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Case Report

Symptomatic cerebral vasospasm in the setting of carmustine wafer placement for glioblastoma: A case presentation and review of literature

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ABSTRACT

Background: Gliadel placement in glioblastoma resection, particularly with concurrent chemoradiation, has demonstrated an improvement in survival. There have been several reported adverse effects, some of which lend to significantly increased morbidity and mortality. With only two other cases described in literature, cerebral vasospasm secondary to carmustine-impregnated wafers is an extremely rare side effect.

Case Description: We report the case of a 51-year-old female who presented with the left lower limb paresis 8 days after high-grade glioma resection provoked by carmustine wafer placement.

Conclusion: We urge surgeons to reconsider placement of carmustine wafers in nations where the surgical resection cavity includes exposed large cerebral vasculature. We also propose the early identification of this devastating complication in the postoperative period by maintaining a high clinical suspicion and prompt utilization of computed tomography and digital subtraction angiography in the management and treatment of these patients accordingly.

Keywords: Carmustine wafer, Cerebral vasospasm, Glial tumor, Glioma, Tumor resection

BACKGROUND

Carmustine-impregnated wafers (Gliadel®, Eisai, Baltimore, Maryland, USA) are biodegradable polymers designed to deliver high concentrations of 1,3-bis-2-chloroethyl-1-nitrosourea (BCNU) into cerebral tumor resection cavities while surpassing the blood-brain barrier and avoiding the constitutional side effects of systemic chemotherapy.[13,14,26] Several studies have demonstrated the role of Gliadel in improving overall survival in patients with recurrent and newly diagnosed high-grade gliomas, particularly in the setting of concurrent radiation and chemotherapy.[2,3,6,9,12,15,16,32] Despite its efficacy, Gliadel has also been associated with a number of serious adverse events, including seizures, brain edema, intracranial hypertension, cerebrospinal fluid leakage, intracranial infection, healing abnormalities, hydrocephalus, and cyst formation.[2,3,15,32] Although symptomatic cerebral vasospasm (sCVS) has been described in the context of temporal lobectomy for refractory epilepsy[11,19,20,24] and largely in the setting of skull base tumor resection,[1,5] only two cases in the literature describe vasospasm as a result

of carmustine-impregnated wafer implantation.[21,22] The phenomenon is believed to be secondary to a combination of the vasospastic properties of carmustine, local toxicity, and inflammation.[21,22] In this case report, we present a patient who experienced sCVS attributed to carmustine-impregnated wafer placement after the resection of glioblastoma (GBM). Given the high morbidity of sCVS, the authors propose high index of clinical suspicion, timing of this phenomenon, recommendations for placement, and early identification by utilizing CT angiography in the setting of postoperative symptoms. We aim to prepare physicians for the proactive identification and treatment of patients susceptible to this morbid phenomenon.

CASE REPORT

A 51-year-old female was transferred to our institution after the discovery of a large right temporal and insular lesion on magnetic resonance imaging (MRI). The patient initially presented with a few months of headaches and smelling odd odors to her primary care physician, who then prescribed the patient levetiracetam (Keppra, UCB Pharmaceuticals, Belgium) and referred her to our institution. The patient had no deficits on gross physical examination. Contrasted computed tomography (CT) with contrast revealed a large mass with irregular enhancement and MRI revealed moderate perilesional edema with mass effect [Figure 1].

The patient underwent an uncomplicated right frontotemporal craniotomy for tumor resection. At the anteromedial extent of the tumor, the Sylvian vessels were encountered and preserved. The dissection of the tumor was performed with preservation of the pial demarcation of the Sylvian fissure. The anterior choroidal artery and MCA branches were met and not manipulated as this indicated the anterior extent of the tumor. An amygdalohippocampectomy was also performed. Intraoperative monitoring, with somatosensory evoked potentials, motor evoked potentials, and electroencephalography, was performed throughout the course of the operation and remained at baseline. Frozen pathology confirmed the presence of high-grade glioma, and at that time, the decision was made to line the surgical cavity, including exposed Sylvian fissure and large cerebral vessels, with four carmustine wafers. Two wafers were placed along the cephalad resection cavity in direct contact with the exposed portion of the Sylvian fissure and the remainder along the posterior resection wall [Figure 1]. The wafers were secured with oxidized regenerated cellulose. There was minimal intraoperative bleeding.

