CASE REPORT

J Korean Neurosurg Soc 38: 54-60, 2005

# Three Cases of Intracranial Clear Cell Meningioma

Yeon-Seong Kim, M.D.,[1] In-Young Kim, M.D.,[1] Shin Jung, M.D.,[1] Min-Cheol Lee, M.D.[2]

*Department of Neurosurgery,[1] Pathology,[2] Chonnam National University Hospital & Medical School, Gwangju, Korea*

The clear cell meningioma(CCM) is a rare and recently described as a histologic variant of meningioma. It has been identified and included in new World Health Organization(WHO) classification of the Central Nervous System(CNS) tumors recently. The CCMs are histologically characterized by sheets of spindled to polygonal cells with clear cytoplasm, which is the expression of high glycogen concentration. The CCMs occur in younger patients and usually are located in the spinal canal and posterior fossa. The most interesting aspect of CCM is the high recurrence rate and aggressiveness. Poor outcome has been shown in intracranial and spinal tumor location, but the indicators that predict outcome have not been established. Until now 22 intracranial CCM cases had been reported in English literature and 3 cases in Korean. The authors report two cases of CCM located at cerebral convexity and one at cavernous sinus those were totally removed (Simpson Grade I~II) by subfrontal, frontal and orbitocranial approaches. The clinical, radiological, histopathological, and neurosurgical features of these cases are discussed with the relevant literatures.

**KEY WORDS :** Meningioma · Clear cell · Aggressive behavior · Glycogen.

## Introduction

The current World Health Organization Classification of Tumors of the Central Nervous System (WHO 2000) grouped meningiomas by likelihood of recurrence and grade (I~III), and clear cell meningioma(CCM) is described as a variant of the meningioma[4,9,13,15]. The subtypes included as grade II (meningiomas as low or uncertain malignant potential or borderline malignancy) are clear cell (intracranial), chordoid, and atypical meningiomas[9]. CCMs are histologically characterized by whorled, syncytial architecture and sheets of spindled-to-polygonal, bland-appearing nuclei with clear cytoplasm[6,9]. The spinal canal and the posterior fossa (especially, cerebello-pontine angle) are the most common sites of CCMs[4,14-16,25]. CCM occurs at a young age and, although it has benign histologic nature, recurrence and metastasis are very common. Furthermore, CCM has more aggressive behavior than other subtypes[14,19]. To the best of our knowledge, 22 cases of intracranial CCM have been previously reported in the recent English literature and 3 cases in Korean literature[1,3-5,7,8,10,12,13,15,16,18-20,22,23,25].

In this report, we present additional three cases of CCM, one arising from the cavernous sinus in a middle-aged male and the others from the cerebral convexity in old-aged male and female with review of the previous literature.

## Case Report

### Case 1

A 63-year-old male was admitted with complaints of dysphasia and memory disturbance. He had suffered from liver cirrhosis for 21years and got an endoscopic varix ligation due to varix bleeding 1year ago. Physical examination revealed abdominal distension and neurologic examination revealed slight memory disturbance, acalculia and slight right hemiparesis. Laboratory examination revealed low platelet count (57,000/mm$^3$), low hemoglobin (9.2g/dL) and slight high AST (49U/L). Magnetic resonance imaging(MRI) of brain showed a intracranial mass attached to left temporoparietal convexity margin of 5.5×4.5cm in size with peritumoral edema and midline shift (Fig. 1A, B). The mass was heterogeneously enhanced after intravenous contrast injection (Fig. 1C). Antiedema medication was started and medical consultation for liver cirrhosis was done. After correction of laboratory abnormalities, an elective left temporo-parietal craniotomy was performed and gross total removal of the tumor mass and dura mater

• Received : November 26, 2004 • Accepted : February 11, 2005

• Address for reprints : Shin Jung, M.D., Department of Neurosurgery, Chonnam National University Hospital & Medical School, 160 Ilsim-ri, Hwasun-eup, Hwasun-gun, Gwangju 519-809, Korea Tel : +82.61-379-7666, Fax : +82.61-379-7673, E-mail : sjung@chonnam.ac.kr

