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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 as well 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

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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.[23] The prevalence of intracranial aneurysm is estimated to be 0.2–8.9% of the population.[31] Brain tumors, particularly high-grade glioma, are rarely associated with intracranial aneurysm (0.19–4%).[10,14,25,30,32] 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

efficacious and safe surgical resection combined with a regimen of concurrent temozolomide and fractionated radiotherapy followed by adjuvant temozolomide. This approach has been shown to improve median overall survival from 12.1 to 14.6 months and 2-year survival rates were enhanced from 10.4% to 26.5%.[29] Although the 5-year survival rates quintupled with this regimen, 5-year mortality remains unacceptably high.[23,28]

While rare, aneurysms are an important cause of morbidity and mortality in patients with malignant glioma and should be recognized as such by treating physicians. Additionally, the development of aneurysms in the high-grade glioma patient, given the pathophysiology of the tumor and that which is induced by treatment of the tumor, lends critical insights into the pathophysiology underlying malignant gliomas. In this article, we present the current literature and theory underlying the pathophysiology and development of aneurysms in high grade glioma through the lens of a case seen by our service throughout 2005–2011. We review all reported cases of patients with GBM that were also found to harbor intracranial aneurysms.

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 in the reports were also searched for completeness. Reports that were reported or cited multiple times in the literature were only included once in the study. From all the studies, we extracted the following information: Patient demographics (age and gender), presenting symptoms (tumor vs aneurysm related), location of 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), psuedoaneurysm 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].[2,4,5,11–13,16,17,26,33]

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%), temporo-occipital (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 communication 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. Outcomes 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.

Table 1: Studies reporting patients with GBM and intracranial aneurysms

Author and year	Age (years)*, sex	Presentation	Tumor location	Tumor histology	Aneurysm location	Aneurysm type	Treatment	Outcome
Cheng *et al.*, 2004[4]	67, F	Neuro deficit sec to tumor	Left parietal	GBM	Left supraclinoid ICA	UnA	Staged; Clipping then tumor resection	Good at 1 year
Joki *et al.*, 2013[16]	31, M	SAH	Left uncus	GBM	Angio negative SAH	No aneurysm	Biopsy	Unknown
Hashiguchi *et al.*, 2007[11]	44, F	Intratumoral hemorrhage	Left frontal	GBM	Left Lenticulostriate artery	FaA	Observation	Poor (vegetative)
Aoki *et al.*, 1999[2]	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[33]	57, M	Headaches + SAH	Right frontal	GBM	Right distal ACA	UnA	Subtotal tumor resection; aneurysm clipped	Good
Gokalp *et al.*, 1980[9]	50, M	Visual disturbances	Left frontal	GBM	Anterior communicating artery	UnA; giant aneurysm	Tumor resection + aneurysm clipping	Poor (dead)
Plangger *et al.*, 1987[26]	69, M	Hemiparesis and hemianopsia	Right temporo-occipital	GBM	Posterior communicating artery	UnA	Tumor resection; aneurysm not addressed	Poor (dead)
Andrews *et al.*, 1985[1]	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[13]	59, F	Hemiparesis	Left temporal	GBM	Anterior communicating artery	UnA	Aneurysm clipped, tumor partially resected	Good
DeChiara *et al.*, 1986[7]	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[24]	59, M	Confusion, intracranial HTN	Left frontal	GBM	Left MCA trifurcation	UnA	Tumor resected and aneurysm clipped	Good; KPS 70
Cohen *et al.*, 2005[5]	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	Good
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[12]	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[30]	33, M	Tumor symptoms	Right frontal	GBM	Right ACA	Unknown	Aneurysm clipped only	Poor (dead)
	-	Tumor symptoms	Right fronto temporal	GBM	Right MCA	Unknown	Aneurysm clipped only	Poor (dead)
	-	Tumor symptoms	Right temporal	GBM	Right ICA	Unknown	Unknown	Unknown
	-	Tumor symptoms	Right frontal	GBM	Right ICA	Unknown	Unknown	Unknown
Krayenbuehl *et al.*, 1965[17]	50, -	Tumor symptoms	Left frontal	GBM	ACA	Unknown	Tumor resection only	Poor (dead)
	50, -	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, *Age at time of presentation, †This patient developed a traumatic pseudoaneurysm following surgical resection of GBM, ‡Quoted by Pia *et al.*[25], UnA: Aneurysm unrelated to the tumor; FaA: Aneurysm on the artery feeding the tumor; PsA: Pseudoaneurysm or dissecting aneurysm (tumor invasion of vessel or iatrogenic)

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

Subsequently he progressed and underwent second-line chemotherapy with Bevacizumab and Irinotecan followed by sterotactic 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, Glascow 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.

DISCUSSION

Glioblastoma development is most common in the sixth through eighth decades of life.[23] 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.[8] 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.[7]

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.[3] It has been proposed that postradiotherapy aneurysmal 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.[27] 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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