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# The Radiologic Features of Cystic versus Noncystic Glioblastoma Multiforme as Significant Prognostic Factors[1]

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**Purpose:** The purpose of this study was to determine the preoperative radiological characteristic and survival differences of glioblastoma multiforme (GBM) with and without cysts.

**Materials and Methods:** Twenty-one GBMs were collected retrospectively; these tumors were pathologic confirmed as GBM. Based on the preoperative MR imaging, we compared the cystic GBMs with the noncystic GBMs according to the the tumor size, the tumor interface, the tumor wall thickness and peritumoral edema.

**Results:** Seven cases were classified as cystic GBMs and fourteen were noncystic GBMs. The cystic GBMs had a well-defined tumor interface, a less than 2 cm thickness of the tumor wall and less than 40 $cm^3$ thick peritumoral edema as compared to that of the noncystic GBMs. There was a statistically significant difference in age between the patients with cystic tumors and those with noncystic tumors. For the patients with cystic GBMs and noncystic GBMs, median survival time after surgery was 43.8 months and 12.5 months, respectively.

**Conclusion:** The cystic GBMs had a well-defined tumor interface, a thin wall and minimal edema, as compared with that of the noncystic GBMs. The patients with cystic GBMs were significantly younger and they had more favorable survival outcomes than did the patients with noncystic GBMs.

**Index words :** Glioblastoma
Brain Neoplasms
Magnetic Resonance Imaging

Glioblastoma multiforme (GBM) is the most malignant form of astrocytoma. GBMs are histologically characterized by the glial cells with abnormal morphologies, giant cells, necrotic areas, vascular hyperplasia and a large number of cells with mitotic figures. Despite the advancements in surgery, adjuvant chemotherapy and radiotherapy, the prognosis of these patients remains poor and the disease undergoes fairly rapid progression to a fatal outcome. The several papers have reported that the median patient survival after diagnosis is approximately 1 year. Chandler et al found 5% survived until 5 years, and Salford et al report 0.5% survived at 10 years (1-3). Several well known variables affecting the prognosis of these patients with GBM have been investigated, including the patients' age, the Karnofsky

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performance score (KPS), the extent of surgical removal, reoperation for recurrent tumors, neurologic symptoms and the duration of disease (4–8). The reported image prognosis factors are the preoperative MR imaging characteristics of the tumor and the associated grade of necrosis, with edema, enhancement and necrosis being the most important histological prognostic factors (4, 8). Hammoud et al demonstrated that patients with little or no necrosis survive longer than those patients with profuse necrosis seen on the preoperative MRI scans, and Maldaun et al recently revealed that the patients with GBMs that included cystic components had a more favorable survival outcome than those patients without cysts (4, 9). Several authors have mentioned the clinical differences between the cystic and necrotic changes in GBMs; however, it is difficult to distinguish cystic change from necrotic changes within tumors and there is little published data related to the different MRI features between cystic GBMs and noncystic GBMs. The purpose of this study was to determine the preoperative radiological characteristics and survival differences of GBMs with and without cysts.

## Materials and Methods

### *Patient Population*

We retrospectively reviewed the medical records and diagnostic images to obtain information on the study patients. We identified 24 patients who were diagnosed with GBMs and who were treated between January 2000 and March 2009. A review of the patients' charts was conducted to obtain information on their ages, gender, KPS and treatment. The patients underwent total or subtotal mass removal and stereotactic biopsy of a histologically confirmed supratentorial glioblastoma according the World Health Organization (WHO) classification. Preoperative MRI images had been obtained for all the patients. The three patients with incomplete or poor quality preoperative imaging data were excluded from the analysis; the remaining 21 patients made up the study population: there were 12 males and 9 females with a mean age of 46 years (range: 15–80).