Postoperatively, the patient experienced no worsening deficits. Immediate postoperative CT revealed small volume hemorrhage within the posteromedial aspects of the resection cavity, measured to be about 1.7 cm^3 per volumetric analysis through OsiriX software (Apple, California) [Figure 2].

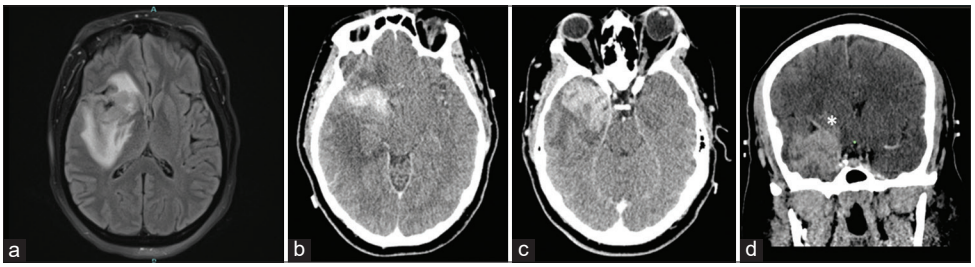

Figure 1: (a) FLAIR magnetic resonance imaging showing right temporal and insular lesion with right to left mass effect and surrounding edema. (b and c) CT with contrast demonstrating irregular enhancement of a large right temporal and insular lesion. (d) A coronal view of the preoperative CT with contrast, with an emphasis on the Sylvian fissure and middle cerebral artery (MCA) being pushed upward (asterisk).

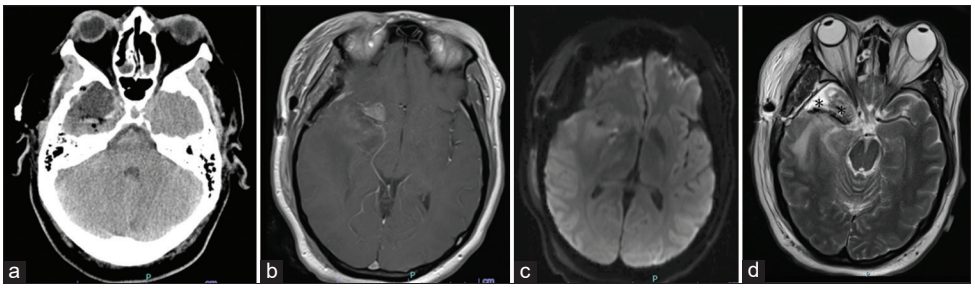

Figure 2: (a) Postoperative CT with minimal hemorrhage in the posterior aspect of the resection cavity. (b) The T1 magnetic resonance imaging (MRI) with contrast respectively, with small amount of residual tumor anterosuperiorly and medially, with small amount of hemorrhage within the resection cavity and no evidence of hemorrhage within the basal cisterns. (c) Diffusion-weighted imaging without any evidence of infarct. (d) A T2 MRI with hypointensities along exposed middle cerebral artery (asterisks), representing Gliadel wafer lining the vessel and cavity.

MRI on postoperative day 1 revealed subtotal resection with improved mass effect but persistent edema [Figure 2]. She was eventually discharged from our care on postoperative day 5.

