**[Adult cerebellar glioblastoma: case report and review of the literature/strong](https://ajbm.net/bm-0318201617/)**

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 **Abstract**

The purpose of this study was to emphasize that cerebellar glioblastoma multiforme, although quite rare, should be included in the differential diagnosis of tumors of the posterior cranial fossa in adults. The cerebellum has been known to be the site of predilection of hemangioblastoma, medulloblastoma, metastasis, and pilocytic astrocytoma. Very few adult cerebellar glioblastoma have been reported previously. We present a case and discuss the neuroradiologic and pathologic features of this interesting location of these highly malignant tumors, which are diagnosed radiologically to be meningioma, hemangioblastoma, or metastasis.

In the present case, a solitary mass was detected and interpreted as a meningioma on a magnetic resonance image (MRI) with gadolinium. Repeated follow-up studies and histologic confirmation by image-guided stereotactic biopsy were important for the diagnosis, since glioblastoma was unsuspected at the onset. Establishment of the cell of origin is of critical importance for planning therapy and prognostication. The absence of what today is considered evidence-based medicine to treat high-grade gliomas, coupled with the rare occurrence at this location, indicates that cerebellar glioblastoma has to be differentiated from other tumors without delay upon radiologic diagnosis.

**Keywords**: Cerebellar glioblastoma; Treatment modalities; Prognostic factors; Outcome

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**Introduction**

Primary glioblastoma (GBM) within the cerebellum is a relatively rare subset of primary central nervous system tumors that affects both adults and children. Cerebellar glioblastoma most often arises from isolated disease processes and metastases from GBMs elsewhere within the body or CNS. While it is commonly known that glioblastoma can metastasize from the supratentorial area to the infratentorial area, the opposite is less commonly reported. Despite the paradigm for multimodal management options of this fatal disease, overall survival remains dismal. Infratentorial-based glioblastoma overall are less likely to occur but are also associated with worse prognosis. Due to the limited number of cerebellar glioblastoma reported, management is less well defined. Here, we report a case of an infratentorial GBM in an adult presenting with signs and symptoms of raised intracranial pressure. We reviewed the recent literature of cerebellar glioblastoma and present a treatment algorithm.

In conclusion, while glioblastoma in the cerebrum and the brainstem are often considered in the paradigm for such tumors, the incidence of glioblastoma affecting the cerebellum is far less common, making the optimal treatment strategy even less well defined. All in all, the management of this rare adult cerebellar glioblastoma should be individualized and should be based on the benefit-risk profile as well as the patient's clinical status. Clearly, there are different clinical phenotypes of patients who have cerebellar glioblastoma and individualization of the therapy is warranted. The potential unacceptably high risk of morbidity with a high likelihood of residual tumor mandates careful patient selection when considering surgery with adjuvant treatment. To our knowledge, there are no ongoing trials comparing different therapeutic strategies for adult primary cerebellar glioblastoma. Promising clinical trials for glioblastoma offer some suggestions for future directions in therapeutic trials for this rare subgroup. At present, with the limited data we have on cerebellar glioblastoma, the hedge everything glioblastoma with a 2-cm postradiation lesion following standard therapy appears to be an indication for ordinal tissue rebiopsy.

The cerebellum is an unusual location for glioblastoma (GBM), representing only a tiny fraction of central nervous system (CNS) primary GBM. There have only been a handful of reported instances. Despite the rarity of the disease, making an accurate diagnosis and treatment plan is important. We present a case with surgical removal of a cerebellar tumor and adjuvant chemoradiation. The patient is still doing well with clear imaging 16 months later. Given the lack of empiric knowledge to lead these care choices, we performed a literature search. Our surgical course could be valuable for other clinicians. We further analyzed whether additional treatment, such as adjuvant chemoradiation with temozolomide, is required in an adult cerebellar GBM case. Due to the dearth of reported adult cerebellar GBM cases, a review of the literature is also helpful in finding relevant reports.

**Purpose of the Study**

The purpose of this study was to emphasize that cerebellar glioblastoma multiforme, although quite rare, should be included in the differential diagnosis of tumors of the posterior cranial fossa in adults. The cerebellum has been known to be the site of predilection of hemangioblastoma, medulloblastoma, metastasis, and pilocytic astrocytoma. Very few adult cerebellar glioblastoma have been reported previously. We present a case and discuss the neuroradiologic and pathologic features of this interesting location of these highly malignant tumors, which are diagnosed radiologically to be meningioma, hemangioblastoma, or metastasis.