### *Imaging Protocol*

All the MRI images were obtained with a 1.5-T MR imaging unit, Magnetom Vision (Siemens Medical Systems, Erlangen, Germany). The MRI protocol included the following pulse sequences: the axial T1-weighted and axial fast spin-echo T2-weighted MR images. The contrast agent-enhanced T1-weighted images were obtained in all patients after intravenous injection of 0.1 mmol per kilogram of body weight gadolinium-based contrast agent (Gadoterate meglumine, Dotarem, Guerbet, Paris, France; or gadodiamide, Omniscan, GE Healthcare, Belfast, Ireland).

### *Definition of Variables*

The preoperative MR images of all the patients were retrospectively reviewed and several imaging features were identified. Our criteria for the radiologic diagnosis of a cyst on an MRI scan were the appearance of an area of well defined, spherical or ovoid lesions with an even lining. Necrosis was defined as an area of irregular bordered components within the tumors, which had decreased signal intensity on a T1-weighted MRI scan and this was surrounded by a contrast enhanced tumor nodule (4, 10, 11). In all cases, we regarded cystic tumor as a mass contained a cystic component, based on the preoperative MR imaging study. The cystic portion of the tumor showed an ovoid or spherical shape, a well defined interface, homogeneous hypointensity, an enhanced even lining and a thin, smooth wall on the Gd-enhanced T1-weighted MR images (Fig. 1). On the other hand, noncystic GBM showed an irregular shape, poor interface and an enhanced thick, uneven wall on the Gd-enhanced T1-weighted MR images (Fig. 2).

The tumor volume on the MRI appears as a maximal enhanced area from the post-contrast T1-weighted image with using 3D volume reconstruction. Tumor interface was defined as the interface between the tumor and peritumoral brain parenchyma and this was classified as a well defined or poorly defined interface. Tumor wall thickness was calculated by the mean value between the maximum and minimum width on the contrast-enhanced T1-weighted images. Peritumoral edema on the MRI appears as a region of increased signal intensity of the extratumoral infiltrative portion outside the tumor on the T2-weighted image. Intratumoral hemorrhage was considered as the high signal area on the T2- & T1-weighted MR images without definite contrast enhancement on the contrast-enhanced T1-weighted MR images. The tumor resection rate was obtained by measuring the difference of tumor volume on the pre and postoperative brain MRI. The volume assessments of the mass or the peritumoral edema were performed by use of the commercial 3D reconstruction computer program (Aquarius, intuition edition, version 4.4, Terarecon, USA).

### *Statistical Methods*

The Mann-Whitney U test and the chi-square test were used to assess the differences in patient age between the cystic and noncystic GBMs. Fisher's exact test was used to assess the differences in tumor size, peritumoral edema and tumor interface irregularity between the cystic and noncystic GBMs. The Kaplan-Meier method was used to estimate the progression free survival parameters and log-rank tests were performed to determine any statistically significant differences between the groups. All the statistical analyses were performed by using the SPSS/PC_ version 15.0 software (SPSS, Seoul, Korea). For all evaluations, $p$ values $< 0.05$ were considered statistically significant.

## Results

Seven cases were classified as cystic GBMs and fourteen were noncystic GBMs. The cystic GBM group consisted of three men and four women, and the noncystic GBM group included nine men and five women. Table 1 summarizes the results of the statistical differences between the cystic and noncystic GBM groups. There was a statistically significant difference in age (Mann-Whitney U test; $p < 0.05$) between the cystic GBM group (mean age: 34 years, range: 2–18 years) and the noncystic GBM group (mean age: 52 years, range: 26–81 years). The median and range of the preoperative and postoperative KPS of the cystic GBM patient were 70, 30–80 and 70, 40–80, respectively. The median and range of the preoperative and postoperative KPSs of the noncystic GBM patient were 60, 30–80 and 65, 40–90, respectively.

The tumor volume was calculated to be more than 70 $cm^3$ in four of the seven cystic GBM patients (57%) and in four of the fourteen noncystic GBM patients (28%). Six cystic GBM patients (85%) had a well defined interface between the tumor and the normal brain tissue.