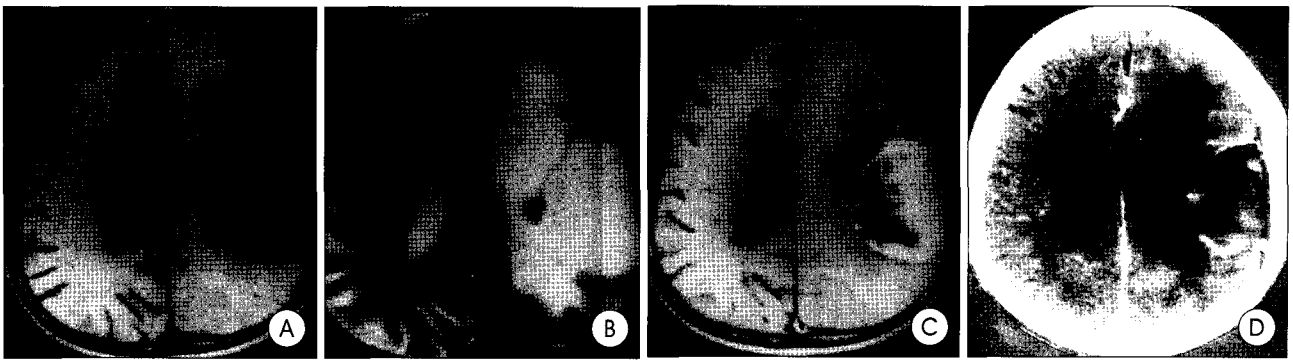

**Fig. 1.** Magnetic resonance imaging showing a intracranial mass attached to the left temporoparietal convexity margin of 5.5×4.5cm in size with peritumoral edema and mid line shift (A and B). The mass heterogeneously enhancing after intravenous contrast injection (C). Gross total removal of the tumor mass and dura mater (Simpson grade I) is achieved with meticulous bleeding control (D).

(Simpson grade I) was achieved with meticulous bleeding control (Fig. 1D). Microscopically, the tumor was consisted of polygonal cells with a clear cytoplasm (Fig. 2A). The immunohistomchemical studies were positive for epithelial membrane antigen (EMA) and Vimentin, but negative for S-100 and glial fibrillary acidic protein (GFAP) (Fig. 2B). The Ki-67 labelling index showed 1% and the histopathological diagnosis was CCM. In second postoperative day, the patient experienced an episode of seizure and sudden mental deepening but recovered immediately. After 4years of follow-up he has been in good condition, and neurological examination revealed no deficits except for a intermittent paresthesia of right hand and foot.

## Case 2

A 63-year-old female was admitted with motor dysphasia, memory disturbance and confusion for 3months. Neurologic examination revealed the mental confusion, left anosmia, acalculia and urinary incontinence. MRI showed a large (3.5×3.0cm sized) homogeneously contrast enhancing mass which filled the left frontal lobe. The mass was attached to the left frontal convexity and a dural tail, enormous peritumoral edema and midline shift were observed (Fig. 3A, B, C). Mannitolization and steroid therapy were started and an elective left frontal craniotomy was performed and a total excision of the mass and the originating dura mater (Simpson grade I) was done (Fig. 3D). Microscopically, the tumor was composed of oval to polygonal cells. There was abundant cytoplasm with an absence of cell borders. The cytoplasm varied in appearance from clear to foamy (Fig. 4A). The immunohistochemical studies were positive for CD34, Factor Ⅷ, Vimentin, S-100 (focally) and EMA, but negative for GFAP and Ki-67 labelling (Fig. 4B). The final diagnosis was CCM. She was recovered within 1week without neurological deficit and has received no chemo- or radiation therapy. One year later, a MRI scan was performed but showed no evidence of a recurrence. At 2-year follow-up, computerized tomography(CT) scan showed the same result. Currently, she is free from symptoms and neurologic examinations.

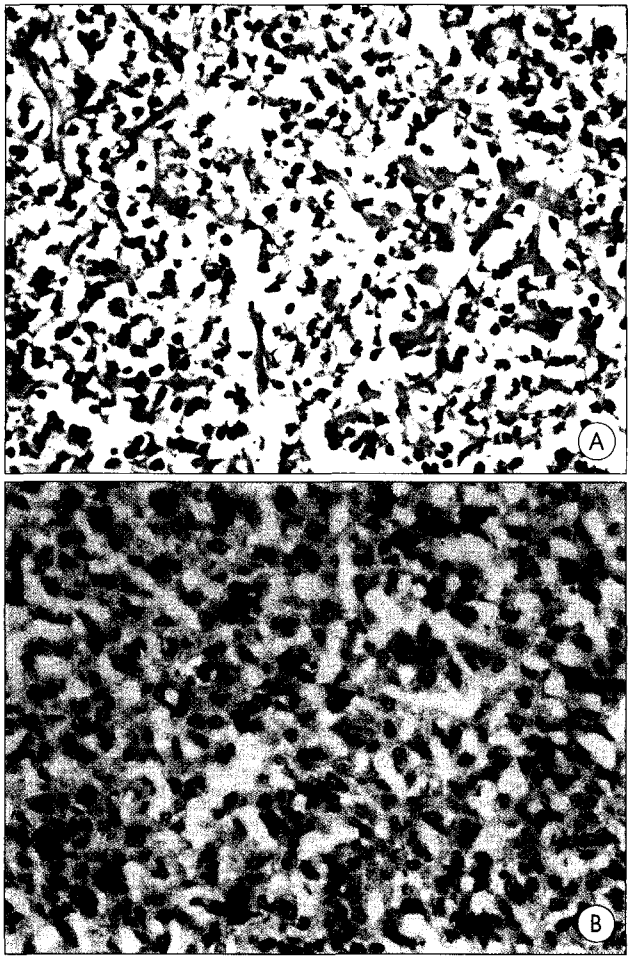

**Fig. 2.** Neuropathological and immunohistochemical features of case #1. Photomicrograph showing polygonal bland meningeal cells with clear cytoplasms (A. H & E,×400). The tumor cells are positive for epithelial membrane antigen (B×400).