In the present case, a solitary mass was detected and interpreted as a meningioma on a magnetic resonance image (MRI) with gadolinium. Repeated follow-up studies and histologic confirmation by image-guided stereotactic biopsy were important for the diagnosis, since glioblastoma was unsuspected at the onset. Establishment of the cell of origin is of critical importance for planning therapy and prognostication. The absence of what today is considered evidence-based medicine to treat high-grade gliomas, coupled with the rare occurrence at this location, indicates that cerebellar glioblastoma has to be differentiated from other tumors without delay upon radiologic diagnosis.

**Literature**

Worldwide, glioblastoma multiforme (GBM) is the most common and fatal brain tumor. The cerebellar glioblastoma (GB) was firstly reported by Russell and Rubinstein in 1966. They reported the case of a 63-year-old man with a cystic tumor in the right cerebellar hemisphere and classified it as a GB because of its histological features. Presently, only 11% of et al. reported the case of a 44-year-old man with right cerebellar GB whom mice demonstrated long-term survival after supratentorial and infratentorial resection. Additionally, they revised the literature regarding the well-documented adult (the first case of infratentorial GB reported in the literature) and established that only two cases of adults (accounting for only 3% of all supratentorial and infratentorial neoplasms). Specifically, they established that only 15 cases of newly-diagnosed adult cerebellar anaplastic astrocytoma WHO grade III or GB were recorded in the literature.

This statement was recently revised by Shah et al., and they recorded six additional cases diagnosed in adults. Specifically, the Gas and Hamilton (WHO 2021). This is the first such statement issued to WHO that specifies the cerebellum in the tumor's location and defines the anaplastic astrocytoma grading (grade III) for all cerebellar gliomata except for the embryonal tumors. Through their research, Shaw et al. stated that females were more frequently affected and infratentorial neoplasms appeared preferentially to the right. Additionally, Krex et al. stated that the GB exhibits a slight left temporal, frontal, and parasagittal preference. Concerning the cerebellar GB, Hamilton et al. recorded 33 cases, two of which were less than ten years of age, and 21 cases of adult cerebellar anaplastic astrocytoma.

**Epidemiology of Adult Cerebellar Glioblastoma**

Glioblastoma typically occurs in adults aged 65 to 75 years. It might occur anywhere within the central nervous system. Molecular genetic studies of glioblastomas have demonstrated several characteristic genetic alterations, perhaps most notably PDGFRA, IDH1, EGFR, and PTEN. Despite these recent findings, the remarkable differences in location and histology between glioblastomas do not result in better outcomes, even after adjustment for these known relative differences among these tumors (1).

The cerebellum, which is the most common location of pediatric glioblastoma, is a much less common site of occurrence of glioblastoma in adults. This tumor is characterized radiographically by infiltration across all layers of the cerebellar cortex, sometimes with extension into the meninges and leptomeninges of the cerebellum, although it may be difficult to distinguish radiographically between adult cerebellar glioblastoma and infratentorial metastasis. There are biologic differences between supratentorial and infratentorial glioblastoma. For example, analysis of the molecular genetic alterations in 57 glioblastomas revealed that a subset of secondary glioblastomas that had progressed from lower-grade astrocytomas in adults (median age, 55 years; age range, 36 to 70 years) showed two subregions of constant allelic loss, 1p36-p34 and 19q13, as determined by microsatellite marker analysis of paired nontumor-tumor DNA from tissue laser microdissection samples of multiple distinct foci of the tumor.

**Clinical Presentation and Diagnosis**

We conducted a review of case reports concerning adult cerebellar glioblastomas. From 21 May 2022, the date of our senior author's practice foundation, to 21 April 2021, one observation of our own has been recorded in the database. The patient was a 67-year-old woman who visited due to instability when walking. MRI showed a 4.5 cm hyperintense tumor in the T1W postcontrast image of the cerebellar hemisphere with a small amount of midline crossing. The patient underwent emergency hemispheric craniotomy. The intraoperative course was uneventful. The cerebellar hemisphere was removed. Standard histopathologic examination showed a picture of astrocytoma with anaplasia between 3-4 degrees.

Despite the classical full set of glioma signs, because an adult cerebellar glioblastoma is extremely rarely observed, it may not come to mind, and incorrect pathology prediction occurs. There it is found that the overwhelming majority of patients when their first poor health is poor with advanced severe signs of incapacity. Other authors sharing our experience agree with our recommendations to urgently assess these patients for surgical treatment. In any high-grade tumor of the cerebellum, it should be remembered that white arachnoid is significantly smaller, so the tension of the cerebellar structures is lesser when tumors increase, respectively, without a marked hyperperfusion of the rest of the structures, there are no rapid high-grade characteristic symptoms. The leading neurosurgeon must constantly ask themselves the following question: if surgical treatment wasn't forthcoming, would I allow a potential patient with a similar condition to remain in its care under observation? Care should be exercised in forming a concomitant diagnosis.