On postoperative day 8, the patient experienced new left lower extremity weakness, which progressed to include slurring of speech and left facial weakness. Over the course of the next 24 h, this evolved to plegia in the left upper extremity and severe hemiparesis in the left lower extremity. MRI revealed stable edema and an acute ischemic stroke in the right posterior limb of the internal capsule [Figure 3]. CT angiography and digital subtraction angiography (DSA) on postoperative day 9 revealed moderate vasospasm in the right supraclinoid internal carotid artery (ICA), the carotid terminus, A1 of the anterior cerebral artery, and M1 of the MCA [Figure 3]. After intra-arterial injection of 20 mg of nicardipine and angioplasty of the right MCA and supraclinoid ICA, there was some improvement in vessel diameter and caliber [Figure 3]. The patient remained in the hospital for several days for medical management of vasospasm (fluids, oral nimodipine, and permissive hypertension) and intensive rehabilitation. Histopathology was finalized, showing features consistent with those of the World Health Organization Grade IV GBM, and the patient went onto receive concomitant temozolomide and radiation. At 36-month postoperative follow-up, the patient had resolution of dysphasia but had persistent left upper extremity plegia and left lower extremity paresis.

DISCUSSION

CVS is a well-described entity in patients with aneurysmal subarachnoid hemorrhage (aSAH)[4,8,10,34] and following epilepsy surgery,[11,19,20,24] but its incidence in the postoperative course of tumor resection is low.[7] Bejjani *et al*. conducted a retrospective review of 470 patients with skull base tumors and noted 1.9% incidence of CVS postoperatively.[5] They proposed manipulation of nearby large cerebral vessels during dissection and increased blood spillage into the basal cisterns from skull base approaches as possible causes.[5] Alotaibi and Lanzino, in their systematic review of CVS in the setting of primarily skull base tumor resection, emphasized postoperative subarachnoid hemorrhage, preoperative vessel encasement, mechanical stretching and manipulation, hypothalamic dysfunction, tumor content spillage, and meningitis as contributive factors.[1]

This case does not include the aforementioned factors. There was no manipulation of exposed Sylvian vessels, given that this was the anterior extent of our resection. The vessels were identified and preserved. The delayed timing of the sCVS does not match the course of "traction hemiplegia," which tends to manifest as CVS intraoperatively or immediately in the perioperative course and is short termed.[7,11,19,28,29] Although there was a minimal amount of hemorrhage within the posterior and medial aspects of the operative bed, there was no evidence of postoperative subarachnoid hemorrhage or hemorrhage within the basal cisterns. Lackner *et al*. implicated high bleeding volume on postoperative CT in the