## Case 3

A 42-year-old male was referred with dizziness and a history

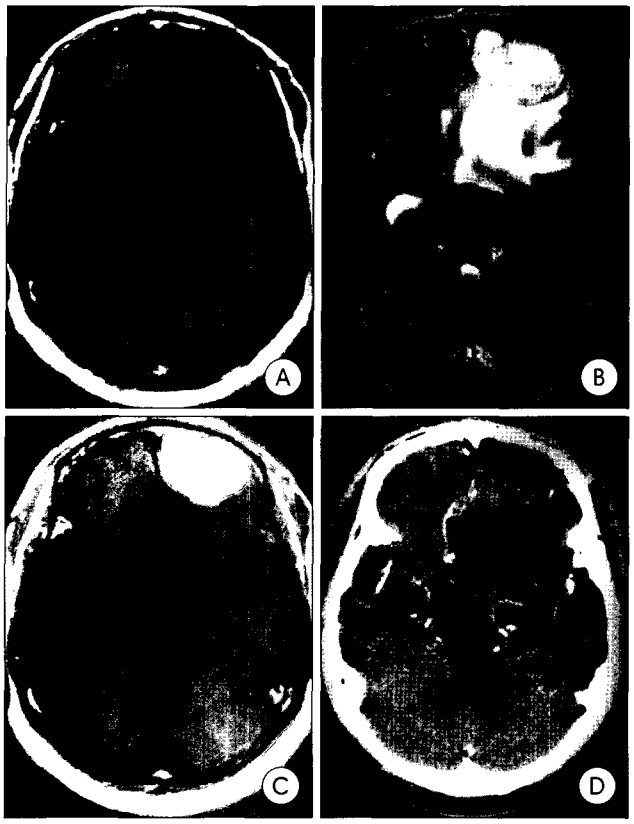


**Fig. 3.** Magnetic resonance imaging showing a large (3.5×3.0cm sized) homogeneously contrast enhancing mass which filled the left frontal lobe. The mass is attached to the left frontal convexity and a dural tail, enormous peritumoral edema and midline shift are observed (A~C). Elective left frontal craniotomy is performed and a total excision of the mass and the originating dura mater (Simpson grade I) was done (D).

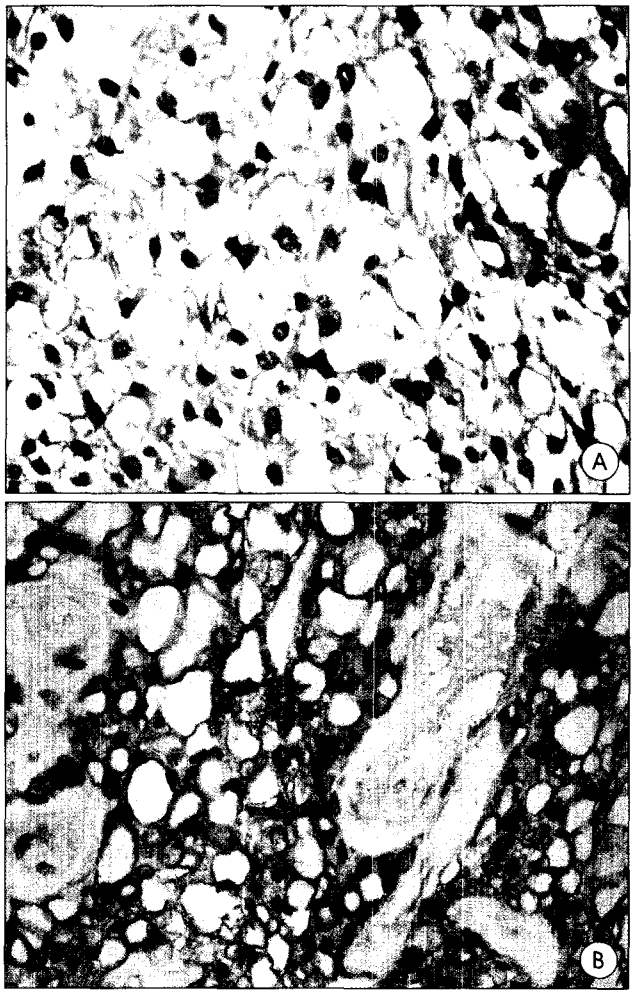


**Fig. 4.** Neuropathological and immunohistochemical features of case #2. The nuclei are round to oval. There is abundant cytoplasm with an absence of cell borders. The cytoplasm varies in appearance from clear to foamy (A. H & E, ×400). The tumor cells are positive for epithelial membrane antigen (B×400).