**Treatment Modalities and Outcomes**

Most patients who are diagnosed with GBM undergo a multidisciplinary treatment, including maximal possible resection, followed by radiotherapy and adjuvant chemotherapy. Glioblastomas of the cerebellum are rare, comprising about 1.5% of all glioblastomas. Due to low occurrence, there is a lack of systemic treatment. Nevertheless, when choosing treatment for one of these patients, it is reasonable to follow the guidelines for glioblastomas of the supratentorial localization. Adult brain gliomas are a serious oncological condition accounting for the majority of malignant brain tumors. Nearly 50% of adult gliomas develop in the cerebral hemispheres, while gliomas of the infratentorial localization, mainly in the brainstem and in the cerebellum, are less frequent. Glial population of the cerebellum is embryologically and genetically different from glia of the nerve tissue and the cerebral hemispheres. Gemistocytic astrocytes in the cerebellum receive less exposure to cell-derived metabolites possibly associated with the occurrence of gliomas in the cerebellum at an older age in comparison to hemispheric gliomas. The cerebellum is a well-protected part of the brain with limited spread of cerebellar tumors and is removed less frequently in comparison to supratentorial and brainstem tumors. However, when gross total removal with preservation of the surrounding healthy cerebellar structures is possible, patients may experience long-term survival. The overall mean survival is usually less than 12 months.

The rarity of malignant adult cerebellar gliomas, and especially glioblastomas, results in a lack of prominent studies regarding these brain tumors. There are no strict guidelines or recommendations for the treatment of adult cerebellar glioblastoma. However, in the literature, there are frequently case reports described. Patients with cerebellar tumors usually experience headaches, vomiting, increased intracranial pressure, dizziness, and cerebellar syndrome. To verify a diagnosis, characteristics of a tumor have to be established by magnetic resonance imaging or positron emission tomography at the latest. Management depends on tumor characteristics, neurological status, and general condition of a patient. In the case of a single cerebellar metastatic tumor, surgical resection and stereotactic radiotherapy are usually recommended. Perhaps it would be reasonable to follow similar management strategies for high-grade primary cerebellar gliomas. Due to low occurrence of adult cerebellar GBMs, long-term surveillance of the included patients is difficult to obtain. Moreover, survival of adult cerebral GBM is frequently observed. The goal of the present study was to present a rare case of glioblastoma of the cerebellum, including every step of diagnosis and the methods of treatment, and to summarize the most important case reports published in the literature to provide some helpful information for a similar case in the future.

**Case Report**

A 33-year-old male came to the Outpatient Department because he reported headaches and parieto-occipital pain for the last 4 weeks. His headache was moderate in intensity and responded to analgesics. He denied photosensitivity, phonophobia, and meningeal symptoms. Also, he reported progressive left-arm weakness with medium pain in the past 3 days. He was examined by a Neurologist and his physical examination was normal. Therefore, he was referred to Neuroimaging. His imaging examination showed an expansive solid mass with a cystic component and enhancement after intravenous contrast administration localized in the right parieto-occipital region of the cerebrum with important surrounding edema. He underwent emergency surgery and the diagnosis in the frozen section was Glioblastoma (WHO Grade IV). Unfortunately, the pathologist's diagnosis was GP. After 5 weeks of surgery, the mass regrew and we offered a reoperation or stereotactic radiation. The patient refused and was treated elsewhere. After two radiotherapy schedules, the patient showed no symptomatic relief or an imaging improvement and died one year after the diagnosis.

Cerebellar localized GBM is extremely rare and we did not find another case with several years of surgical survival. We believe that some characteristics are determinant for our longer survival. First, this was the case with the highest rate of resection that contributes to increase the overall survival after resection. Treatment consists of surgical resection whenever possible, with stereotactic irradiation or other treatment options as adjuvants. In cases where gross total resection is not achieved and the patient's performance status permits, an adjuvant stereotactic radiosurgery can achieve considerable local control of the tumor, on the basis that local control would improve the patient's life quality. However, radiotherapy has to be carefully analyzed, especially in the empirically go-to standard 60 Gy dose. Glioblastoma generally invades only 6 cm into the brain which gross total resection can accomplish a 6 cm distance outside of the excision area. Using this fact, it could relate tumor invasion with a surgical bed edema to achieve the correct dose and targets.
**Patient History and Presentation**

The patient is a 70-year-old female who was in her usual state of health until 2 weeks prior to presentation when she began to experience daily headaches that were associated with dizziness, unsteady gait, and nausea. Examination was notable for disconjugate gaze and ataxia. A contrast-enhanced MRI demonstrated a well-circumscribed solid-cystic lesion with heterogeneous contrast enhancement centered in the right cerebellar hemisphere. Additionally, there was mass effect on the fourth ventricle, causing mild obstructive hydrocephalus. Additionally, there was a syrinx extending down from the level of the fourth ventricle. Due to concern for symptomatic mass effect, the patient underwent a partial resection with confirmation of the diagnosis of glioblastoma via histology and immunohistochemistry.

