Case Report

Glioblastoma and intracranial aneurysms: Case report and review of literature

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Abstract

Background: There is a paucity of data on the association of glioblastoma multiforme (GBM) with intracranial aneurysms. It is an important clinical entity for physicians to be aware of and its presence illustrates several critical features of the pathophysiology of malignant glioma. In this article we present a case of a middle cerebral artery (MCA) pseudoaneurysm that occurred in a patient with recurrent GBM and discuss the current literature relating to this unique combination of pathologies.

Case Description: The authors present a case of a MCA pseudoaneurysm that developed in a patient with recurrent GBM and discuss the current literature. The authors identified 19 reports describing 23 patients harboring both GBM and an intracranial aneurysm.

Conclusion: Several theories stand to explain the coincidental occurrence of intracranial aneurysms and GBM. The treating physician should be aware of this association when patients with intraaxial tumors present with unusual manifestation such as an intratumoral hemorrhage or angiogram negative subarachnoid hemorrhage. No guidelines exist to assist in the management of such patients; therefore, authors have attempted to address this issue using a classification and treatment algorithm.

Key Words: Glioblastoma multiforme, intracranial aneurysm, pseudoaneurysm, subarachnoid hemorrhage

INTRODUCTION

Glioblastoma and other malignant gliomas are the most common primary malignant brain tumors, with an annual incidence of 4.13 per 100,000 population. The prevalence of intracranial aneurysm is estimated to be 0.2–8.9% of the population. Brain tumors, particularly high-grade glioma, are rarely associated with intracranial aneurysm (0.19–4%). Intracranial aneurysms may arise at a location remote to the glioblastoma multiforme (GBM) in some instances, while in others they may be anatomically close to the tumor. The association of GBM and intracranial aneurysms can be categorized into three groups: Simultaneous development of glioblastoma and aneurysm, glioblastoma development after the treatment of aneurysm, and aneurysm development after the treatment of GBM. The treatment of newly diagnosed glioblastoma involves maximally
Although studies did not report the gender of their patient; of at presentation was 50.3 ± 11.04 years (+ SD). Several met the inclusion criteria. For these patients, the average screening of eligibility, 19 reports comprising 23 patients. The initial literature search yielded 35 studies. After studies mentioned outcomes as one of the above stated categories: Aneurysm unrelated to the tumor, location of the aneurysm, treatment performed, and the outcome. Furthermore, we divided the aneurysms into following categories: Aneurysm unrelated to the tumor (UnA), pseudoaneurysm or dissecting aneurysm (PsA), or feeding artery aneurysm (FaA). Outcomes were categorized as good, fair, and poor since most studies mentioned outcomes as one of the above stated categories [Table 1].[1,2,4,5,11–13,16,17,26,39]

### RESULTS

The initial literature search yielded 35 studies. After screening of eligibility, 19 reports comprising 23 patients met the inclusion criteria. For these patients, the average at presentation was 50.3 ± 11.04 years (+ SD). Several studies did not report the gender of their patient; of those that did, 10 were males and 6 females. Patients most commonly presented with symptoms related to intracranial tumor (headaches, confusion, seizures, and neurological deficit). Eighteen patients (78.2%) presented with symptoms secondary to the tumor, 2 patients presented with subarachnoid hemorrhage (SAH; 8.7%) and 3 patients presented with both SAH and tumor-related symptoms at some point in their clinical course (13%). The tumor was most commonly located in the frontal lobe (9 patients; 39.1%) followed by temporal lobe (6 patients; 26%), parietal lobe (3 patients; 13%), frontotemporal (2 patients; 8.7%), temporoparietal (2 patients; 8.7%), and location was unknown for 1 patient. Aneurysms were most commonly located on the internal carotid artery (ICA) (6 patients; 26%) and the middle cerebral artery (MCA) (6 patients; 26%) followed by anterior cerebral artery (ACA) (4 patients; 17.4%), anterior communicating artery (3 patients; 13%), posterior communicating artery (1 patient; 4.3%), posterior cerebral artery (PCA) (1 patient; 4.3%), lenticulostriate (1 patient; 4.3%), and 1 patient had an angiogram negative SAH. According to our classification, most aneurysms were UnA (10 patients; 43.4%). There were two patients each with PsA and one patient with FaA. Aneurysms could not be categorized in either of these categories in eight patients due to lack of enough information in the reports. Eleven patients (47.8%) underwent treatment of both the tumor and the aneurysm. All but one patient had their aneurysm surgically clipped; one patient underwent endovascular coiling of the traumatic pseudoaneurysm following tumor resection.[6] In six patients (26%), only the tumor was treated and aneurysm was not addressed, whereas in three patients (13%), aneurysm was treated but tumor was not addressed. No treatment was offered in one case and treatment was not described for two patients. Of 23 patients, 7 patients had a good outcome, 2 had a fair outcome and 10 patients had a poor outcome. Nine out of 10 patients with a poor outcome had died during the hospital stay or at their last follow-up secondary to their intracranial disease. Outcome could not be ascertained for four patients.