of seizure on that day. Neurologic examination showed drowsy mental status and laboratory examination revealed high AST (189U/L), ALT (55U/L) and gamma-GTP (661U/L). CT images revealed 2.5×2.5cm sized, contrast enhancing mass at left cavernous sinus with well demarkable margin. MRI indicated a 2.5×2.5cm sized, heterogeneously contrast enhancing mass which attached to left cavernous sinus extending to left temporal lobe without peritumoral edema (Fig. 5A, B, C). Digital subtraction angiography(DSA) showed normal appearance of left cavernous sinus and both carotid arteries. Anticonvulsant medication and liver protecting therapy were started. After correction of laboratory abnormalities, an elective left frontotemporal craniotomy with orbital osteotomy was done. The tumor mass was originated from the posterior portion of left cavernous sinus and penetrated through adjacent dura mater with intradural growth. Total removal of the tumor mass (Simpson grade II) was performed (Fig. 5D). Microscopically, the tumor showed proliferation of polygonal cells with clear cytoplasm (Fig. 6A). The immunohistomchemical studies were positive for Vimentin, but negative for EMA, GFAP, S-100, CD34 and CK7 (Fig. 6B). The Ki-67 labelling index showed 3% and the histopatho-logical diagnosis was CCM. No adjuvant therapy was done and at the time of discharge, he had intermittent dizziness with no neurologic deficits or additional seizure.

## Discussion

According to the World Health Organization Classification of Tumors of the Central Nervous System(WHO 2000), CCM is a benign variant of meningioma as grade II but its clinical behavior has not been fully characterized because there are only few reports in the literature[6,9]. Since it may recur promptly, spread locally, and even metastasize, it is clinically aggressive despite its bland histologic appearance[13,16,19]. So CCM should be differentiated from other meningiomas of the central nervous system. CCM has a rare incidence and Zorlud-

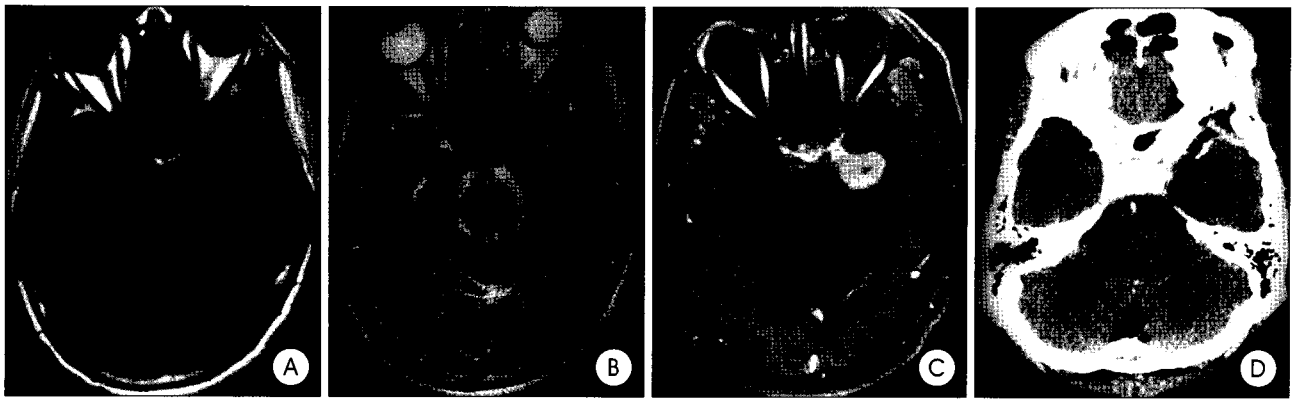


**Fig. 5.** Magnetic resonance image indicating a 2.5×2.5cm sized, heterogeneously contrast enhancing mass which attached to left cavernous sinus extending to left temporal lobe without peritumoral edema (A~C). Elective left frontotemporal craniotomy with orbital osteotomy is done. The tumor mass is originated from the posterior portion of left cavernous sinus and penetrated through adjacent dura mater with intradural growth. Total removal of the tumor mass (Simpson grade II) is performed (D).