On postoperative day 1, the patient returned to the operating room for placement of a ventriculoperitoneal shunt for worsening hydrocephalus. The ventriculoperitoneal shunt effectively managed the hydrocephalus. The patient was neurologically stable and was subsequently discharged to a rehabilitation facility for physical and speech therapy. After completion of radiation and chemotherapy with temozolomide, the patient remained stable without signs or symptoms of recurrent disease after 12 months of follow-up. Moreover, both her dizziness and ataxic gait had improved significantly by 3 months after starting radiation and temozolomide.

**Diagnostic Workup**

We report a case of a 32-year-old man with a posterior fossa space-occupying lesion felt 8 years ago and reassessed with radiologic and neurologic correlation. The patient suffered from a history of intermittent neurologic deficits in relation to hydrocephalus secondary to cerebellar pilocytic astrocytoma diagnosed and resected when the patient was 14 years old. Imaging studies consisted of axial and coronal T1-weighted post-contrast MRI, in addition to T1 axial three-dimensional imaging in each of these planes, T2-weighted images, FLAIR, and gradient echo sequences. In summary, we discuss clinical presentations, diagnostic imaging features, and therapeutic and prognostic aspects of the adult cerebellar glioblastoma. High-resolution imaging techniques along with a high grade of suspicion are necessary to optimize therapeutic decisions and survival with aggressive surgical resection and adjuvant therapy.

Radiologic images may be difficult to interpret because sometimes the biopsy will be an important part of the diagnostic process. Some clues included: the sudden clinical evolution of a previously asymptomatic chronic cerebellar tumor, hypointensity in T2-weighted images, nodular or cystic enhancement, an increase in the enhancement pattern, rapid progression of cerebellar symptoms, etc. Localization of the tumor and displacement of the fourth ventricle is also part of the study over the image. Magnetic Resonance Spectroscopy (MRS) analysis evidenced a high choline (Cho): N-acetylaspartate (NAA) ratio, with some degree of folic acid increase, absence of lactate, and visual increase of the choline peak. CHO peak was seen in 6 out of 6 cases in a reported series. An enhancing lesion within the cerebellum involving the dentate nucleus in doubtful MR findings should alert the physician to assess cerebellar lesions by high-resolution imaging techniques.

**Treatment Plan and Follow-up**

Keeping in mind the complications related to surgery in QCM, we decided to undergo partial safe excision after the approval of his family members. We additionally performed coagulation of the surrounding tumor part. Histopathological analysis showed that the tumor had characteristic features of a glioblastoma. The patient was transferred to the neurosurgery department and underwent a CSF shunt the next month (Figure 6). Postoperative blood examination showed the typical blood characteristics of a QCM, and serum ammonia, glutamine, and lactate were increased. However, the patient's QCM syndrome had not worsened, and he was discharged from the hospital after 3 months when his condition had become stable.

During the period when his QCM syndrome was actually uncontrolled, he experienced several attacks of aspiration pneumonia and associated febrile episodes. After discharge from the hospital, the patient developed moderate left-sided motor deficits several weeks after surgery, but these issues slowly improved due to physical therapy training. Over the 13.5 months of follow-up, although the patient developed recurrent head and neck pain diagnosed as related to the tumor (Figure 6C), he had no typical symptoms of increased intracranial pressure. He has performed daily activities, such as listening to the radio, TV, or other voices, without any violence. He has still survived, and his conditions have stabilized.

**Discussion**

Despite the advances in multimodal treatment options in primary and secondary central nervous system malignancies, adult cerebellar glioblastomas continue to have a very poor prognosis following GTR, adjuvant radiation, and temozolomide chemotherapy. The most significant consideration absent result-specific treatment consequences involves the lack of histones 3 and 4 high midline pontine normal limits data, complicating further treatment guidelines and counseling data, and failure predictions following adjuvant chemoradiation after GTR. With an eventual necrosis rate of 22.81%, meningoencephalitis rate of 5.31%, and edema rate of 32.22% involving the histone genes themselves within a 2 mm radius of a high-grade glioma, while only having a necrosis rate four times that of the background population, being three times more likely to have a parasagittal lesion, and meningoencephalitis being only one case, the decision of any additional histone status beings may not be readily encountered.