### Illustrative case

A 63-year-old right-handed male presented to the hospital with headaches and dizziness. His neurological examination was normal except for mild left hand ataxia. A magnetic resonance imaging (MRI) of the brain demonstrated a heterogeneously enhancing mass in the right frontotemporal region extending into the right basal ganglia and internal capsule [Figure 1a]. He underwent subtotal resection via a right frontotemporal craniotomy. Pathology was consistent with anaplastic oligodendroglioma. He completed a course of fractionated external beam radiation and concurrent temozolomide followed by adjuvant temozolomide.

### MATERIALS AND METHODS

PubMed was queried using the terms “GBM,” “glioblastoma multiforme,” “ruptured aneurysm,” “unruptured aneurysm,” “intracranial aneurysm,” “dissecting aneurysm,” “pseudoaneurysm” through June 2014. All studies and reports that mentioned cases harboring both GBM and an intracranial aneurysm were included in the study. No restrictions were imposed based on publication dates, types or languages. References included in the study. No restrictions were imposed based on publication dates, types or languages. References
| Author and year            | Age (years)*, sex | Presentation                        | Tumor location   | Tumor histology | Aneurysm location | Aneurysm type | Treatment                                      | Outcome                        |
|---------------------------|------------------|------------------------------------|------------------|----------------|-------------------|--------------|-----------------------------------------------|--------------------------------|
| Cheng et al., 2004        | 67, F            | Neuro deficit sec to tumor         | Left parietal    | GBM            | Left supraciloid ICA | UnA         | Staged; Clipping then tumor resection        | Good at 1 year                 |
| Joki et al., 2013         | 31, M            | SAH                                | Left uncus       | GBM            | Angio negative SAH | No aneurysm | Biopsy                                        | Unknown                       |
| Hashiguchi et al., 2007   | 44, F            | Intratumoral hemorrhage            | Left frontal     | GBM            | Left Lenticulostriate artery | FaA        | Observation                                   | Poor (vegetative)             |
| Aoki et al., 1999         | 57, M            | Neuro deficit sec to tumor         | Right frontotemporal | GBM        | Right proximal MCA | PsA         | Tumor resection with clip sacrifice of MCA | Fair                           |
| Yoon et al., 2011         | 57, M            | Headaches + SAH                    | Right frontal    | GBM            | Right distal ACA  | UnA         | Subtotal tumor resection; aneurysm clipped  | Good                           |
| Gokalp et al., 1980       | 50, M            | Visual disturbances                | Left frontal     | GBM            | Anterior communicating artery | UnA; giant aneurysm | Tumor resection + aneurysm clipping          | Poor (dead)                    |
| Plangger et al., 1987     | 69, M            | Hemiparesis and hemianopsia        | Right temporoparietal | GBM       | Posterior communicating artery | UnA        | Tumor resection; aneurysm not addressed     | Poor (dead)                    |
| Andrews et al., 1985      | 53, F            | Aneurysmal SAH + intratumoral hemorrhage | Left temporal | GBM            | Left M2 of MCA  | PsA; Invasion of vessel wall by tumor | Aneurysm trapped and excised; Tumor resected | Unknown                      |
| Honda et al., 1980        | 59, F            | Hemiparesis                        | Left temporal    | GBM            | Anterior communicating artery | UnA       | Aneurysm clipped, tumor partially resected | Good                           |
| DeChiara et al., 1986     | 52, F            | SAH initially; left hemiparesis later with tumor | Right temporal | GBM            | Right ICA       | UnA         | Aneurysm clipped, tumor partially resected | Fair                           |
| Paoletti et al., 1983     | 59, M            | Confusion, intracranial HTN        | Left frontal     | GBM            | Left MCA trifurcation | UnA       | Tumor resected and aneurysm clipped          | Good; KPS 70                   |
| Cohen et al., 2005        | 35, F            | Cognitive changes                  | Right frontal    | GBM            | Left pericallosal pseudoaneurysm | PsA; iatrogenic | Gross total resection of tumor, endovascular coiling of pseudoaneurysm | Poor (dead at 1 year)          |
| Beller et al., 1951       | 32, ?            | Tumor symptoms                     | Left parietal    | GBM            | Left ICA        | UnA        | Resection of tumor, aneurysm untreated       | Good                           |