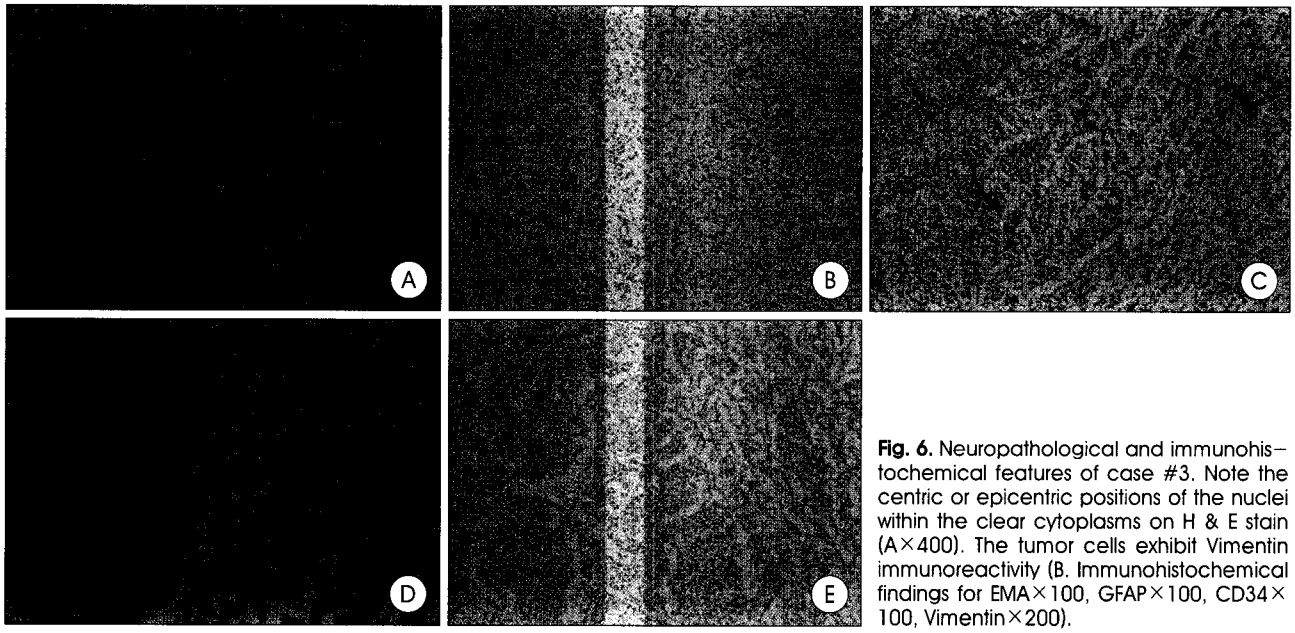


**Fig. 6.** Neuropathological and immunohistochemical features of case #3. Note the centric or epicentric positions of the nuclei within the clear cytoplasms on H & E stain (A×400). The tumor cells exhibit Vimentin immunoreactivity (B. Immunohistochemical findings for EMA×100, GFAP×100, CD34×100, Vimentin×200).

emir et al. represented that CCMs consisted of 0.2% of all meningiomas[25].

In the literature data, the clinical characteristics of CCM are reported to be somewhat different from those of ordinary meningioma subtypes[1,3-5,7,10,13,15,16,19,20,22,25]. Firstly, while ordinary meningiomas are likely to occur during the fifth and sixth decades with female predominance, CCM occurs at any age and without gender predilection[1,6,7,9-12,14,15,21,25]. Secondly, the average age at the time of surgery is reported to be younger in CCM cases than in those of ordinary meningiomas[1,6,9,11,21,25]. Lee et al. reported that a mean age of CCMs was 29.8years (22months~67years) and Zorludemir et al. also reported 29years (9years~82years)[13,25]. Thirdly, spinal location is more frequent (spinal, 55.5%; posterior fossa, 22.3%; supratentorial, 16.7%; and skull base, 5.5%)[4,14,16,25]. Fourthly, local recurrence rate and leptomeningeal metastasis (or multiple sites) are common in comparison with ordinary meningiomas[1,4,5,13,15,24,25]. Zorludemir et al. reported that CCMs, in particular those located intracranially, behaved in an 'inordinately aggressive' fashion be lying their bland histological appearance[25]. In their 13-case series, 61% of patients experienced one or more recurrences, 15% had local discontinuous spread, 8% had cerebrospinal fluid-borne cranial to spinal metastases, and 23% died of disease. Factors such as mitotic activity, ploidy status and proliferating cell nuclear antigen index were not predictive of recurrence or clinical outcome, although the MIB-1 proliferation index was 'appreciably higher' in recurring tumors[22,25]. According to Jellinger and Slowik, the recurrence rates in cases ordinary spinal and intracranial meningioma are 4.8% and 14.2%, respectively[6]. In contrast, the recurrence rates in spinal and intracranial CCMs are 80% and 46%, respectively, with an overall recurrence rate of 60.9%[13]. The most likely reason for multiple recurrence is that the

extensive infiltrative growth pattern of the tumor hinders complete microscopic surgical resection[16,24]. Since cellular anaplasia was not present, and during the first operation and all subsequent recurrences the growth fraction was low, histologic parameters were not predictive of recurrence[16,24]. Fifthly, nondural-based tumors are often found in CCMs (11.1%)[14,25].