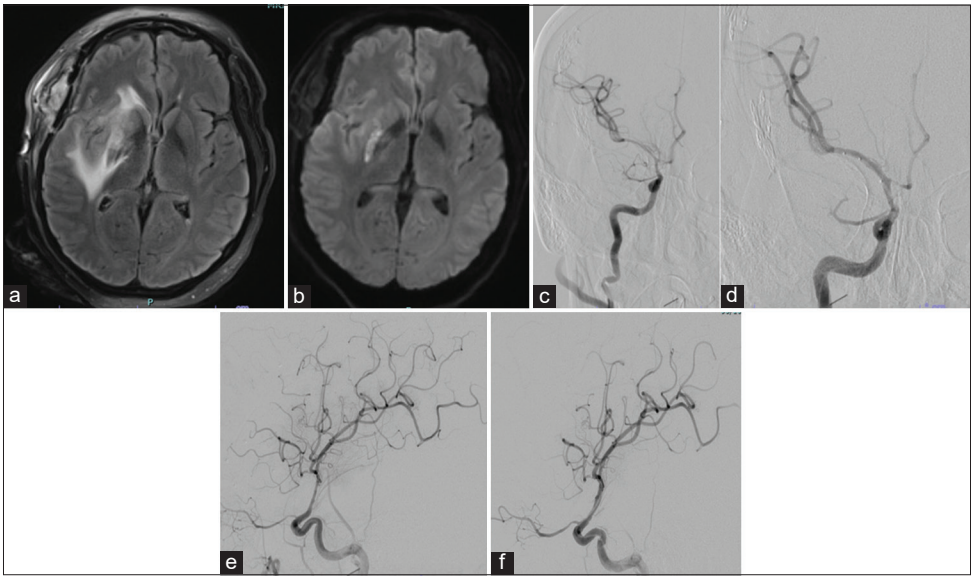

Figure 3: (a) FLAIR magnetic resonance imaging revealing stable perilesional edema on postoperative day 8. (b) Diffusion-weighted imaging showcasing a right posterior limb of the internal capsule acute infarct. (c-f) Cerebral angiographic images of the anterior circulation displaying moderate vasospasm in the right supraclinoid internal carotid artery (ICA), the carotid terminus, A1 of the anterior cerebral artery, and M1 of the middle cerebral artery (MCA). (e and f) Improvement in caliber and diameter of supraclinoid ICA and MCA after angioplasty and intra-arterial injection of nicardipine.

development of sCVS.[19] The volumetric analysis conducted in our study through OsiriX software, the same method as that used in Lackner *et al*.'s study, yielded a measurement of 1.8 cm^3, lower than the mean postoperative bleeding in their anterior temporal lobectomy group (5.5 cm^3).[19] Worsening edema has also been implicated in the development of sCVS.[18] In our case, postoperative edema immediately after surgery and at the time of sCVS 8 days later was stable and similar to preoperative imaging.

Gliadel polymers function through controlled release of contents over a period of 2–3 weeks after implantation.[13,14,26] The concentration of BCNU at the site of implantation is 1200 times higher than that achieved with administration of systemic carmustine.[13,14,21,22,26] The exact mechanism of sCVS secondary to BCNU implantation is unclear, though it is likely a combination of local toxicity, inflammatory mediators released in response to foreign body reaction to the polymer, and the vasospastic effects of carmustine.[21,22] High-dose intravenous carmustine (800/m^2) has been associated with retinal artery narrowing and obstruction.[31] Carmustine has also been shown to induce vasospasm in rabbit models through inhibition of glutathione reductase activity, indicating presence of local inflammatory reactions secondary to the foreign wafers.[30] In addition, Shibahara *et al*. demonstrated a significant increase in surrounding immune cells with BCNU implantation in GBM patients and augmentation of inflammatory response in nearby tissue.

The previous cases of sCVS with Gliadel implantation along with ours are outlined in [Table 1]. The timing of the CVS is similar in two cases and differs in the other. sCVS occurred immediately after surgery in the case described by Muzii *et al*. and likely follows the course of sCVS secondary to manipulation.[7,11,19,21,28,29] Given the fact that, the peak effect of carmustine wafers occurs around day 5–7 after implantation,[13,14,26] a delayed course of vasospasm as displayed in our case and the case presented by Nakada *et al*. seems more likely to follow the pathogenesis.[22] Therefore, the delayed onset of symptoms in future cases of Gliadel placement should raise suspicion of this phenomenon for clinicians.

In all the cases of sCVS with Gliadel implantation [Table 1], Gliadel was laid directly over or in close proximity to the vasculature with vasospasm on angiographic imaging. Nakada *et al*. described a case in which Gliadel was placed in close proximity to the lenticulostriate artery, which manifested as selective sCVS.[22] In our case and the case described by Muzii *et al*., Gliadel lined the surgical cavity which included exposed large cerebral vessels.[21] Unfortunately, other than safety guidelines of the product label, there are no evidence-based recommendations to guide implantation of carmustine wafers.[15] Gutenberg *et al*. reported two cases of significant perioperative bleeding believed to be secondary to rupture, dissection, or thromboembolic events as a result of direct contact between the wafers and vasculature.[15] Sato *et al*. reported a case of delayed pseudoaneurysm formation and subsequent rupture secondary to dissection induced by similar proposed mechanisms.[27] Interestingly, all three cases of sCVS occur in temporal and insular regions. Several authors have proposed avoiding Gliadel placement in eloquent areas secondary to a risk of worsening cerebral edema and compressive deficits.[18] We argue for caution when directly applying carmustine wafers in close proximity to large cerebral vessels, particularly in the temporal and insular regions.[15,18] Further studies are necessary to elucidate the molecular mechanisms of this occurrence and whether there is an enhanced risk of vasospasm secondary to the interactions of BCNU and specific vasospastic mediators released by the peritumoral environment.