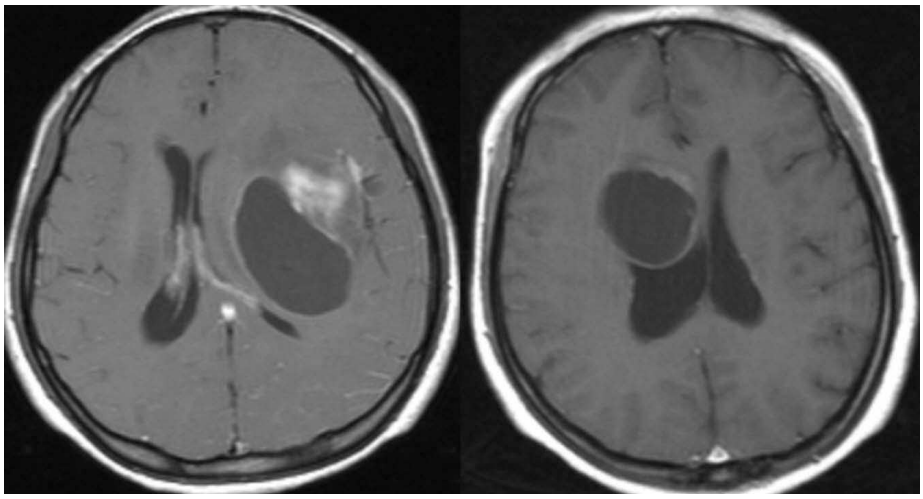


Fig. 1. The axial Gd-enhanced T1-weighted MR images obtained from two patients with cystic GBMs. The cystic portion of the tumor shows an ovoid shape, a well defined interface, homogeneous hypointensity and an enhanced even lining on a thin, smooth wall on the Gd-enhanced T1-weighted MR images.

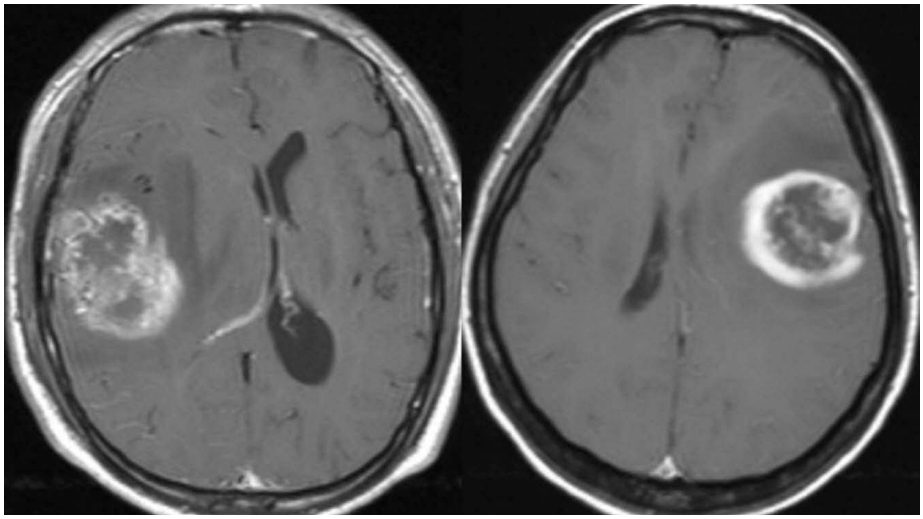


Fig. 2. The axial Gd-enhanced T1-weighted MR images obtained from two patients with non cystic GBMs. The non cystic GBM shows an irregular shape, a poor interface and an enhanced thick and uneven wall on the Gd-enhanced T1-weighted MR images.

The micrographs of the cystic GBM patients showed bizarre tumor cells with abundant eosinophilic cytoplasm and these cells were found in an edematous stroma. The pleomorphic tumor cells showed variations of size and shape. The large tumor cells had abundant eosinophilic cytoplasm with fibrillary processes (Fig. 2).