Cytopathologically CCMs have a whorled syncytial architecture and spindled to polygonal, bland-appearing nuclei with clear, glycogen-rich (PAS-positive, diastase-labile) cytoplasm, variable amounts of hyaline connective tissue intermingled with tumor cells being also a typical morphological feature[3,5,14,15,24]. In contrast to the more classic meningioma, the clear-cell variety has vacuolated cytoplasm[5,7].

Immunohistochemistry may be required to make the correct diagnosis[25]. Immunohistochemically, CCMs show reactivity for vimentin and EMA and do not show reactivity for S-100 protein, GFAP, and cytokeratin[10,13,25]. Other intracranial masses including oligodendroglioma or clear cell epedymoma are not heavily glycogenated, may be GFAP-immunoreactive (a feature foreign to meningioma), and do not exhibit the membranous EMA expression of meningioma[7,10]. In our case #1 and #2, immunohistochemical stain for EMA and Vimentin were positive and that of GFAP was negative. But S-100 protein was positive focally in case #2. Exclusively, there was no reactivity for EMA in case #3, but positive reactivity for vimentin (Table 1).

**Table 1.** Immunohistochemical findings of 3 patients in the current study

| Patient | EMA | Ki-67 LI | GFAP | Vimentin | S-100 |
|---|---|---|---|---|---|
| #1 63/M | (+) | <1% | (-) | (+) | (-) |
| #2 63/F | (+) | (-) | (-) | (+) | Focally (+) |
| #3 42/M | (-) | 3% | (-) | (+) | (-) |

EMA : Epithelial membrane antigen LI : Labelling index GFAP : Glial fibrillary acidic protein

CCM is histologically unique but should be differentiated from other similar clear cell tumors on the central nervous system, such as metastatic renal cell carcinoma, hemangioblastoma, oligodendroglioma, germinoma, seminoma, pleomorphic xanthoastrocytoma, lipid-rich glioblastoma, and clear cell ependymoma[1,3,7,12,13,19].

The MRI features of CCM are not much different from the those of ordinary meningiomas[12]. The tumor homogeneously enhances after gadolinium injection[13,24]. But, in our case #1 and #3, contrast enhancing patterns are somewhat heterogeneous, especially in central areas of the tumors.

Lee et al. suggested that a extensive contrast enhancement of the entire cisternal spaces on MRI suggested a extensive CSF seeding[13]. They also reported that histologic parameters

**Table 2.** Summary of all patients with intracranial CCMs reported in the English literature

| Authors (Year) | Case No. | Age/Sex | Location | Treatment | Follow-up results | Adjuvant therapy |
|---|---|---|---|---|---|---|
| Shiraishi (1991) | 1 | 49/F | Frontal lobe | GTR | No recurrence | None |
| Kakita et al (1995) | 1 | 64/F | Frontal lobe | GTR | Not known | Not known |
| Zorludemir et al (1995) | 7 | 32/F | CPA | GTR | Recurrence (+) | GTR + RS |
| | | 82/M | Frontotemporal lobe | STR | Dead (1 month) | None |
| | | 11/M | Frontal lobe | GTR | Recurrence (+) | GTR + RT |
| | | 16/M | CPA | GTR | Recurrence (+) | GTR + RT |
| | | 12/F | Tentorium-clinoid | GTR | No recurrence | None |
| | | 34/M | Foramen magnum | GTR | Recurrence (+) | GKRS (twice) |
| | | 21/F | CPA | STR + RT | Recurrence (+) | STR + RT |
| Shih et al (1996) | 1 | 12/M | CPA | GTR | No recurrence | None |
| Imlay et al (1998) | 1 | 21/F | Posterior fossa | PR + RT | Metastasis (+)* | GTR* |
| Primentel et al (1998) | 3 | 24/F | Tentorium-clinoid | STR | Recurrence (+) | Not known |
| | | 61/M | Convexity | GTR | No recurrence | Not known |
| | | 19/M | Basal skull | STR | Recurrence (+) | Not known |
| Teo et al (1998) | 1 | 2/F | Brain stem | STR | Not known | Refusal by patient |
| Lee et al (2000) | 1 | 17/M | Parietal lobe | GTR | LM seeding (+)* | GTR + RT* |
| Heth et al (2000) | 1 | 31/F | Foramen magnum | STR | Regrowth (+) | Not known |
| Kuzeyli et al (2001) | 2 | 52/F | Occipital lobe | TR | No recurrence | None |
| | | 50/M | Temporal lobe | STR | Dead (45 days) | None |
| Yu et al (2002) | 1 | 17/F | CPA | PR | Not known | Not known |
| Carlotti et al (2003) | 2 | 28/F | Fourth ventricle | GTR | No recurrence | None |
| | | 23/F | Fourth ventricle | TR | No recurrence | None |
| Present report (2004) | 3 | 63/M | Convexity | TR | No recurrence | None |
| | | 63/F | Convexity | TR | No recurrence | None |
| | | 42/M | Cavernous sinus | TR | Follow-up (3 month) | None |

