congruence on the location of epileptiform discharges and MA lesions. The remainder of the patients presented multifocal spikes, generalized spike waves or sporadic spikes. ECoG data, obtained from 18 patients, recorded spike or sharp waves in accordance with MA in 9 patients (50 %), whereas, the other 9 (50 %) patients showed spikes originating from the surrounding cortex of the lesion. There were no differences in ECoG data between the MA-M and pure MA cases (Table 2).

### Histopathology and immunohistochemistry

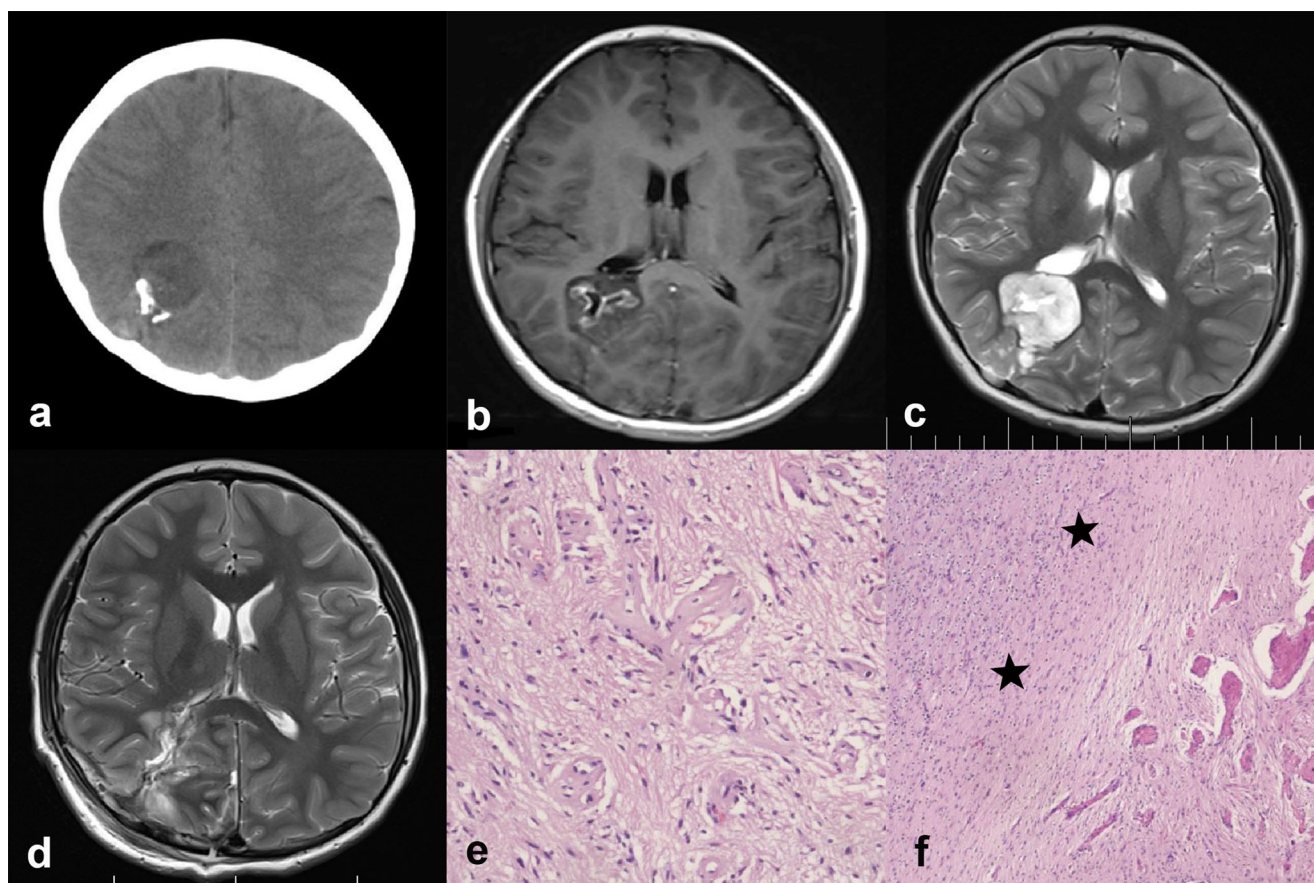
Histopathologically, the main features of MA were leptomeningeal meningotheelial proliferation and meningovascular proliferation. MA-M included features of

MA and meningioma; a transition zone between the MA and meningioma components was sometimes presented. The meningiomas were mostly of the transitional subtype (Table 3).