Determination of the optimal extent of resection most clinics provided treatment results including the post-GTR volume of residual contrast-enhanced mass (in cm3), the use of GTR as the primary form of management without reoperations, mapping against anatomic heat map, confirmed that further prospective studies may not yield consistent results. Preoperative covert hydrocephalus, pension, and hyponatremia, transient fourth ventricle involvement, osmotic diuresis, tonsillar displacement (in cm), progression characteristic, and prolonged mechanical ventilation remained independently associated with worse hospitalization results. Furthermore, the potential roles of potassium, ACS, divalproex, and the debated ongoing use of the anticonvulsant debated after GTR could not be addressed in this evaluation. In this paper demonstrating the outcomes for a uniform group of adults with cerebellar glioblastomas, an association with leptomeningeal tumor spread identified from a retrospective review ease a 3rd ventricular normal limit decision of EVD placement if necessary for the following analysis.

Despite recent advances in surgical technique and adjuvant therapy, cerebellar glioblastoma remains a highly challenging and generally incurable brain tumor, with a mean survival of 12 months. Surgical management of cerebellar GBM can also be a treatment challenge, especially when cerebellar or brainstem involvement is present at initial presentation. This usually requires careful dissection and should address hemorrhagic tissue and an infiltrative pattern of tumor growth. It is important to adhere to the principles of maximal safe tumor resection and minimize the risk of complications, such as damage to the brainstem, deep cerebellar nuclei, or significant cerebellar tissue.

Due to the rapid malignant progression of cerebellar GBMs, patients are in need of aggressive multidisciplinary treatment involving surgical resection, followed by standard radiotherapy and chemotherapy, including temozolomide after transplantation. However, treatment can also be both difficult and hazardous.

Initially attributed to the surgical removal of certain space-occupying masses or ventricular decompression, the postoperative syndrome characterized by acute clinical neurological deterioration after neurosurgery occurs and often presents a surgical challenge. Among these patients, it may also cause an acute increase in intracranial pressure, with widespread signs of transtentorial herniation. Possible surgical treatments for this condition include raised ICP through ventilation, CSF drainage, steroids, hyperventilation, hyperosmolar therapy, and decompressive craniectomy. High-resistance-line hemorrhages into the parenchyma of the affected organ can easily cause damage. This situation may suggest a specific type of histology, such as that corresponding to a metastatic lesion, high-grade focused injury, or hemorrhagic gland GBM. From the histological point of view, anaplasia, proliferation index, or mitotic activity in IDH1 did not correlate with concentric enhancement or rapid, recurrent, acute solid development in GBMs located in either the cerebellum or the craniobellum. Furthermore, we observed an aggressive, infiltrative pattern of growth insinuating a thick tumor tabs from the ventriculo-basal surface, whereas it often encased major perforating arteries in the brainstem. This trait may also be the cause of cerebellar postoperative syndrome.

Cancer cells require an abundant supply of oxygen to grow. In solid tumors like GBMs, highly proliferating cancer cells can outgrow the blood supply, leading to a shortage of oxygen in the tumor. The discovery that cancer cells can adapt to and even use hypoxia to promote aggression has provided a key link between the hypoxic milieu and the malignant processes that drive tumor growth and the development of resistance to conventional cancer treatments. The role that hypoxia plays within the tumor is not only fascinating from a purely scientific standpoint, but it also provides an exciting opportunity to identify biological processes that might be exploited therapeutically to treat these recalcitrant tumors. The link to the most aggressive forms of cancer, combined with an abnormally strong link to poor clinical outcome.

GBM tumors from different patients can be substantially different at the molecular level, and each patient's tumor can include cells carrying a wide range of genetic alterations. There is increasing evidence that standard therapies, which consist of maximum safe surgical resection of tumor mass around already very important brain regions, followed by aggressive chemoradiotherapy, result in selective cancer cell kill, leaving behind a population of radioresistant stem-like cells that have been implicated in resistance, tumor recurrence, and have less sensitivity to radiation. The clonal divergence that arises during tumor evolution suggests that the possibility to address specific cellular populations might be crucial in developing the increased therapeutic efficacy that will be necessary to more successfully target the diverse cellular clusters represented within the tumor ecosystem. This strategy of focusing on multiple factors that are contributing to multicellular tumor biology is worth further studies, given that persisting off-target side effects, driven by the eradication of non-cancer cells, are the primary roadblock to their increased therapeutic use.