CPA, cerebellopontine angle; GTR, gross total resection; TR, total resection; STR, subtotal resection; PR, partial resection; RS, radiosurgery; RT, radiation therapy
* in spine

did not predict recurrence, because cellular anaplasia was lacking and growth fraction was low in most CCM and that neuroimaging findings of localized leptomeningeal enhancement associated with meningioma might be the predictable findings of CCM.

22cases of intracranial CCMs had been reported in the English language literature (Table 2), 2cases of all were totally (Simpson grade I and II), 11 cases gross totally (Simpson grade III), 7cases subtotally (Simpson grade IV), and the remaining 2cases were partially resected (Simpson grade V)[1,3-5,7,10,12,13,15,16,19,20,22,25]. In 11 patients resected gross totally, 4patients experienced tumor recurrences (36.4%), one patient revealed leptomeningeal seeding and 5patients had been disease-free state. In 7patients resected subtotally, 4patients revealed tumor recurrences (57.1%) and 2patient died at postoperative 1month and 45days, respectively. The progression-free states were not seen in all subtotal and partial resected patients.

We found 8 cases of recurrences, one case of leptomeningeal seeding and one case of distant metastasis in 22 intracranial CCMs described in the English literature. So the overall recurrence rate was 36.4% and the metastasis rate was 9.1%. But in our cases, there was no recurrence and no metastasis.

The goal of the treatment in CCM is to achieve radical surgical removal as much as possible[1,4,12,13,24,25]. According to the literatures and all our cases, there was no recurrence and no metastasis in the total resected patients. While meningiomas have traditionally been considered radioresistant, radiation therapy and radiosurgery might be important treatment options for patients who are not good candidates for reoperation in recurring cases and surgically inaccessible meningiomas[12,22]. Chang and Adler reported the value of linear accelerator(LINAC) radiosurgery in the treatment of cranial base meningiomas where tumor stabilization was achieved in 69% of patients and shrinkage in 29%[2]. Chemotherapeutic agents have not been promising in the treatment of meningiomas, although there has been suggestion that hormonal therapy may be of some benefit[17].

In our cases the diagnosis was based on histologic characteristics and immunohistochemical stains and corroborated by imaging and the surgical findings. The symptom duration of them was mean 1.4months (0.1~3months). The mean follow up period of all our patients is 33months (3~57months). The patient #1 and #2 have had long-term follow-up (4.5years and 3years) which is longer than the mean time for recurrence estimated in the literature (5.3months, ranged 4~23months) with no recurrence and no metastasis[1,4,12,25]. But the follow-up period of the patient #3 is 3months, it is obvious that a longer follow-up is necessary for this case. When compared with other meningioma operations, neither macroscopic nor surgical differences could be detected in all our operations.

Simpson grade I operations were achieved in our patient #1 and #2 but Simpson grade II operation was done in patient #3 because the tumor originated from the cavernous sinus. There were no specific surgical morbidity and mortality in all cases with no recurrence evidence.

## Conclusion

Although CCM is a rare subtype, it should be considered in the differential diagnosis of a central nervous system mass with neuroimaging findings, pathological and immunohistochemical features of meningioma, especially in young patients because it is characterized by aggressive nature and high rates of recurrence including either metastasis or CSF seeding. Clinical outcome appears to be relatively unassociated with conventional histologic indicators of aggressive behavior.

Our report could suggest that CCMs resected totally have low-malignant potential and have no recurrence and no metastasis. The reports of other authors showed that CCMs removed subtotally or partially had higher rate of recurrences and local metastases. So the aggressiveness of the CCMs are considered to be related to not only the tumor-related factors but also the surgery-related factors. Ultimately, CCM must be followed up carefully because of its aggressive behavior and the high risk of recurrence, even when benign histologic results are obtained.