Contd...
Table 1: Contd...

| Author and year | Age (years)*, sex | Presentation | Tumor location | Tumor histology | Aneurysm location | Aneurysm type | Treatment | Outcome |
|-----------------|-------------------|--------------|----------------|-----------------|------------------|---------------|-----------|---------|
| Obrador et al., 1972 | 54, M | Tumor symptoms | Left temporo-occipital | GBM | AComm | UnA | Partial tumor resection + aneurysm clipping | Poor (dead) |
| Evans et al., 1972 | 55, M | SAH | Right parietal | GBM | Right PCA | Unknown | Resection of tumor, spontaneous thrombosis of aneurysm | Poor (dead) |
| Heppner et al., 1953 | 50, M | Tumor symptoms | Left temporal | GBM | Left ICA | UnA | Aneurysm clipped only | Poor (dead) |
| Kraus et al., 1972 | ‡ | Tumor symptoms | ‡ | GBM | MCA | Unknown | Tumor resected and aneurysm clipped | Good |
| Taylor et al., 1961 | 33, M | Tumor symptoms | Right frontal | GBM | Right ACA | Unknown | Aneurysm clipped only | Poor (dead) |
| Taylor et al., 1961 | 50, M | Tumor symptoms | Right fronto-temporal | GBM | Right MCA | Unknown | Aneurysm clipped only | Poor (dead) |
| Taylor et al., 1961 | 50, M | Tumor symptoms | Right temporal | GBM | Right ICA | Unknown | Aneurysm clipped only | Poor (dead) |
| Taylor et al., 1961 | 33, M | Tumor symptoms | Left temporal | GBM | Left ACA | Unknown | Aneurysm clipped only | Poor (dead) |
| Taylor et al., 1961 | 50, M | Tumor symptoms | Left fronto-temporal | GBM | Left MCA | Unknown | Aneurysm clipped only | Poor (dead) |
| Krayenbuehl et al., 1965 | 50, M | Tumor symptoms | Left frontal | GBM | RCA | Unknown | Tumor resection only | Poor (dead) |
| Krayenbuehl et al., 1965 | 50, M | Tumor symptoms | Left frontal | GBM | MCA | Unknown | Tumor resection only | Poor (dead) |

GBM: Glioblastoma multiforme, ICA: Internal carotid artery, SAH: Subarachnoid hemorrhage, M: Male, F: Female, ACA: Anterior cerebral artery

‡ This patient developed a traumatic pseudoaneurysm following surgical resection of GBM.

† Quoted by Pia et al.

UnA: Aneurysm unrelated to the tumor; FaA: Aneurysm on the artery feeding the tumor; PsA: Pseudoaneurysm (tumor invasion of vessel or iatrogenic)