The other GBMs had poorly defined interfaces with tumor cells infiltrating into the brain parenchyma. The tumoral wall thickness was less than 2 cm in six of the seven cystic GBM patients (85%) and in three of the fourteen noncystic GBM patients (22%). The peritumoral edema was calculated to be less than 40 $cm^3$ in five of the seven cystic GBM patients (71%) and in two of the fourteen noncystic GBM patients (14%). Two cystic GBM patients (28%) and eight noncystic GBM patients (56%) showed intratumoral hemorrhage. All the cystic GBMs and 71% of the noncystic GBM patients received tumor resection. The median and range of the resection rate of cystic GBM patients were 78% and 53∼98%, respectively. The median and range of the resection rate of the noncystic GBM patient were 79% and 25∼100%, respectively. Five cystic patients and six noncystic patients received combined radiotherapy and chemotherapy, respectively. The other two and four cystic and noncystic GBM patient underwent radiotherapy alone.

In summary, the cystic and noncystic GBMs differed significantly for peritumoral edema, the tumor interface and the tumor wall thickness (Fisher's exact test; $p < 0.05$), but there was no significant difference for tumor volume and intratumoral hemorrhage (Table 1). The postoperative cumulative survival outcomes for the GBMs groups are shown in Table 2. For those with cystic GBMs, the 6-month, 1-year, 2-year and 3-year cumulative survival rates were 100%, 100%, 67% and 50%, respectively. For the patients with noncystic GBMs, the 6-month, 1-year, 2-year and 3-year cumulative survival rates were 69%, 49%, 14% and 7%, respectively. For the

Table 2. Outcomes of the Cystic and Noncystic GBMs

| Outcome | Cystic GBM | Non Cystic GBM |
|---|---|---|
| Median survival rate (month) | 43.8 | 12.5 |
| 6-month cumulative survival (%) | 100 | 69 |
| 1-year cumulative survival (%) | 100 | 49 |
| 2-year cumulative survival (%) | 67 | 14 |
| 3-year cumulative survival (%) | 50 | 7 |

Table 1. Clinical Characteristics of the Cystic and Noncystic GBMs

| Characteristic | Cystic GBM (%) | Non Cystic GBM (%) | *p*-value |
|---|---|---|---|
| No. of patients | 7 | 14 | |
| Age (year) | 15–50 | 26–81 | |
| Mean age ± SD | 34 ± 12.4 | 52 ± 11.6 | $p < 0.05$ |
| Gender (male: female) | 3:4 | 9:5 | |
| Preop KPS score (Median & range) | 70, 30–80 | 60, 30–80 | |
| Postop KPS score (Median & range) | 70, 40–80 | 65, 40–90 | |
| Tumor location | | | |
| Frontal | 3 | 6 | |
| Parietal | 2 | 7 | |
| Temporal | 3 | 3 | |
| others | - | 1 | |
| Tumor volume | | | $p = 0.35$ |
| ≥ 70 $cm^3$ | 4/7 (57) | 4/14 (28) | |
| < 70 $cm^3$ | 3/7 (42) | 10/14 (71) | |
| Tumor interface | | | $p < 0.05$ |
| Well defined | 6/7 (85) | 0/14 (0) | |
| Poor defined | 1/7 (14) | 14/14 (100) | |
| Tumor wall thickness | | | $p < 0.05$ |
| ≥ 2 cm | 1/7 (14) | 11/14 (78) | |
| < 2 cm | 6/7 (85) | 3/14 (22) | |
| Peritumoral edema | | | $p = 0.009$ |
| < 40 $cm^3$ | 5/7 (71) | 2/14 (14) | |
| ≥ 40 $cm^3$ | 2/7 (28) | 12/14 (85) | |
| Intratumoral hemorrhage | 2/7 (28) | 8/14 (56) | NS |
| Tumor resection rate (%) (Median & range) | 78, 53–98 | 79, 25–100 | |
| Postopeartive treatment | | | |
| Radiotherapy & Chemotherapy | 5/7 (71) | 6/10 (60) | |
| Radiotherapy | 2/7 (29) | 4/10 (40) | |

patients with cystic GBMs, the median survival was 43.8 months (range: 21.8-74.1 months). For the noncystic GBM patients, the median survival time was 12.5 months (range: 0.1-49.1 months). The differences in median survival were statistically significant (log-rank test; $p$ = 0.003) according to the Kaplan-Meier estimates (Fig. 3).