Immunohistochemically, Vim was expressed in nearly all of the MA cases: 38 (92.7 %) of 41 MA were Vim positive. Other immunostaining results, including for CD34, EMA, GFAP and S-100, were heterogeneous. There were no differences in Vim, CD34, EMA, GFAP and S-100 between the MA-M and pure MA groups (Table 4). Ki-67 was consistently low in MA (ranging from 0.1 to 15 %). There were no differences in Ki-67 between the MA-M and pure MA groups ( $1.2 \pm 0.3$  % vs.  $1.4 \pm 0.5$  %,  $p=0.67$ ) (Table 4). However, in MA-M, Ki-67 was higher in the meningioma component than in the MA component ( $1.2 \pm 0.3$  % vs.  $6.1 \pm 1.1$  %,  $p<0.001$ ).

### Outcomes after surgery

Only 2 patients had MA recurrence after surgery of a total of 111 patients [33, 39]. Seizure outcomes were followed up in 102 patients, with 89 (87.2 %) patients becoming seizure free (Engel I). Data obtained from 51 patients regarding the extent of surgery and the seizure outcome revealed that the seizure-free rate (88.6 %, 39 of 44) with total resection was slightly



**Fig. 5** Case 12. **a** CT scan revealing a hypodense round mass with calcification in the right parietal lobe. **b** Enhanced MRI scan showing enhancement of the lesion with surrounding hypointensity. **c** MRI showing hyperintensity of the lesion on T2WI. **d** Postoperative 3-month MRI (T2WI) indicating total removal and no recurrence of the MA lesion.

**e** Proliferating blood vessels are ensheathed by spindle cells. Hematoxylin and eosin, original magnification  $\times 100$ . **f** At the boundary of the MA in the white matter, gliosis is observed (asterisks). Hematoxylin and eosin, original magnification  $\times 40$

higher than that (71.4 %, 5 of 7) with partial resection, but with no significant difference ( $p=0.24$ ). Congruence on location of epileptiform discharges and MA on ECoG, as well as seizure type, had no correlation with seizure outcome ( $p=0.63$ ;  $p=0.92$ ). Additionally, the age, sex, MA size and duration of symptoms and whether MA was combined with a meningioma were not correlated with seizure outcomes. The location of the MA had a marked effect, with lesions in the temporal lobe indicating an unsatisfactory seizure outcome more often than extratemporal lesions ( $p=0.02$ , OR=4, 95 % confidence interval, 1.24–15.89) (Table 5).

## Discussion

MA is a rare, focal intracranial lesion that was first described by Bassoe and Nuzum in 1915 as an incidental autopsy finding in a patient with neurofibromatosis-2 (NF2) [81]. Takeshima et al. demonstrated that only 25 % of MA cases were associated with NF2, while in the remaining 75 %, it

occurred sporadically [72]. Occasionally, MA has been reported to coexist with meningiomas [11, 39, 54, 82], arteriovenous malformations [23], encephaloceles [75], oligodendrogliomas [50], meningeal hemangiopericytomas [39] and focal cortical dysplasia [65]. Among these, MA with meningioma is the most frequent combination. MA occurs mainly in children, adolescents and younger adults suffering from intractable seizures and headache [4, 39]. A previous study suggested that MA-M affected a younger age group than pure MA [15]. However, a similar predilection for young adults, as well as broad age distributions of MA-M patients and pure MA patients, was found. The limited number of MA-M cases may account for such a difference. MA usually presents with a male predisposition [69], consistent with the literature [4], and male predominance was more obvious in MA-M (unlike conventional meningioma) than in pure MA. This finding suggests that hormones may play an important role in the pathogenesis of MA-M. Wiebe et al. suggested that the temporal lobe was the most frequently involved in MA [1], while the frontal lobe seemed to be more affected, based on our analysis. Rarely, MA has been documented to occur in the



**Fig. 6** EEG in case 12 shows slow waves in the right occipital and temporal regions

diencephalon [25], in the brain stem [43] and outside the cerebral cortex [54]. In our series, we reported an MA-M lesion (case 3) that was believed to be the first reported MA-M case involving the corpus callosum (Fig. 3).