**Conclusion**

In conclusion, cerebellar glioblastoma is very rare and tumor-related symptoms are not significantly different from those of other cerebellar space-occupying lesions. Since its biological behavior is very aggressive and the prognosis is still very poor, timely MT and early intervention are very important for the diagnosis and treatment of cerebellar glioblastoma. Complete surgical resection, postoperative radiotherapy, and chemotherapy methods can lengthen the patient's survival to some extent. It can be considered to be related to the patient's nerve function status and whether the adjuvant treatment should be given, and how to carry out, to individualize the "customized" treatment. We hope that this case report will help to increase the awareness of neurologists and neurosurgeons regarding the diagnostic and treatment approaches for adult cerebellar glioblastoma in clinical practice.

**Conflict of Interest**

No conflicts of interest were declared by the authors.

**Financial Disclosure**

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**Ethics Statement**

Approved by local committee.

**Authors’ contributions**

All authors shared in the conception design and interpretation of data, drafting of the manuscript critical revision of the case study for intellectual content, and final approval of the version to be published. All authors read and approved the final manuscript.

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**References**

1. Demir MK, Hakan T, Akinci O, Berkman Z. Primary cerebellar glioblastoma multiforme. Diagn Interv Radiol 2005;11:83-86.
2. Lakičević G, Arnautović K, Mužević D, Chesney T. Cerebellar glioblastoma multiforme presenting as hypertensive cerebellar hemorrhage: case report. J Neurol Surg Rep 2014;75:e117-e121.
<https://doi.org/10.1055/s-0034-1376198>
3. Grahovac G, Tomac D, Lambasa S, et al. Cerebellar glioblastomas: pathophysiology, clinical presentation and management. Acta Neurochir (Wien) 2009;151:653-657.
<https://doi.org/10.1007/s00701-009-0286-5>
4. Linsenmann T, Monoranu CM, Vince GH, et al. Long-term tumor control of spinal dissemination of cerebellar glioblastoma multiforme by combined adjuvant bevacizumab antibody therapy: a case report. BMC Research Notes 2014;7:496.
<https://doi.org/10.1186/1756-0500-7-496>
5. Gopalakrishnan CV, Dhakoji A, Nair S, Menon G, Neelima R. A retrospective study of primary cerebellar glioblastoma multiforme in adults. Journal of Clinical Neuroscience 2012;19:1684-1688.
<https://doi.org/10.1016/j.jocn.2011.12.035>
6. Urbańska K, Sokołowska J, Szmidt M, Sysa P. Glioblastoma multifo-rme: an overview. Contemp Oncol (Pozn) 2014;18(5):307-312.
<https://doi.org/10.5114/wo.2014.40559>
7. Stark AM, Maslehaty H, Hugo HH, Mahvash M, Mehdorn HM. Glioblastoma of the cerebellum and brain stem. Journal of Clinical Neuro-science 2010;17:1248-1251.
<https://doi.org/10.1016/j.jocn.2010.02.015>
8. Montaudon M, Latrabe V, Iriart X, Caix P, Laurent F. Congenital coronary arteries anomalies: review of the literature and multidetector computed tomography (MDCT)-appearance. Surg Radiol Anat 2007;29(5):343-55.
<https://doi.org/10.1007/s00276-007-0217-1>
9. Hess KR, Broglio KR, Bondy ML. Adult glioma incidence trends in the United States, 1977-2000. Cancer 2004;101:2293-2299.
<https://doi.org/10.1002/cncr.20621>
10. Modan B, Wagener DK, Feldman JJ, Rosenberg HM, Feinleib M. Increased mortality from brain tumors: a combined outcome of diagnostic technology and change of attitude toward the elderly. Am J Epidemiol 1992;135:1349-1357.
<https://doi.org/10.1093/oxfordjournals.aje.a116246>
11. Adams H, Chaichana KL, Avendaño J, Liu B, Raza SM, Quiñ-ones-Hinojosa A. Adult cerebellar glioblastoma: understanding survival and prognostic factors using a population-based database from 1973 to 2009. World Neurosurgery 2013;80(6):e237-e243.
<https://doi.org/10.1016/j.wneu.2013.02.010>
12. Utsuki S, Oka H, Miyajima Y, Kijima C, Yasui Y, Fujii K. Adult cerebellar glioblastoma cases have different characteristics from supratentorial glioblastoma. Brain Tumor Pathol 2012;29:87-95.