## Discussion

Glioblastomas have historically been classified as primary or secondary. The terms primary and secondary glioblastomas were first used by the German neuropathologist Hans-Joachim Scherer (12). Primary GBMs have been termed de novo. This term refers to the lack of a precursor lesion resulting from a single-step malignant transformation. Secondary GBMs has been referred to as progressive GBM. They develop by malignant progression from a low-grade astrocytoma or anaplastic astrocytoma. The development time varies from months to years. Primary and secondary GBMs are distinct entities according to their different genetic pathways, the associated patient ages and the different responses to therapy. Based on genetic typing, some authors also refer to primary and secondary glioblastomas as glioblastoma types 2 and 1 (9, 12-14). Several authors have studied the difference between primary and secondary GBMs as related to the genetic pathways and the clinical and radiologic features. Kleihues and Ohgaki have classified primary and secondary GBMs by the different genetic types and they found that secondary GBMs tend to develop at a younger age. For the radiologic features, the existence and nonexistence of necrosis and cystic formation is an important factor to evaluate the subtypes of GBMs. In general, approximately 10% of the tumors of the CNS are accompanied by a cystic area (15). There is a growing interest in the cystic lesions that accompany brain tumors. A cyst is commonly found in low-grade astrocytomas and in most pilocytic astrocytomas. The cause of cyst formation re-

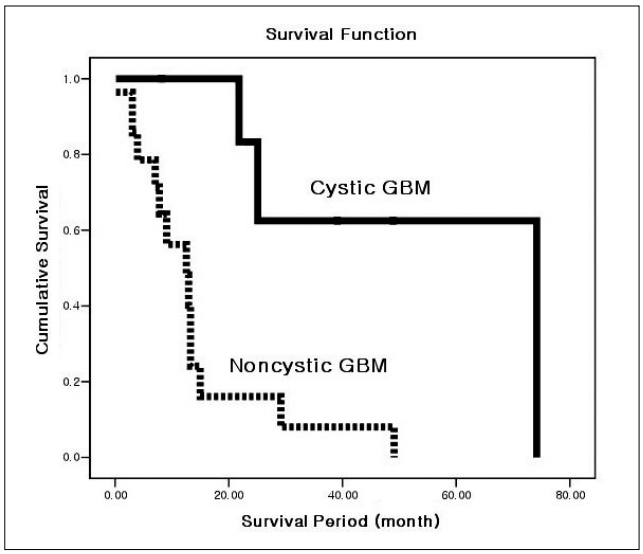
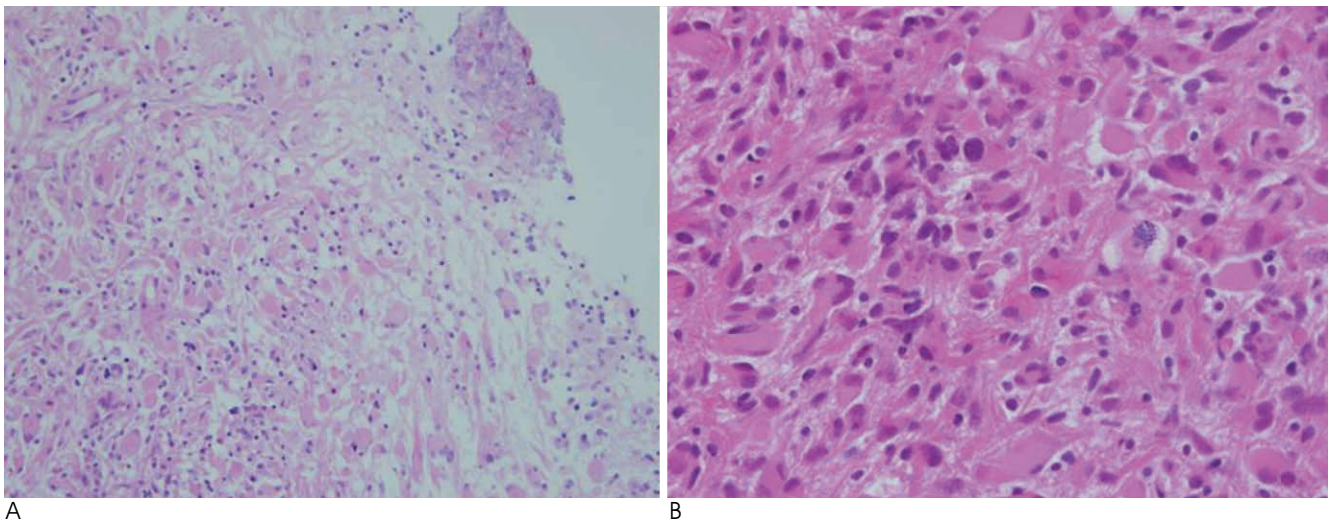