### References


1. Carlotti CG, Neder L, Colli BO, Santos MB, Garcia AS, Elias J, et al : Clear Cell Meningioma of the Fourth Ventricle. **Am J Surg Pathol 27** : 131-135, 2003
2. Chang SD, Adler JR : Treatment of cranial base meningiomas with linear accelerator radiosurgery : **Neurosurgery 41** : 1019-1027, 1997
3. Gökden M, Roth KA, Carroll SL, Wick MR, Schmidt RE : Clear Cell Neoplasms and Pseudoneoplastic Lesions of the Central Nervous System. **Semin Diagn Pathol 14** : 253-269, 1997
4. Heth JA, Kirby P, Menezes AH : Intraspinal familial clear cell meningioma in a mother and child. Case report : **J Neurosurg (Spine) 93** : 317-321, 2000
5. Imlay SP, Snider TE, Raab SS : Clear Cell meningioma: diagnosis by fine-needle aspiration biopsy. **Diagn Cytopathol 18** : 131-136, 1998
6. Jellinger K, Slowik F : Histological subtypes and prognostic problems in meningiomas : **J Neurol 208** : 279-298, 1975
7. Kakita A, Takahashi H, Fusejima T, Konno K, Nakazawa T, Aoki K, et al : Clear cell variants of intracranial tumors : meningioma and ependymoma. **Noshuyo Byori 12** : 111-116, 1995
8. Kim DH, Kim Y, Park CO, Ha YS : A Case of Clear Cell Meningioma : A Case Report. **J Korean Neurosurg Soc 26** : 589-595, 1997
9. Kleihues P, Louis DN, Scheithauer BW, Rorke LB, Reifenberger G, Burger PC, et al : The WHO classification of tumors of the nervous system : **J Neuropathol Exp Neurol 61** : 215-225, 2002
10. Kubota T, Sato K, Kabuto M, Hasegawa M, Kitai R, Nakagawa T, et al : Clear cell (glycogen-rich) meningioma wit special reference to spherical collagen deposits. **Noshuyo Byori 12** : 53-60, 1995
11. Kurland LT, Schoenberg BS, Annegers JF, Okazaki H, Molgaard CA : The incidence of primary intracranial neoplasms in Rochester, Minnesota, 1935-1977 : **Ann N Y Acad Sci 381** : 1-16, 1982
12. Kuzeyli K, Cakir E, Usul H, Karaarslan G, Reis AK, Temiz C, et al : Clear cell meningioma : case report and literature review. **J Clin**

Neurosci 10 : 264-266, 2003
13. Lee W, Chang KH, Choe G, Chi JG, Chung CK, Kim IH, et al : MR imaging features of clear-cell meningioma with diffuse leptomeningeal seeding : **AJNR 21** : 130-132, 2000
14. Matsui H, Kanamori M, Abe Y, Sakai T, Wakaki K : Multifocal clear-cell meningioma in the spine : A Case Report : **Neurosurg Rev 21** : 171-173, 1998
15. Pimental J, Fernandes A, Pinto AE, Fonseca I, Nunes JF, Antunes JL : Clear cell meningioma variant and clinical aggressiveness. **Clin Neuropathol 17** : 141-146, 1998
16. Prinz M, Patt S, Mitrovics T, Cervos-Navarro J : Clear-cell meningioma : report of a spinal case : **Gen Diagn Pathol 141** : 261-267, 1996
17. Schrell UMH, Fahlbusch R : Hormonal manipulation of cerebral meningiomas; in Al-Mefty(ed) : **Meningiomas.** New York, Raven Press : 273-283, 1991
18. Seo EK, Lee KS : Clear Cell Meningioma : Case Report. **J Korean Neurosurg Soc 27** : 285-288, 2002
19. Shih DF, Wang JS, Pan RG, Tseng HH : Clear cell meningioma : a case report. **Chung Hua I Hsueh Tsa Chih 57** : 452-456, 1996
20. Shiraishi K : Glycogen-rich meningioma. Case report and short review : **Neurosurg Rev 14** : 61-64, 1991
21. Sutherland GR, Florell R, Louw D, Choi NW, Sima AA : Epidemiology of primary intracranial neoplasms in Manitoba, Canada : **Can J Neurol Sci 14** : 586-592, 1987
22. Teo JG, Goh KY, Rosenblum MK, Muszynski CA, Epstein FJ : Intraparenchymal clear cell meningioma of the brainstem in a 2-year-old child. Case report and literature review. **Pediatr Neurosurg 28** : 27-30, 1998
23. Yi JS, Park SC, Park HK, Cho KK, Park YS, Choi CR : Clear Cell Meningioma : Case Report. **J Korean Neurosurg Soc 25** : 2331-2335, 1996
24. Yu KB, Lim MK, Kim HJ, Suh CH, Park HC, Kim EY, et al : Clear-cell meningioma : CT and MR imaging findings in two cases involving the spinal canal and cerebellopontine angle. **Korean J Radiol 3** : 125-129, 2002
25. Zorludemir S, Scheithauer BW, Hirose T, Houten CV, Miller G, Meyer FB : Clear Cell Meningioma. A Clinicopathologic Study of a Potentially Aggressive Variant of Meningioma. **Am J Surg Pathol 19** : 493-505, 1995