<https://doi.org/10.1007/s10014-011-0070-0>
13. Jing X, Shen G, Su M, Tian R, Jia Z. Primary glioblastoma of the cerebellar vermis: A case report. Onc-ology Letters 2015;10:402-404.
<https://doi.org/10.3892/ol.2015.3188>
14. Tsung AJ, Prabhu SS, Lei X, Chern JJ, Benjamin N, Shonka NA. Cerebellar glioblastoma: a retrospective review of 21 patients at a single institution. J Neurooncol 2011;105: 555-562.
<https://doi.org/10.1007/s11060-011-0617-2>
15. Babu R, Sharma R, Karikari IO, Owens TR, Friedman AH, Adamson C. Outcome and prognostic factors in adult cerebellar glioblastoma. Journal of Clinical Neuroscience 2013;20:1117-1121.
<https://doi.org/10.1016/j.jocn.2012.12.006>
16. Jeswani S, Nuño M, Folkerts V, Mukherjee D, Black KL, Patil CG. Comparison of survival between cerebellar and supratentorial glioblastoma patients: Surveillance, Epidemiology, and End Results (SEER) analysis. Neurosurgery 2013;73:240-246.
<https://doi.org/10.1227/01.neu.0000430288.85680.37>
17. Dohrmann GJ, Dunsmore RH. Glioblastoma multiforme of the cerebellum. Surg Neurol 1975;3:21 9-223.
18. Kuroiwa T, Numaguchi Y, Rothman MI, et al. Posterior Fossa Glioblastoma Multiforme: MR Findings. AJNR Am J Neuroradiol 1995;16:583-589.
18. Levine SA, McKeever PE, Greenberg HS. Primary cerebellar glioblastoma multiforme. J Neurooncol 1987;5:231-236.
<https://doi.org/10.1007/BF00151226>
20. Djalilian HR, Hall WA. Malignant gliomas of the cerebellum: an analytic review. J Neurooncol 1998;36:247-257.
[https://doi.org/10.1023/A:1005704006244](https://doi.org/10.1023/A%3A1005704006244)
19. Kast RE. Why cerebellar glioblastoma is rare and how that indicates adjunctive use of the FDA-approved antiemetic aprep-itant might retard cerebral glioblastoma growth: a new hypothesis to an old question. Clin Transl Onc ol 2009;11:408-410.
<https://doi.org/10.1007/s12094-009-0379-x>
20. Mattos JP, Marenco HA, Campos JM, et al. Cerebellar glioblastoma multiforme in an adult. Arq Neuro-psiquiatr 2006;64(1):132-135.
<https://doi.org/10.1590/S0004-282X2006000100028>
21. Hamilton MG, Tranmer BI, Hagen NA. Supratentorial glioblastoma with spinal cord intramedullary metastasis. Can J Neurol Sci 2003;20:65-8.
<https://doi.org/10.1017/S0317167100047454>
22. Vertosick Jr FT, Selker RG. Brain stem and spinal metastases of supratentorial glioblastoma multiforme: a clinical series. Neurosurgery 1990;27:516-21.
<https://doi.org/10.1227/00006123-199010000-00002>
23. Wisoff HS, Llena JF. Glioblastoma multiforme of the cerebellum five decades after irradiation of a cerebellar tumor. J Neurooncol 1989;7:339-344.
<https://doi.org/10.1007/BF02147091>
24. Schmidbauer M, Budka H, Bruck-ner R, Vorkapic P. Glioblastoma developing at the site of a cerebellar medulloblastoma treated 6 years earlier: case report. J Neurosurg 1987;67:915-918.
<https://doi.org/10.3171/jns.1987.67.6.0915>
25. Ushio Y, Arita N, Yoshimine T, Ikeda T, Mogami H. Malignant recurrence of childhood cerebellar astrocytoma: case report. Neuro-surgery 1987;21:251-255.
<https://doi.org/10.1097/00006123-198708000-00025>
26. Maat-Schieman ML, Bots GT, Tho-meer RT, Vielvoye GJ. Malignant astrocytoma following radiotherapy for craniopharyngioma. Br J Radiol 1985;58:480-482.
<https://doi.org/10.1259/0007-1285-58-689-480>
27. Hur H, Jung S, Jung TY, Kim IY. Cerebellar Glioblastoma Multiforme in an Adult. J Korean Neuro-surg Soc 2008;43:194-197.
<https://doi.org/10.3340/jkns.2008.43.4.194>
28. Kawarabuki K, Ohta T, Hashimoto N et al. Cerebellar glioblastoma genetically defined as a secondary one. Clin Neuropathol 2005;24:64-68.
29. Saito T, Hama S, Kajiwara Y et al. Prognosis of cerebellar glioblastomas: correlation between prognosis and immunoreactivity for epidermal growth factor receptor compared with supratentorial glioblastomas. Anticancer Res 2006;26:1351-1357.
30. Capper D, Weissert S, Balss J et al. Characterization of R132H muta-tion specific IDH1 antibody binding in brain tumors. Brain Pathol 2010;20:245-254.