Fig. 4. Kaplan-Meier estimates of overall survival for the patients with cystic GBMs and noncystic GBMs. The differences between the two groups are significant (log-rank test; $p$ = 0.003).



A B

Fig. 3. A. Micrographs show the cystic GBM with the surrounding brain parenchyma. Bizarre tumor cells with abundant eosinophilic cytoplasm are found in an edematous stroma.
B. The pleomorphic tumor cells show variations of size and shape. The large tumor cells have abundant eosinophilic cytoplasm with fibrillary processes.

mains controversial. One hypothesis has claimed that large cysts are malignant transformations of previously undiagnosed cystic low-grade glioma. Laws et al reported that the low grade astrocytomas that include cysts have a better prognosis (16). According to Satoshi et al, the prognosis for cystic GBMs was significantly better than that for noncystic GBMs. They reported that cystic GBM patients were significantly younger than the non cystic GBM patients (13). Other studies have concluded that the secondary GBM patients are younger than the primary GBM patients (12). These observations suggest that most of the cystic GBMs have clinical features that are like those of secondary GBMs. Accordingly, it has been postulated that the large cysts accompanying tumors develop by malignant transformation from undetected low-grade glioma. Other hypotheses to explain the presence of cysts in GBMs include necrotic degeneration of brain tissue or blood-brain barrier (BBB) disruption. Maldaun et al revealed that the cystic patients' clinical characteristics such as age and the clinical manifestations differ from the previously published data about GBMs and they were not the same as the features of secondary GBM (a long preoperative clinical history and younger patients). Accordingly, no clinical data has been found to support the hypothesis of malignant transformation (8–10, 17, 18). In addition, Lohle et al modified the second hypothesis in several of their studies. In the past literature, it was believed that tumor cyst formation was caused by degeneration or necrosis of tumor tissue. The postulated hypothesis of intratumoral cyst formation is secretion of fluid by the tumor cells and cystic degenerative change within the tumor. However, the more recent reports by Lohle et al have suggested that cyst formation is due to an edematous process caused by BBB disruption and it is not the result of necrosis, based on the chemical assessment. This hypothesis proposes that cyst formation is caused by exudation of blood plasma proteins and the accumulation of interstitial fluid in the brain parenchyma. Lohle et al revealed a high protein concentration in the cysts with a highly similar spectrum of proteins in the tumor cyst fluid and the blood plasma. Brain tumor cyst fluid analysis found that blood plasma proteins constituted a major fraction (92%) of the tumor cyst fluid proteins. Fifty- fold higher concentrations of proteins were present in the tumor cyst fluid compared with that of the cerebrospinal fluid. This data suggests there is increased barrier permeability due to BBB disruption followed by exudation of plasma proteins into the brain parenchyma with formation of edema and the transition of the edematous tissue into cystic tumors (11, 19, 20).