The variety of imaging findings of MA usually leads to erroneous diagnoses [1, 80]. Half of the sporadic MA cases (51.6 %) showed calcifying changes on CT, corresponding to the 55 % reported in the literature [1]. This suggests that, as a degenerative change, calcification on CT may be one of the most helpful features favoring the diagnosis of MA [39, 83]. In our analysis, we found the majority (73.2 %) of sporadic MA cases present hyperintensity rather than a mixed signal on T2WI as previously described [1, 24, 83]. MA is believed to be mainly confined to the cerebral cortex; the subcortical white matter is rarely involved [1, 45, 74]. Recently, an excellent correlation between sporadic MA located in the subcortical white matter and hyperintensity on T2WI was established by Jeon et al. [35]. With our imaging findings, this supports the notion that MA may involve the subcortical white matter as well as cortex [35].

It is difficult to distinguish between pure MA and MA-M on imaging. Pure MA can even present as meningioma-like dural attachment and homogeneous contrast enhancement

[21, 28]. In a few reported cases [1, 17, 32, 45, 69, 73, 80], including case 8 and case 9 in our series, pure MA has exhibited a unique presentation of gyriform alteration or gyriform enhancement (Fig. 1). This finding could be explained by the thickened cortex, where abundant proliferating leptomenigeal microvessels and spindle-shaped cells were located. However, if the lesion is completely calcified, it might present hypointensity without gyriform alteration on MRI [80]. Although the reason why gyriform changes were only found in pure MA is unclear, such gyriform alteration could help to distinguish pure MA from MA-M.

Immunohistochemistry has no specific diagnostic value for MA [41]. Only Vim, as a nonspecific marker of mesenchymal cells, was consistently positive [1, 74]. Interestingly, we found that the majority of MAs were CD34 positive (23 of 33). This finding suggests that, as an endothelial cell marker to detect microvascular proliferation activity, CD34 could be used as an ancillary method for identifying sporadic MA. As a proliferation index, Ki-67 was consistently low in MA; however, all of the literature pertaining to Ki-67, as well as our cases, has found that Ki-67 is higher in the meningioma component than in the MA portion [3–5, 61]. One reasonable explanation is that meningioma arises from the MA component with an





Fig. 7 Postoperative EEG in case 12 is normal with no epileptic activity

additional genetic alteration, which may confer more proliferation potential on it.

Overall, the pathogenesis of MA is unclear. A presumed hamartomatous or non-neoplastic nature is favored by its typically benign clinical course and the absence of marked proliferation activity in most cases [15]. For pure MA, genetic

studies have supported that the majority of pure MA lesions are non-neoplastic, based on no evidence of NF2 gene deletions [4, 5, 84]. However, a few cases have shown loss of heterozygosity on 22q12 and 9p21, raising the possibility of a neoplastic nature [3, 72]. For MA-M, meningioma-associated genetic alterations have always been found in both the MA and meningioma components, substantiating the neoplastic nature of MA-M [3–5]. Based on the aforementioned genetic studies, we postulate that pure MA and MA-M have distinct biological natures, with pure MA lesions more similar to non-neoplastic lesions and MA-M more similar to neoplastic lesions, which can result in different clinical features. Our findings supported this notion. Compared to pure MA, MA-M patients showed a lower seizure incidence. It is believed that the type of tumor plays an important role in epileptogenesis. Developmental lesions have a greater likelihood of causing seizures than neoplastic lesions because these lesions consist of well-differentiated cells that can release neurotransmitters or modulators with a role in epileptogenesis [85]. Therefore, the difference in pathogenesis might explain the discrepancy in seizure frequency between MA-M and pure MA. Moreover, MA-M cases showed a shorter symptom duration before