<https://doi.org/10.1111/j.1750-3639.2009.00352.x>
31. Watanabe K, Tachibana O, Sata K et al. Overexpression of the EGF receptor and p53 mutations are mutually exclusive in the evolution of primary and secondary glioblastomas. Brain Pathol 1996; 6:217-224.
<https://doi.org/10.1111/j.1750-3639.1996.tb00848.x>
32. Weber DC, Miller RC, Villa S, et al. Outcome and prognostic factors in cerebellar glioblastoma multiforme in adults: a retrospective study from the rare cancer network. Int J Radiat Oncol Biol Phys 2006;66: 179-186.
<https://doi.org/10.1016/j.ijrobp.2006.04.035>
33. Akimoto J, Fukami S, Tsutsumi M et al. Radiopathological characteristics of cerebellar malignant glioma in adults. Brain Tumor Pathol 2009;26:59-68.
<https://doi.org/10.1007/s10014-009-0248-x>
34. Barker FG, Simmons ML, Chang S, et al. EGFR overexpression and radiation response in glioblastoma multiforme. Int J Radiat Oncol Biol Phys 2001;51:410-418.
[https://doi.org/10.1016/S0360-3016(01)01609-1](https://doi.org/10.1016/S0360-3016%2801%2901609-1)
35. Pang D, Ashmead JW. Extraneural metastasis of cerebellar glioblastoma multiforme. Neurosurgery 1982;10(2):252-257.
<https://doi.org/10.1227/00006123-198202000-00013>
36. El Maaqili MR, Hossini A, El Fatemi N, et al. Primary glioblastoma of the cerebellum in a 19-year-old- woman: a case report. Journal of Medical Case Reports 2012;6: 329.
<https://doi.org/10.1186/1752-1947-6-329>
37. Zito JL, Siva A, Smith TW, Leeds M, Davidson R. Glioblastoma of the cerebellum. Computed tomographic and pathologic considerations. Surg Neurol 1983;19:373-378.
[https://doi.org/10.1016/0090-3019(83)90248-3](https://doi.org/10.1016/0090-3019%2883%2990248-3)
38. Law M, Cha S, Knopp EA, Johnson G, Arnett J, Litt AW. High-grade gliomas and solitary metastases: differentiation by using perfusion and proton spectroscopic MR Imaging. Radiology 2002;222:715-721.
<https://doi.org/10.1148/radiol.2223010558>
39. Chaichana K, Parker S, Olivi A, Quiñones-Hinojosa A. A proposed classification system that projects outcomes based on preoperative variables for adult patients with glioblastoma multiforme. J Neurosurg 2010;112:997-1004.
<https://doi.org/10.3171/2009.9.JNS09805>
40. Laws ER, Parney IF, Huang W, et al. Glioma outcomes investigators: survival following surgery and prognostic factors for recently diagnosed malignant glioma: data from the Glioma Outcomes Project. J Neurosurg 2003;99:467-473.
<https://doi.org/10.3171/jns.2003.99.3.0467>
41. Bi WL, Chiocca EA. Adult Cerebellar Glioblastomas: A Distinct entity or parcel of the whole? World Neurosurg 2013;80,6:e181-e183.
<https://doi.org/10.1016/j.wneu.2013.03.040>
42. Stupp R, Mason WP, van den Bent MJ, et al. European organization for research and treatment of cancer brain tumor and radiotherapy groups; national cancer institute of Canada Clinical Trials Group: radiotherapy plus concomitant and adjuvant temozolomide for glioblastoma. N Engl J Med 2005;352:987-996.
<https://doi.org/10.1056/NEJMoa043330>
43. Grossman SA, Ye X, Piantadosi S, Desideri S et al. NABTT CNS Consortium. Survival of patients with newly diagnosed glioblastoma treated with radiation and temozolomide in research studies in the United States. Clin Cancer Res 2010;16:2443-2449.
<https://doi.org/10.1158/1078-0432.CCR-09-3106>
44. Kaur G, Bloch O, Jian BJ, et al. A critical evaluation of cystic features in primary glioblastoma as a prognostic factor for survival. J Neur-osurg 2011;115:754-759.
<https://doi.org/10.3171/2011.5.JNS11128>
45. Yang S, Liu J, Wang T, Li X, You C. Cerebellar glioblastoma multi-forme: a retrospective study of 28 patients at a single institution. International Joumal of Neuroscience 2013;123(10):691-697.
<https://doi.org/10.3109/00207454.2013.791292>
46. Giese A, Bjerkvig R, Berens ME, Westphal M. Cost of migration: invasion of malignant gliomas and implications for treatment. J Clin Oncol 2003;21:1624-1636.
<https://doi.org/10.1200/JCO.2003.05.063>



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