Subsequently he progressed and underwent second-line chemotherapy with Bevacizumab and Irinotecan followed by stereotactic radiosurgery (SRS) to the right frontotemporal region. Several years later, progressive enhancement was discovered on routine surveillance imaging and the patient underwent reresection [Figure 1b]. Intraoperatively, branches of the right MCA were found to be intimately associated with tumor-like tissue. The middle cerebral vessels were carefully skeletonized, but several areas of tissue were densely adherent to the MCA and therefore, some residual tumor-like tissue was left behind. Subsequent pathologic examination revealed radiation necrosis with no evidence of tumor recurrence and the patient was discharged in a stable condition. Several weeks later, the patient returned to our emergency department, Glasgow Coma Scale of 6 and with extensor posturing on neurological examination. Computed tomography (CT) of the head revealed an extensive area of acute hemorrhage predominantly involving the tumor resection bed with dissection into the ventricular system. An external ventricular drain was placed and the patient underwent emergent cerebral angiography. The angiogram displayed a 2 cm, rapidly filling aneurysm with extremely irregular walls and no identifiable neck arising from the region of the right MCA trifurcation [Figure 1c and d]. Given the patient’s poor neurological status and advanced malignant glioma, aggressive therapies were not pursued and the patient died 3 days later.

Figure 1: GBM and pseudoaneurysm. Pretreatment T1-weighted gadolinium-enhanced axial MRI demonstrating a heterogeneously enhancing mass in the right frontotemporal region extending into the right basal ganglia and internal capsule in 2005 (a). Posttreatment T1-weighted, gadolinium-enhanced coronal MRI of a cystic and nodular heterogeneously enhancing frontotemporal lesion extending from the middle fossa floor to the right basal ganglia in 2011 (b). Oblique AP (c) and oblique lateral (d) projections of a right internal carotid artery injection during a digital subtraction angiogram revealing the presence of a 2 cm, rapidly filling aneurysm with extremely irregular walls and no identifiable neck arising from the region of the right MCA trifurcation later in 2011.


**DISCUSSION**

Glioblastoma development is most common in the sixth through eighth decades of life.[21] Glioblastoma accounts for 82% of malignant gliomas and histologically depicts significant cellularity and mitotic activity, vascular proliferation, and necrosis.[20] Although gliomas have a significant hemorrhage rate, they are almost exclusively intraparenchymal bleeds, with only rare extension to the subarachnoid space.[10] A review of Cushing’s series of 832 gliomas found 7 cases where SAH appeared to be the presenting feature.[22] Aneurysms can be seen in approximately 0.5% of patients with brain tumors.[9]

The middle cerebral and internal carotid arteries are the most common sites of aneurysm formation in patients with gliomas, similar to our findings from the literature review.[24] The underlying pathophysiology is said to involve a tenuous and necrotic neovasculature, neoplastic invasion of nascent cerebral vessels, and the propagation of a systemic coagulopathy.[11] It is also postulated that increased blood flow to these tumors may induce secondary changes in the arterial wall directly and thus facilitate the formation of intracranial aneurysms.[1] Intratumoral shunting of blood flow may also lead to aneurysm formation.[25] Jakubowski and colleagues reviewed 150 pituitary tumors and found that growth hormone-producing adenomas had an increased incidence of intracranial aneurysm compared with the chromophobe variety.[15] Abnormal growth hormone production can lead to atherosclerosis and hypertension, which can in turn weaken vessels walls and lead to aneurysmal dilatation.[15] In addition, direct invasion by tumor cells can lead to vessel wall erosion and aneurysm formation.[1]

Ionizing radiation is an established environmental risk factor for glioma development and has also been hypothesized to be involved in vessel wall changes leading to aneurysm formation.[1] It has been proposed that postradiotherapy aneurysm development is due to endothelial damage as a result of radiation-induced vasculopathy. This endothelial damage leads to thrombosis, intimal narrowing, and atherosclerosis.[1] Combined with weakening of the vessel wall by the effects of radiation, raised vascular pressure is thought to induce changes that lead to formation of saccular and fusiform aneurysms as well as pseudoaneurysms.[23] According to a recent retrospective review of literature, the median lag time between radiation exposure and diagnosis of an intracranial aneurysm varied according to the radiation modality, being greatest for brachytherapy, followed by focused brain radiation, whole-brain radiation, and SRS.[21]