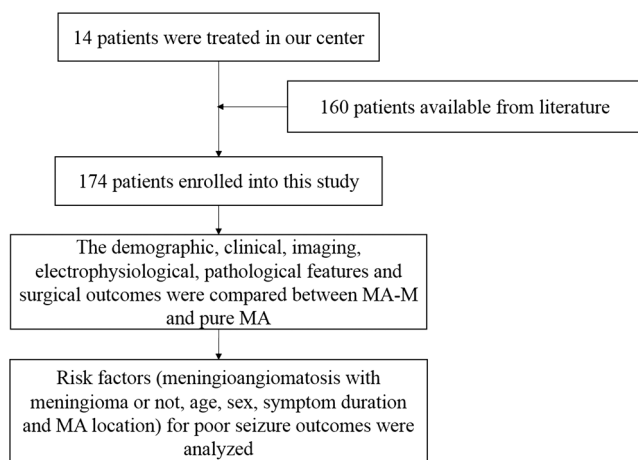


Fig. 8 Participant flow diagram

**Table 2** Comparison of MA-M and pure MA

| Parameter   | MA-M<br>(n=42) | Pure MA<br>(n=132) | P value         |
|---|----------------|--------------------|-----------------|
| Patient age (years)   | 17.4±2.5       | 20.0±1.3           | <i>p</i> =0.37  |
| Gender (M:F ratio)  | 4.2:1          | 1.6:1              | <i>p</i> =0.04  |
| Symptom duration (years)  | 2.8±0.8        | 5.2±0.6            | <i>p</i> =0.02  |
| Seizures incidence  | 53.6 %         | 89.3 %             | <i>p</i> <0.001 |
| Seizure types   |                |                    | <i>p</i> =0.38  |
| Partial seizures  | 61.5 %         | 52.4 %             |                 |
| Generalized and other types of seizures                               | 38.5 %         | 47.6 %             |                 |
| Location  |                |                    | <i>p</i> =0.18  |
| Frontal lobe  | 42.0 %         | 29.7 %             |                 |
| Temporal lobes  | 38.7 %         | 33.9 %             |                 |
| Other locations   | 19.3 %         | 36.4 %             |                 |
| Imaging   |                |                    |                 |
| Hypodense mass with calcification on CT                               | 53.3 %         | 51.3 %             | <i>p</i> =0.55  |
| Hyperintense on T2WI  | 60.0 %         | 71.7 %             | <i>p</i> =0.30  |
| Enhancement on MRI  | 84.6 %         | 75.0 %             | <i>p</i> =0.37  |
| Gyriform alteration   | 0              | 17                 |                 |
| Congruence on location of epileptiform discharges and lesions on EcoG | 60.0 %         | 46.2 %             | <i>p</i> =0.50  |
| Seizure free  | 92.3 %         | 85.7 %             | <i>p</i> =0.45  |

diagnosis. One potential explanation for this difference is that with a neoplastic biological behavior, MA-M caused progressively intolerable symptoms and eventually led to surgery at early stages. Given the neoplastic nature of MA-M, a more systematic and longer term follow-up might be favored in future clinical practice.

Surgery prompts favorable seizure outcomes for MA; however, seizures persisted in a proportion of patients, and more than 70 % patients required continuous antiepileptic drug treatment [1]. Although EcoG findings demonstrated a range of electrophysiological abnormalities, complex epileptogenicity did not show a correlation with poor seizure outcomes. We hypothesize that this finding was due to small sample bias, with only 18 cases available for EcoG data. We believe that it is still

**Table 3** Summary of histopathological features of the meningioma of the MA-M

| Histopathological subtypes of the meningioma in MA-M | Number in total (literature and our cases, n=42) |
|--|--|
| Transitional   | 23   |
| Fibroblastic   | 9  |
| Meningothelial                                       | 4  |
| Microcystic  | 2  |
| Atypical   | 2  |
| Psammomatous   | 1  |
| Metaplastic  | 1  |