Most GBMs have massive edema in the peritumoral area. This edema is due to the infiltration of tumor cells into the white matter and the mass effect of the tumors themselves. Vasogenic edema is caused by exudation of blood plasma proteins and the accumulation of interstitial fluid in the brain parenchyma through a damaged BBB, and this is similar to that producing cyst formation. According to Satoshi et al, cystic GBMs had smaller peritumoral edema regions and sharper boundaries than did the noncystic GBMs. Sawaya et al revealed a significant correlation between tPA (tissue plasminogen activators) and the presence and amount of peritumoral edema (21). Our study revealed that the cystic GBM patients were significantly younger and they had more favorable survival outcomes than did the patients with noncystic GBMs. This is similar to Satoshi's results. They reported the prognosis for cystic GBM was significantly better than that for noncystic GBM and the cystic GBM patients were significantly younger than the non cystic GBM patients (13). These observations suggest that most of the cystic GBMs have clinical features that are similar to those of secondary GBMs. However, this is not good evidence that cystic GBMs are secondary GBMs. This hypothesis additionally could be tested by looking at the gene expression and the IDH1 mutation status.

This study has some limitations. First, it is designed to retrospectively collect data from the preoperative MR images and medical records. A small patient population was enrolled in this study. As mentioned earlier, it is difficult to distinguish cysts with necrosis, regardless of our several criteria. We also had some problems measuring the edema in some cases of infiltrating tumors due to the indistinct separation between the tumor and the surrounding edema.

In conclusion, the cystic GBMs had a well-defined tumor interface, a thin wall and minimal edema as compared with the noncystic GBMs. The cystic GBM patients were significantly younger than the noncystic GBM patients. In addition, our analysis demonstrated that the patients with cystic GBMs had more favorable survival outcomes than did the patients with noncystic GBMs.

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# 낭성 다형교모세포종과 비낭성 다형교모세포종 환자의 수술 전 영상의학적 차이와 생존율에 미치는 영향[1]

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**목적:** 낭성 다형교모세포종과 비낭성 다형교모세포종 환자의 수술 전 영상의학적 차이와 생존율의 차이를 알아보고자 하였다.

**대상과 방법:** 조직학적으로 진단된 21명의 다형교모세포종 환자를 대상으로 하였으며 낭성 다형교모세포종과 비낭성 다형교모세포종 환자의 수술 전 MRI에서 종괴의 크기, 종괴와 주변 뇌조직의 경계면, 종괴벽의 두께와 종괴주위 부종의 차이를 비교하였다.

**결과:** 7명의 낭성 다형교모세포종과 14명의 비낭성 다형교모세포종으로 분류되었으며 낭성 다형교모세포종은 비낭성 다형교모세포종보다 경계가 좋으며 85%에서 2 cm 미만의 종괴벽을 가지고 71%에서 40 $cm^3$ 종괴주위 부종을 동반하였다($p < 0.05$). 또한, 낭성 다형교모세포종과 비낭성 다형교모세포종은 발병 나이와 수술 후 중앙생존기간(median survival) 간에 유의한 차이를 보였다($p < 0.05$).

**결론:** 낭성 다형교모세포종은 비낭성 다형교모세포종보다 주변 뇌조직과의 경계가 좋고, 종괴벽의 두께가 얇으며 상대적으로 적은 종괴주위 부종을 동반하였다. 또한, 낭성 다형교모세포종은 비낭성 다형교모세포종에 비해 어린 나이에 발병하며 수술 후 중앙생존기간(median survival)도 더 긴 것으로 나타났다.