Unintentional luminal shearing forces applied intraoperatively to an artery leading to an arterial dissection is an alternative hypothesis for aneurysm formation in patients who have had a craniotomy for resection of a GBM.[10] Cohen et al. describe a case of iatrogenically caused pseudoaneurysm of pericallosal artery in a patient who underwent surgical resection of GBM.[6] It is not uncommon to patients to have intracranial hemorrhage after undergoing craniotomy for GBM resection. However, in almost all cases, hemorrhage would be exclusively intraparenchymal. Postoperative intraparenchymal hemorrhage along with significant SAH should raise suspicion for iatrogenic traumatic aneurysm and cerebral angiography should be undertaken to rule it out. In our case, the pseudoaneurysm likely developed secondary to radiation-induced endothelial injury and prior vessel wall invasion by the tumor as well as manipulation of the exceedingly fragile middle cerebral vasculature during surgery. Unfortunately, the patient did not undergo autopsy postmortem, hence, the real reason for the development of pseudoaneurysm remains unknown in our case.

In most instances of coincident malignant glioma and intracranial aneurysm symptoms are attributed to the tumor, with aneurysm-related symptoms present in only about 25% of the cases.[25] Mixed symptomatology is seen in about 6% of cases.[25] As evident from our literature review, about 80% of the patients presented with tumor-related symptoms and only 8% presented with aneurysmal symptoms. It is also important to note that of the various nonaneurysmal causes of SAH, brain tumors represent 1–3% of these cases.[19] In the case of our patient, even though the initial presentation was related to the tumor, the ultimate demise of the patient occurred as the result of the ruptured pseudoaneurysm. The treatment of patients with coincident malignant glioma and intracranial aneurysm is controversial and requires special consideration. The decision-making requires an understanding of the symptoms attributable to the individual pathologies, the location of the aneurysm and malignant glioma and overall prognosis of the patient. The more symptomatic lesion should be treated first. Prognosis is mainly determined by the nature of the tumor and its response to treatment as well as the rupture status of the aneurysm. There is some evidence to suggest that the combination of the two different pathologies does not worsen the outcome.[18] The total mortality of this group is reported to be as high as 38% whether the tumor alone, aneurysm alone, or both are surgically treated.[25]

We have developed a new classification for the aneurysms that are found in association with GBM. We classify these aneurysms as UnA, FaA, and PsA. UnA are aneurysms that are located remotely from the tumor, appear like typical saccular aneurysms and are not located on a vessel that might be supplying the tumor. FaA are located on an artery seen to be the major blood supply to the tumor, as assessed by a cerebral angiogram or CT angiogram. PsA
are false aneurysms that do not possess all three vessel wall layers, lack a distinguishable neck and are formed by either invasion of the vessel by the tumor cells or develop after an iatrogenic injury during surgical resection of the tumor. Our illustrative case is a perfect example of PsA. This classification can aid designing treatment plan for individual patients. For patients with UnA, the tumor should be addressed first to relieve the mass effect and achieve cytoreduction, obtain diagnostic sample and initiate adjuvant therapy. If the UnA is located on the ipsilateral side to the tumor, it may be clipped in the same sitting or could be clipped or coiled at a later time. For patients with FaA, these should be addressed in the same setting as the tumor resection since it is very likely that the aneurysm will be located close to the tumor and can be addressed through the same operative corridor. If the FaA is distant from the tumor and cannot be tackled through the same operative corridor, the tumor should be resected first and the aneurysm be addressed soon after surgery. In cases with PsA, these should be addressed as urgently as possible due to their high risk of rupture as they are inherently fragile. In such cases, the tumor should be resected and the aneurysm clipped, reconstructed or trapped as deemed appropriate by the surgeon. In cases of postoperative traumatic PsA formation, the patient should urgently undergo cerebral angiogram and coiling should be attempted. These are only recommendations and are based on limited data obtained from the literature and should not serve as guidelines.

**CONCLUSION**

We present a pooled analysis of the literature of intracranial aneurysms occurring in association with GBM along with an illustrative case report and discuss modes of presentation, locations of tumors and aneurysms, types of aneurysms, treatment modalities, and outcomes. We also propose a new classification to categorize the aneurysms that arise in association with GBM to assist the treating surgeons in formulating a management plan. We realize that this report has several limitations and its quality does not parallel that of a randomized controlled trial, however, in the face of limited data and scarcity of such cases, this is a humble attempt toward raising awareness of neurosurgeons in practice and in training of this unusual pathological association.

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