**Table 4** Comparison of the immunohistochemical markers of MA-M and pure MA

| Markers (positive ratio) | MA-M      | Pure MA   | P value        |
|--------------------------|-----------|-----------|----------------|
| Vim                      | 88.9 %    | 95.7 %    | <i>p</i> =0.83 |
| CD34                     | 78.6 %    | 63.2 %    | <i>p</i> =0.57 |
| EMA                      | 66.7 %    | 51.7 %    | <i>p</i> =0.31 |
| GFAP                     | 50.0 %    | 50.0 %    | <i>p</i> =1.00 |
| S-100                    | 54.5 %    | 47.1 %    | <i>p</i> =1.00 |
| Ki-67                    | 1.2±0.3 % | 1.4±0.5 % | <i>p</i> =0.67 |

necessary to perform EcoG covering MA and the adjacent cortex during surgery. Except for lesions located in the temporal lobe, no other factors emerged as a determinant of seizure outcomes, including age, sex, MA size, duration of symptoms and the presence or absence of meningioma (Table 5). A potential explanation may be that early onset seizures due to temporal sporadic MA kindle the hippocampus and finally result in dual pathology [86]. Dual pathology refers to the coexistence of hippocampal sclerosis and an extrahippocampal lesion, in which the lesion most commonly involves the temporal lobe [87]. Moreover, the hippocampus is more vulnerable in early childhood, and the dual pathology was almost exclusively found in patients whose lesions were congenital or occurred early in life and have epileptogenic potential [88]. Sporadic MA perfectly matches such features of extrahippocampal lesions with dual pathology, and temporal MA with hippocampal sclerosis has been reported [1]. However, because subtle microscopic malformations of the hippocampus may not show abnormalities on MRI, underlying hippocampal sclerosis may have been underestimated. Hence, temporal lobe MA should draw closer attention in future clinical practice. Volumetric MRI, stereotactic EEG and EcoG during surgery could be valuable in identifying subtle hippocampal sclerosis in temporal sporadic MA.

In conclusion, compared to pure MA, MA-M presents with a more prominent male predilection, lower seizure incidence and shorter symptom duration. Such clinical differences, combined with previous genetic findings, substantiate the possibility that MA-M is more similar to a neoplasm and that pure

**Table 5** Odds ratios of potential risk factors for poor seizure outcomes (Engel II/III/IV) in sporadic MA patients undergoing surgery (n=87)

| Risk factors         | <i>p</i> -value | OR    | 95 % CI      |
|----------------------|-----------------|-------|--------------|
| Sex                  | 0.137           | 0.387 | 0.111–1.354  |
| Age                  | 0.435           | 1.023 | 0.967–1.082  |
| Duration of symptoms | 0.754           | 1.001 | 0.993–1.010  |
| MA-M                 | 0.998           | 0.000 | 0.000–       |
| Temporal location    | 0.022           | 4.435 | 1.238–15.886 |

CI confidence interval, OR odds ratio

MA is more similar to a non-neoplastic lesion. Gyriform alterations on imaging can help to distinguish pure MA from MA-M. In addition, MA-M should be differentiated from malignant childhood meningioma, and a long-term follow-up is required due to its potential neoplastic nature. Given the unsatisfactory seizure outcomes of MA located in the temporal lobe, which can be associated with dual pathology, recognition of underlying hippocampus sclerosis and the performing of complete resection are critical for temporal sporadic MA.

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## Comments

Zhang and colleagues analyzed the clinical and morphological data and treatment results of 14 patients affected by a rare cerebral disease characterized by a leptomeningeal meningotheelial proliferation called “meningioangiomas.” Such meningeal abnormality was identified by the authors in seven cases as an isolated disease and in seven further cases associated with a meningioma. The authors also analyzed 160 similar cases reported in the literature to date. According to the results presented in the study, meningioangiomas should be considered a non-neoplastic lesion that may, however, be associated with epilepsy. Nonetheless, the contextual presence of other epileptogenic foci (i.e., mesiotemporal sclerosis) is common, and some of the lesions may be associated with gyriform alteration or enhancement. Accurate epilepsy preoperative assessment is therefore mandatory to gain treatment success. Meningiomas associated with meningioangiomas are typical neoplastic lesions, requiring standard follow-up. The study provides interesting information on the histopathological features of these lesions and the differences from meningiomas. On such bases, it can be suggested that when meningioangiomas is associated with meningiomas, these tumors could arise from the non-neoplastic meningioangiomas lesions by additional genetic alterations, which may confer a greater proliferation potential.

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