Treatment of sCVS is similar to that instituted in aSAH.[4,21,22,35] The case described by Nakada *et al*. and our case both had delays in angiographic diagnosis and eventual treatment.[22] Both cases also had infarctions and lasting deficits.[22] Muzii *et al*. described a case with prompt diagnosis and treatment, with sudden resolution of symptoms.[21] Woo *et al*. recommended continued radiographic surveillance of CVS with TCDs and/or angiographic imaging, though the correlation between radiographic CVS and persistent symptoms is still unclear.[19,33,34] We urge clinicians to maintain a high suspicion for sCVS postoperatively in patients with delayed neurological deficits after Gliadel wafer placement in high-grade glioma. We propose the early use of CT angiography in this patient population. If CVS is identified, this should be promptly followed by DSA for further management and treatment.

Given that Gliadel does lend a survival benefit in a devastating disease,[2,3,6,9,12,15,16,32] prophylactic measures ought to be sought to prevent the occurrence of CVS rather than discontinuation of its use in high-risk cases. Perhaps, the topical administration of papaverine can alleviate the effect. Papaverine, a phosphodiesterase inhibitor, has often been used in skull base and cerebrovascular cases to mitigate CVS with success.[23,25] Recently, it has also been shown to harbor anti-cancerous properties by inhibiting GBM cell growth in mice models, making it an interesting adjunct in this setting.[17]

CONCLUSION

We urge surgeons to reconsider universal placement of carmustine wafers in GBM in situations where large intracranial vasculature is exposed within the tumor resection cavity. We also propose the early identification of this devastating complication in the postoperative period by maintaining a high clinical suspicion and prompt utilization of CT and DSA in the management and treatment of these patients accordingly.

Table 1: Cases of symptomatic cerebral vasospasm after Gliadel placement.

Article	Age (years) and gender of patient	Pathology and location of tumor	Onset of symptoms (postoperative day)	Symptoms	Gliadel placement?	Vasospasm location as confirmed by angiographic imaging	Presence of stroke (Y/N)?	Treatment	Results
Nakada *et al.*, 2014[17]	63, Male	GBM§; left temporal lobe and insula	11	Right hemiparesis with progression to hemiplegia	Yes, just above LSAII	Left LSAII on postoperative day 13	Yes, left corona radiata	Ozagrel sodium and edaravone	Improvement of hemiplegia to hemiparesis at 3 months
Muzii *et al.*, 2018[18]	57, Male	GBM§; left temporal lobe and insula	2	Moderate aphasia	Yes, along exposed vessels	Left M2 and M3 of MCA‡	No	Intra-arterial nimodipine, oral nimodipine	Aphasia resolved in 2 days
Present case, 2020	51, Female	GBM§; right frontal, temporal lobe and insula	8	Left facial weakness, dysphasia and worsening hemiparesis with progression to complete left hemiplegia	Yes, along exposed vessels	Right supraclinoid ICA†, carotid terminus, M1 of MCA‡ on postoperative day 9	Yes, right basal ganglia	Intra-arterial nicardipine, angioplasty of right supraclinoid ICA† and MCA‡, oral nimodipine, fluids, blood pressure augmentation	Persistent left upper extremity hemiplegia and improvement to left lower extremity paresis, resolution of dysphasia

§: Glioblastoma multiforme; II: Lenticulostriate artery; ‡: Middle cerebral artery; †: Internal cerebral artery

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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